Third Universal Definition of Myocardial Infarction

2012 ESC/ACCF/AHA/WHF Task Force for the Universal definition of Myocardial Infarction

CRITERIA FOR ACUTE MYOCARDIAL INFARCTION

The term **acute MI** should be used when there is evidence of myocardial necrosis in a clinical setting consistent with ischemia and any one of the following criteria:

1. Rise and/or fall of cardiac biomarkers [preferably cardiac troponin (cTn)] with at least one value > 99th percentile upper reference limit (URL) and at least one of the following:

a) Symptoms of ischemia.
b) New significant ST–T changes or new LBBB.
c) Development of pathological Q waves in the ECG
d) Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
e) Intracoronary thrombus by angiography or autopsy.

2. Cardiac death (SCD) with symptoms of myocardial ischemia & new ischemic ECG changes, or new LBBB, but death occurred before cardiac biomarkers were obtained, or their levels increased.

3. PCI–related MI: cTn ↑ (5 x 99th percentile URL) when baseline value is normal, or 20% ↑ if baseline values are elevated & are stable or falling. In addition: (i) myocardial ischemia symptoms or (ii) new ischemia by ECG or (iii) angiography consistent with procedural complication or (iv) imaging demonstrating new loss of viable myocardium or new regional wall motion abnormality.

4. Stent thrombosis associated with MI detected by angiography or autopsy in the setting of ischemia & with ↑ or ↓ of cardiac biomarkers, with at least one value > 99th percentile URL.

5. CABG–related MI: ↑ of cardiac biomarker values (10 x 99th percentile URL) in patients with normal baseline cTn (99th percentile URL). In addition: (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

CRITERIA FOR PRIOR MYOCARDIAL INFARCTION

Any one of the following criteria meets the diagnosis for prior myocardial infarction:

1. Pathological Q waves with or without symptoms in the absence of non-ischemic causes.

2. Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a non-ischemic cause.

3. Pathological findings of a prior MI.

Table 1. Elevations of Cardiac Troponin Values Because of Myocardial Injury

1. Primary myocardial ischemia
a) Plaque rupture
b) Coronary thrombus

2. Supply/demand imbalance
a) Tachy-/brady-arrhythmias
b) Aortic dissection/severe AV disease
c) Hypertrophic cardiomyopathy
d) Cardiogenic, hypovolemic, septic shock
e) Severe respiratory failure
f) Severe anemia
g) Hypertension with or without LVH
h) Coronary spasm
i) Coronary embolism or vasculitis
j) Coronary endothelial dysfunction

3. Injury not related to myocardial ischemia
a) Contusion, surgery, ablation, pacing, ICD
b) Rhabdo with cardiac involvement
c) Myocarditis
d) Cardiotoxins (anthracyclines, herceptin)

4. Multifactorial/indeterminate injury
a) Heart failure
b) Stress (Takotsubo) cardiomyopathy
c) Severe PTE or pulmonary hypertension
d) Sepsis and critically ill patients
e) Renal failure
f) Neurological disease (CVA, hemorrhage)
g) Infiltrative diseases (amyloid, sarcoid)
h) Strenuous exercise

Table 2. Universal Classification of MI

Type 1
Spontaneous MI: plaque rupture, ulceration, fissure, erosion, or dissection with resulting intraluminal thrombus, decreased myocardial blood flow or distal platelet emboli, causing myocyte necrosis.

Type 2
MI due to ischemic imbalance: coronary endothelial dysfunction, coronary spasm or embolism, tachy/ brady-arrhythmias, anemia, respiratory failure, hypotension, hypertension with/without LVH.

Type 3
MI causing SCD when biomarkers are unavailable: SCD + symptoms suggestive of ischemia & presumed new ischemic ECG changes/LBBB; death occurring before a rise in biomarkers could be documented.

Type 4a
MI related to PCI: cTn ↑ (5 x 99th percentile URL) when baseline value is normal, or 20% ↑ if baseline values are elevated & are stable or falling. Plus symptom/ECG/angiography/imaging evidence.

Type 4b
MI related to stent thrombosis: by angio or autopsy; in setting of ischemia & with ↑ or ↓ of biomarkers, with at least one value > 99th percentile URL

Type 5
MI related to CABG: ↑ of biomarkers (10 x 99th percentile URL) with normal baseline cTn (99th percentile URL). Plus ECG/angio/imaging evidence.

Table 3. ECG Manifestations of Acute Myocardial Ischemia (in Absence of LVH and LBBB)

ST elevation
New ST elevation at the J-point in 2 contiguous leads with the cut-off points: ≥ 0.1 mV in all leads other than leads V2–V3 where the following cut points apply: ≥ 0.2 mV in men ≥ 40 years; ≥ 0.25 mV in men < 40 years, or ≥ 0.15 mV in women.

ST depression and T-wave changes
New horizontal or down-sloping ST depression ≥ 0.05 mV in 2 contiguous leads; and/or T inversion ≥ 0.1 mV in two contiguous leads with prominent R-wave or R/S ratio > 1

Table 4. ECG Changes Associated With Prior Myocardial Infarction

1. Any Q-wave in leads V2–V3 ≥ 0.02 sec or QS complex in leads V2 and V3
2. Q-wave ≥ 0.03 sec and ≥ 0.1 mV deep or QS complex in leads I, II, aVL, aVF, or V4–V6 in any two leads of a contiguous lead grouping (I, aVL; V1–V6; II, III, and aVF)*
3. R-wave ≥ 0.04 s in V1–V2 and R/S ≥ 1 with a concordant positive T-wave in the absence of conduction defect

**Same criteria are used for supplemental leads V7-V9.*

Table 5. Common ECG Pitfalls in Diagnosing Myocardial Infarction

False positives

1. Early repol; LBBB; Pre-excitation; or J-point elevation syndromes (e.g. Brugada syndrome)
2. Peri-/myocarditis; Pulmonary embolism; Subarachnoid hemorrhage
3. Metabolic disturbances such as hyperkalemia
4. Cardiomegaly; Lead transposition; Cholecystitis; Persistent juvenile pattern
5. Malposition of precordial ECG leads
6. Tricyclic antidepressants or phenothiazines

False negatives

1. Prior MI with Q-waves and/or persistent ST elevation
2. RV pacing. 3. LBBB

ACCF 2012 Consensus on Practical Clinical Considerations in Troponin Elevation Interpretation

Figure 1. Conceptual Model for Clinical Distribution of Elevated Troponin

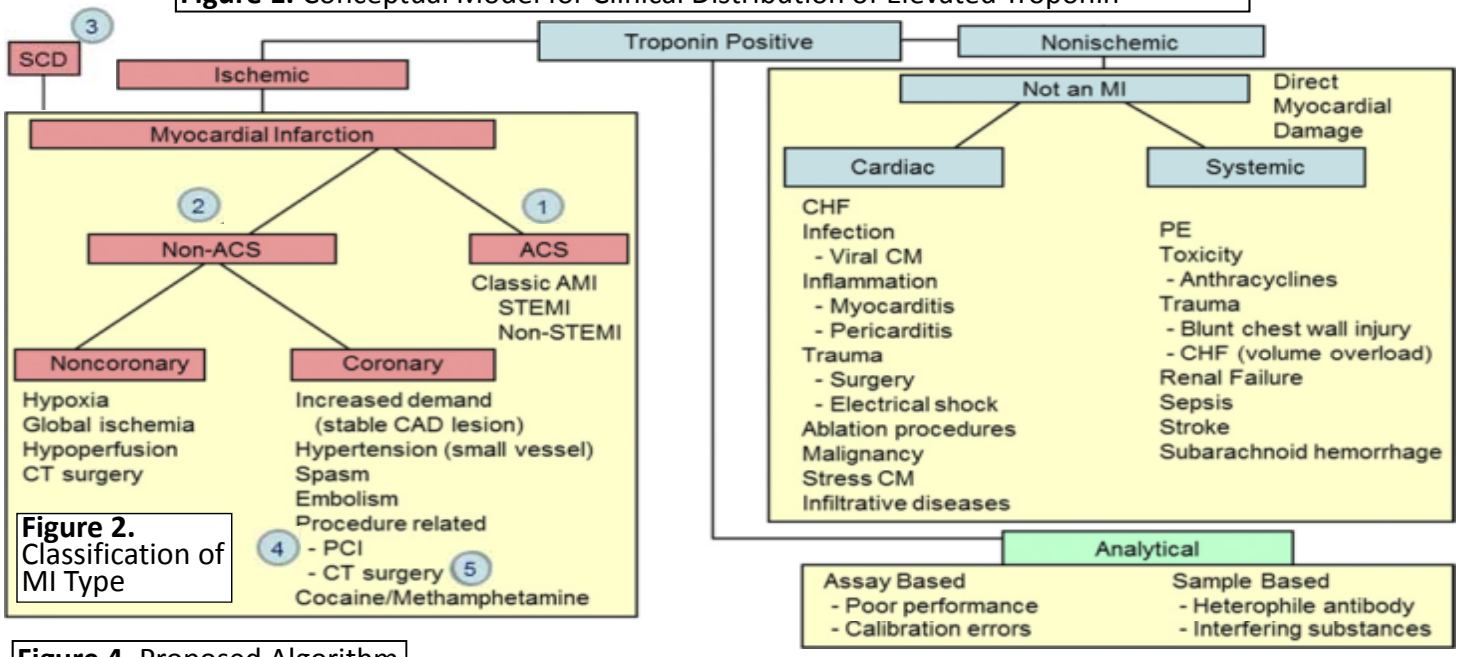
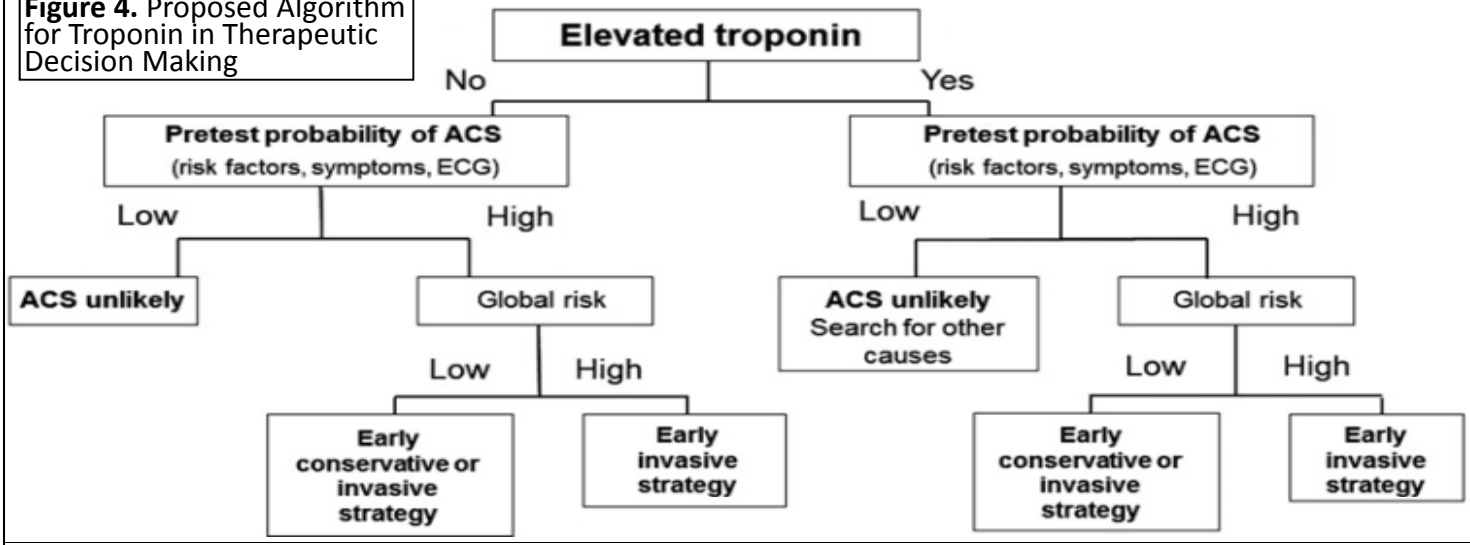


Figure 2. Classification of MI Type

Figure 4. Proposed Algorithm for Troponin in Therapeutic Decision Making



Global risk should be estimated via formal clinical risk scores (TIMI, GRACE, or PURSUIT) or a combination of the following high-risk features: recurrent angina/ischemia at rest or low-level activity, heart failure or worsening mitral regurgitation, high-risk stress test, hemodynamic instability, sustained VT, DM, PCI within 6 months, prior CABG or LVEF < 0.40.