The classification of acute myocardial infarction type 2 - a knotty problem

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Faculty Disclosure

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The nature of the conflicts are:

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Classification of Myocardial Infarction

Type 1  
Spontaneous myocardial infarction related to ischemia due to a primary coronary event such as plaque erosion or rupture, fissuring or dissection

Type 2  
Myocardial infarction secondary to ischemia due to imbalance between oxygen demand and supply e.g. coronary spasm, anemia, or hypotension

Type 3  
Sudden cardiac death with symptoms of ischemia, accompanied by new ST elevation or LBBB, or verified coronary thrombus by angiography or autopsy, but death occurring before blood samples could be obtained

Type 4a  
Myocardial infarction associated with PCI

Type 4b  
Myocardial infarction associated with verified stent thrombosis

Type 5  
Myocardial infarction associated with CABG

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Type 2 Myocardial Infarction

Myocardial infarction secondary to ischemia due to either increased oxygen demand or decreased supply e.g. spasm, anemia, arrhythmia, or hypotension
Criteria for Acute Myocardial Infarction Type 1 and Type 2

Detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit together with evidence of ischaemia with at least one of the following:

- Symptoms of ischemia
- ECG changes of new ischaemia (new ST-T changes or new LBBB)
- Development of pathological Q waves in the ECG
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

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Type 2 Myocardial Infarction

- Of the 5 subtypes of MI in the revised 2007 universal definition of MI, the type 2 MI has proven to be the most difficult to interpret and therefore to implement.

- The problem arises from the fact that multiple clinical conditions can cause myocyte necrosis and lead to abnormal elevations in blood troponin levels.

- Also the criteria for ischaemia may not be fulfilled.

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Type 2 Myocardial Infarction

- The task force envisioned two situations that might lead to myocardial ischaemia severe enough to result in myocardial necrosis:

1. Conditions that would decrease myocardial oxygen supply

2. Conditions that would increase myocardial oxygen demand
Conditions leading to decreased myocardial oxygen supply

- Severe anaemia
- Respiratory failure with severe hypoxemia
- Bradycardia leading to hypotension
- Hypotension or shock
- Transient coronary vasospasm or marked endothelial dysfunction
- Coronary artery embolism
Conditions leading to increased myocardial oxygen demand

- Tachyarrhythmia's, supraventricular or ventricular in origin
- Severe hypertension in a patient with left ventricular hypertrophy with a resultant marked increase in myocardial oxygen demand
Not Type 2 Myocardial Infarction

- Other causes of elevated troponins
- Myocyte necrosis 2° tachycardia without clinical ischaemia
- Apical ballooning syndrome
- Marathon runners
Elevations of Troponin in the Absence of Overt Ischemic Heart Disease

- Cardiac contusion, or other trauma including surgery, ablation, pacing etc
- Congestive heart failure – acute and chronic
- Aortic dissection, aortic valve disease
- Hypertrophic cardiomyopathy
- Tachy- or bradyarrhythmias, or heart block
- Apical ballooning syndrome
- Rhabdomyolysis with cardiac injury
- Pulmonary embolism, severe pulmonary hypertension

French, JK; White HD; Heart 2004
Elevations of Troponin in the Absence of Overt Ischemic Heart Disease

- Renal failure
- Acute neurological disease, including stroke, or subarachnoid hemorrhage
- Infiltrative diseases, e.g., amyloidosis, hemochromotosis, sarcoidosis or scleroderma
- Inflammatory diseases, e.g., myo/pericarditis or myocardial extension of endocarditis
- Drug toxicity or toxins
- Critically ill patients, especially with respiratory failure, or sepsis
- Burns, especially if affecting > 30% of body surface area
- Extreme exertion
Type 2 Myocardial Infarction: considerations

It is often unclear whether patients with conditions that decrease myocardial oxygen supply or increase myocardial oxygen demand have underlying coronary artery disease.
Type 2 Myocardial Infarction

Consider the following patients with elevated troponin levels:

- A 14 year old female with WPW with 5 hours of SVT at a heart rate of 240 bpm, troponins rise and fall and she has myocyte necrosis. She doesn’t have clinical ischaemia ie no ischaemic chest discomfort and no ischaemic ECG changes.
- The diagnosis is myocyte necrosis secondary to tachycardia
Type 2 Myocardial Infarction

- A 70 year old male in ICU with respiratory failure, hemoglobin of 7.3 g/dL, arterial pO₂ of 45 mm Hg, systolic blood pressure of 65 mm Hg, and a history of an inferior wall MI two years ago. The troponins rise and fall and there is new 0.5 mm ST depression.

- The diagnosis is type 2 MI, but could also be type 1.

- Is the history of MI helpful in making the diagnosis of type 1 or type 2 MI?
Type 2 Myocardial Infarction: Considerations

• Should ischaemia be required (ischaemic chest discomfort and or ischaemic ECG changes) or could it be inferred?

• Should the presence of coronary artery disease be required?

• Should the term myocardial injury be used if the Universal Definition for MI is not fulfilled?

• Should the term be myocyte necrosis?
Type 2 Myocardial Infarction: Considerations

- Myocyte necrosis due to clinical ischaemia (ischemic symptoms or ECG changes) in a coronary artery territory
- MRI scar imaging can help
Perioperative Myocardial Infarction

- Type 1 due to plaque fissuring or rupture
- Type 2 due to imbalance in supply and demand
Unstable coronary plaques

- Sympathetic hyperactivity (increased plasma catecholamines)
- Hemodynamic instability (tachycardia / hypertension)
- Coronary vasoconstriction

- Plaque rupture
  - ↑ Coagulability
  - ↓ Fibrinolysis
  - Recent PCI with stent
  - premature cessation of dual antiplatelet therapy

- Plaque erosion
  - ↓ Fibrinolysis
  - Recent PCI with stent

- Acute coronary thrombosis

- ACS – Type I MI

Landesberg G. Circulation 2009;119:2936-44
Severe, yet stable CAD: Type 2 Myocardial Infarction

- ↑ Myocardial O₂ demand
- ↑ Heart rate / arrhythmia
- ↑ Myocardial wall stress

Sympathetic hyperactivity
- Postoperative pain
- Withdrawal of β-blockers
- Hypovolemia
- Cardiac decompensation
- Systemic vasodilatation
- ↑ Blood pressure
- Hypervolemia/
- ↑ LVEDP
- Pulmonary congestion/Atelectasis

Subendocardial O₂ supply
- ↑ Heart rate / arrhythmia

- Hypotension
- ↑ Myocardial wall stress
- Coronary vasoconstriction
- Anemia
- Hypoxemia

Prolonged ST-depression ischemia >> Type II MI

Landesberg G. Circulation 2009;119:2936-44
Probability of type 1 and 2 as a function of CAD severity

Landesberg G. Circulation 2009;119:2936-44
Progression of Atherosclerosis and Troponin values

- Minimal or no disease: troponin detectable with hsTroponins
- Significant structural disease: troponin higher than 99th %

ACS and other acute situations - rising troponin values

Modified from Libby P Circ 104:365,2001
Use of high sensitivity troponin T to diagnose myocardial infarction

Clinical setting consistent with myocardial ischaemia

Baseline

- **< 14 ng/L**
  - Retest hsTnT 6 hours after symptom onset or if timing of symptom onset is unclear at 6 hours after presentation
  - Change < 50%: Adverse Prognosis
  - Change ≥ 50%: Myocardial infarction
- **≥ 14 - 52 ng/L**
  - Retest hsTnT 3 hours later
  - Change < 50%: Adverse Prognosis
  - Change ≥ 50%: Myocardial infarction
- **≥ 53 ng/L**
  - Retest hsTnT 3 hours later
  - Change < 20%: Adverse Prognosis
  - Change ≥ 20%: Myocardial infarction

- ≤14ng/L rules out MI with >90% probability
- If ≥14ng/L then proceed to middle part of algorithm.

Evidence based treatments

White HD; AHJ 2010
Type 2 Myocardial Infarction: Considerations

- The Universal Task Force continues to discuss these issues
- Heart Failure Group
- Potential for other groups e.g.: tachyarrhythmia's, atrial fibrillation
Type 2 Myocardial Infarction

Careful thought and expert clinical judgement is needed to make the diagnosis of type 2 myocardial infarction.