Updates

Extract from expert reports

From the Editorial desk

Updates

Fourth universal definition of myocardial infarction (2018)

European Society of Cardiology

Myocardial infarction type 1

MI caused by atherothrombotic coronary artery disease (CAD) and usually precipitated by atherosclerotic plaque disruption (rupture or erosion) is designated as a type 1 MI.

Criteria for type 1 MI

Detection of a rise and/or fall of cTn values with at least one value above the 99th percentile 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;
- Identification of a coronary thrombus by angiography including intracoronary imaging or by autopsy.

Myocardial infarction type 2

The pathophysiological mechanism leading to ischaemic myocardial injury in the context of a mismatch between oxygen supply and demand has been classified as type 2 MI. By definition, acute atherothrombotic plaque disruption is not a feature of type 2 MI. In patients with stable known or presumed CAD, an acute stressor such as an acute gastrointestinal bleed with a precipitous drop in haemoglobin, or a sustained tachyarrhythmia with clinical manifestations of myocardial ischaemia, may result in myocardial injury and a type 2 MI.

Myocardial infarction type 3

The detection of cardiac biomarkers in the blood is fundamental for establishing the diagnosis of MI. However, patients can manifest a typical presentation of myocardial ischaemia/infarction, including presumed new ischaemic ECG changes or ventricular fibrillation, and die before it is possible to obtain blood for cardiac biomarker determination; or the patient may succumb soon after the onset of symptoms before an elevation of biomarker values has occurred. Such patients are designated as having a type 3 MI, when suspicion for an acute myocardial ischaemic event is high, even when cardiac biomarker evidence of MI is lacking.

Myocardial infarction associated with percutaneous coronary intervention (type 4a myocardial infarction)

Stand-alone post-procedural increases of cTn values are sufficient to establish a diagnosis of procedural myocardial injury but not for the diagnosis of type 4a MI. Type 4a MI requires an elevation of cTn values greater than five times the 99th percentile URL in patients with normal baseline values or, in patients with elevated pre-procedure cTn in whom the cTn levels are stable (<_ 20% variation) or falling, the post-procedure cTn must rise > 20% to an absolute value more than five times the 99th percentile URL. In addition, there should be evidence of new myocardial ischaemia, either from ECG changes, imaging evidence, or from procedure-related complications associated with reduced coronary blood flow such as coronary dissection, occlusion of a major epicardial artery or a side branch occlusion/ thrombus, disruption of collateral flow, slow flow or no-reflow, or distal embolization.

Stent/scaffold thrombosis associated with percutaneous coronary intervention (type 4b myocardial infarction)

A subcategory of PCI-related MI is stent/scaffold thrombosis, type 4b MI, as documented by angiography or autopsy using the same criteria utilized for type 1 MI. It is important to indicate the time of the occurrence of the stent/scaffold thrombosis in relation to the timing of the PCI procedure. The following temporal categories are suggested: acute, 0–24 h; subacute, > 24 h to 30 days; late > 30 days to 1 year; and very late > 1 year after stent/scaffold implantation.

Restenosis associated with percutaneous coronary intervention (type 4c myocardial infarction)

Occasionally MI occurs and is the only angiographic explanation since no other culprit lesion or thrombus can be identified. This PCI-related MI type is designated as type 4c MI, defined as focal or diffuse restenosis, or a complex lesion associated with a rise and/or fall of cTn values above the 99th percentile URL applying, the same criteria utilized for type 1 MI.

Myocardial infarction associated with coronary artery bypass grafting (type 5 myocardial infarction)

Numerous factors can lead to procedural myocardial injury during a CABG procedure. Many of them are related to the details of cardiac preservation, the extent of the direct traumatic injury to the myocardium, as well as any potential ischaemic injury. For that reason, increases in cTn values should be expected after all CABG procedures, which need to be taken into account when comparing the extent of procedural myocardial injury after cardiac surgery with that associated with less invasive approaches.

Myocardial injury and infarction associated with non-cardiac procedures

Perioperative MI is one of the most important complications in major non-cardiac surgery and it is associated with a poor prognosis. Most patients who have a perioperative MI will not experience ischaemic symptoms due to anaesthesia, sedation, or pain relieving medications. Nevertheless, asymptomatic perioperative MI is as strongly associated with 30 day mortality as symptomatic MI. Knowledge about hs-cTn values at baseline can help to identify patients having chronic cTn elevation before surgery, as well as those at increased risk during and after the procedure. Measurement of hs-cTn in post-operative samples reveals that as many as 35% of patients have levels above the 99th percentile URL, and 17% have an elevation and a rising pattern of values indicative of evolving myocardial injury. Those with a rising pattern of elevated hs-cTn values are at particular risk; the greater the rise, the greater the risk.

Myocardial injury or infarction associated with heart failure

Depending on the assay used, detectable to clearly elevated cTn values being indicative of myocardial injury may be seen in patients with heart failure (HF), both with reduced ejection fraction (EF) and with preserved EF. Using hs-cTn assays, measurable hs-cTn concentrations may be present in nearly all patients with HF, with a significant percentage exceeding the 99th percentile URL, particularly in those patients with more severe HF syndromes, such as in acutely decompensated HF.

Takotsubo syndrome

Takotsubo syndrome (TTS) can mimic MI and is found in 1 – 2% of patients presenting with suspected STEMI. The onset of TTS is often triggered by intense emotional or physical stresses, such as bereavement. Over 90% of patients are post-menopausal women. Cardiovascular complications occur in 50% of patients presenting with TTS, and the inpatient mortality is similar to STEMI (4-5%) due to cardiogenic shock, ventricular rupture, or malignant arrhythmias.

TTS usually presents similar to ACS. ST-segment elevation is frequent (44%), but the extent of the ST-segment elevation is usually widespread across the lateral and precordial leads, beyond that of a single coronary artery distribution. ST-segment depression occurs in < 10% of patients and after 12-24 h, deep, symmetric T wave inversion and QTc prolongation are typically observed.

There are usually transient elevations in cTn levels (> 95% of cases), but the peak cTn values observed are modest, and contrast with the large territory of ECG changes or left ventricular (LV) dysfunction. The rise and fall in cTn levels support an acute myocardial injury, secondary to the high catecholamine surges that are known to trigger cTn release from cardiomyocytes.

Myocardial infarction with non-obstructive coronary arteries

It is increasingly recognized that there is a group of MI patients with no angiographic obstructive CAD (≥ 50% diameter stenosis in a major epicardial vessel), and the term myocardial infarction with non-obstructive coronary arteries (MINOCA) has been coined for this entity. The diagnosis of MINOCA, like the diagnosis of MI, indicates that there is an ischaemic mechanism responsible for the myocyte injury (i.e. non-ischaemic causes such as myocarditis have been excluded). The prevalence of MINOCA is estimated to be 6 – 8% among patients diagnosed with MI and more common in women than men.

Atherosclerotic plaque disruption and coronary thrombosis may be a cause of MINOCA, i.e. type 1 MI. However, coronary spasm and spontaneous coronary dissection may be involved.

Myocardial injury and/or infarction associated with kidney disease

Many patients with chronic kidney disease (CKD) have elevation of cTn values. With hs-cTn assays, the majority of patients with end-stage renal disease will have elevation of hs-cTn values above the 99th percentile URL. This is particularly the case for hs-cTnT, which is more often elevated compared with hs-cTnI. It has been shown using hs-cTn assays that renal dysfunction is commonly associated with cardiovascular abnormalities

Myocardial injury and/or infarction in critically ill patients

Elevations of cTn values are common in patients in the intensive care unit and are associated with adverse prognosis regardless of the underlying disease state. Some elevation of cTn values may reflect type 2 MI due to underlying CAD and increased myocardial oxygen demand, whereas in other patients, type 1 MI may occur because of plaque disruption leading to thrombosis in a coronary artery. However, other patients may have elevated cTn values and marked decreases in EF due to sepsis caused by endotoxin, with myocardial function recovering completely with normal EF once the sepsis is treated.

Atrial fibrillation

In patients with atrial fibrillation and rapid ventricular rate or paroxysmal supraventricular tachycardia, ST-segment depression or T wave inversion may occur in the absence of CAD. The causes are not completely understood. Cardiac memory, an electrical remodeling phenomenon characterized by marked diffuse T wave inversions following periods of abnormal ventricular activation, which may also be caused by transient rate-related conduction disturbances or pacing, may explain these findings. In some patients, the tachycardia may result in an insufficient increase in coronary flow to match myocardial oxygen demand, resulting in cellular hypoxia and abnormal repolarization. For these reasons, a patient with new-onset atrial fibrillation, elevated baseline cTn concentration, and new ST-segment depression should not automatically be classified as type 2MI without additional information.

Imaging techniques for MI suggested are:

- Echocardiography
- Radionuclide imaging
- Cardiac magnetic resonance Imaging
- Computed tomographic coronary angiography

What's new in the universal definition of myocardial infarction?

• Differentiation of myocardial infarction from myocardial injury.
• Use of cardiovascular magnetic resonance to define aetiology of myocardial injury.
• Use of computed tomographic coronary angiography in suspected myocardial infarction.
• ST-segment elevation in lead aVR with specific repolarization patterns, as a STEMI equivalent.

Clinical criteria for MI

The clinical definition of MI denotes the presence of acute myocardial injury detected by abnormal cardiac biomarkers in the setting of evidence of acute myocardial ischaemia.

Criteria for myocardial injury

Detection of an elevated cTn value above the 99th percentile URL is defined as myocardial injury. The injury is considered acute if there is a rise and/or fall of cTn values.

Some facts of Brugada Syndrome

American College of Cardiology - Aug 22, 2018

Symptoms of Brugada syndrome include syncope, seizures, nocturnal agonal breathing, or SCD. Lethal arrhythmias often occur during rest or sleep but may occur with fever. Symptoms occur most often in adulthood with apparent influence from sex-related hormones.

Brugada ECG phenocopy may occur with right ventricular or right ventricular outflow tract pathology such as right ventricle ischemia, acute pulmonary embolism, or mechanical compression.

Brugada-like ECG patterns can occur with acute ischemia, left anterior descending artery occlusion, right bundle branch block, left ventricular hypertrophy, pectus excavatum, or arrhythmogenic cardiomyopathy.

Modulating factors such as bradycardia, vagal tone, fever (particularly in children), and certain medications can unmask or exacerbate Brugada syndrome.