PATHOGENESIS, SYMPTOMS, AND DIAGNOSTIC METHODS OF MYOCARDIAL INFARCTION

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Annotation: Acute myocardial infarctions are one of the leading causes of death in the developed world, with prevalence approaching three million people worldwide, with more than one million deaths in the United States annually. This statistic reviews the presentation, evaluation, and management of patients with acute myocardial infarctions and highlights the role of the interprofessional team in caring for these patients.

Key words: Myocardial infarction, heart attack, cardiac troponin, electrocardiogram, upper reference limit.

A myocardial infarction (MI), commonly known as a heart attack, occurs when blood flow decreases or stops to the coronary artery of the heart, causing damage to the heart muscle.[1]

Myocardial infarction is defined as sudden ischemic death of myocardial tissue. In the clinical context, myocardial infarction is usually due to thrombotic occlusion of a coronary vessel caused by rupture of a vulnerable plaque. Ischemia induces profound metabolic and ionic perturbations in the affected myocardium and causes rapid depression of systolic function. Prolonged myocardial ischemia activates a “wavefront” of cardiomyocyte death that extends from the subendocardium to the subepicardium. Mitochondrial alterations are prominently involved in apoptosis and necrosis of cardiomyocytes in the infarcted heart. The adult mammalian heart has negligible regenerative capacity, thus the infarcted myocardium heals through formation of a scar. Infarct healing is dependent on an inflammatory cascade, triggered by alarmins released by dying cells. Clearance of dead cells and matrix debris by infiltrating phagocytes activates anti-inflammatory pathways leading to suppression of cytokine and chemokine signaling. Activation of the renin-angiotensin-aldosterone system and release of transforming growth factor-β induce conversion of fibroblasts into myofibroblasts, promoting deposition of extracellular matrix proteins. Infarct healing is intertwined with geometric remodeling of the chamber, characterized by dilation, hypertrophy of viable segments, and progressive dysfunction. [2]

Risk factors

Most MIs occur due to coronary artery disease.[3]

Risk factors include :

high blood pressure,

smoking,
diabetes,
lack of exercise,
obesity,
high blood cholesterol,

poor diet and excessive alcohol intake. The complete blockage of a coronary artery caused by a rupture of an atherosclerotic plaque is usually the underlying mechanism of an MI.[3] MIs are less commonly caused by coronary artery spasms, which may be due to cocaine, significant emotional stress (commonly known as Takotsubo syndrome or broken heart syndrome) and extreme cold, among others.[4] A number of tests are useful to help with diagnosis, including electrocardiograms (ECGs), blood tests and coronary angiography. An ECG, which is a recording of the heart's electrical activity, may confirm an **ST elevation MI** (STEMI), if ST elevation is present. Commonly used blood tests include troponin and less often creatine kinase MB.

Symptoms

Symptoms of a heart attack vary. Some people have mild symptoms. Others have severe symptoms. Some people have no symptoms.

Common heart attack symptoms include:

- Chest pain that may feel like pressure, tightness, pain, squeezing or aching
- Pain or discomfort that spreads to the shoulder, arm, back, neck, jaw, teeth or sometimes the upper belly
- Cold sweat
- Fatigue
- Heartburn or indigestion
- Lightheadedness or sudden dizziness
- Nausea
- Shortness of breath

Women may have atypical symptoms such as brief or sharp pain felt in the neck, arm or back. Sometimes, the first symptom sign of a heart attack is sudden cardiac arrest.

Some heart attacks strike suddenly. But many people have warning signs and symptoms hours, days or weeks in advance. Chest pain or pressure (angina) that keeps happening and doesn't go away with rest may be an early warning sign. Angina is caused by a temporary decrease in blood flow to the heart.

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.

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.[a]
- cTn = cardiac troponin; ECG = electrocardiogram; URL = upper reference limit.
- [a]Post-mortem demonstration of an atherothrombus in the artery supplying the infarcted myocardium, or a macroscopically large circumscribed area of necrosis with or without intramyocardial haemorrhage, meets the type 1 MI criteria regardless of cTn values.

Criteria for type 2 MI

Detection of a rise and/or fall of cTn values with at least one value above the 99th percentile URL, and evidence of an imbalance between myocardial oxygen supply and demand unrelated to acute coronary athero-thrombosis, requiring 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

- **Criteria for type 3 MI**
- Patients who suffer cardiac death, with symptoms suggestive of myocardial ischaemia accompanied by presumed new ischaemic ECG changes or ventricular fibrillation, but die before blood samples for biomarkers can be obtained, or before increases in cardiac biomarkers can be identified, or MI is detected by autopsy examination.
- **Criteria for cardiac procedural myocardial injury**
- Cardiac procedural myocardial injury is arbitrarily defined by increases of cTn values (> 99th percentile URL) in patients with normal baseline values (≤ 99th

percentile URL) or a rise of cTn values > 20% of the baseline value when it is above the 99th percentile URL but it is stable or falling.

Criteria for PCI-related MI ≤ 48 h after the index procedure (type 4a MI)

Coronary intervention-related MI is arbitrarily defined by an elevation of cTn values more than five times the 99th percentile URL in patients with normal baseline values. In patients with elevated pre-procedure cTn in whom the cTn level are stable (≤ 20% variation) or falling, the post-procedure cTn must rise by > 20%. However, the absolute post-procedural value must still be at least five times the 99th percentile URL. In addition, one of the following elements is required:

- New ischaemic ECG changes;
- Development of new pathological Q waves;[a]
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology;
- Angiographic findings consistent with a procedural flow-limiting complication such as coronary dissection, occlusion of a major epicardial artery or a side branch occlusion/thrombus, disruption of collateral flow, or distal embolization.[b]

[a]Isolated development of new pathological Q waves meets the type 4a MI criteria if cTn values are elevated and rising but less than five times the 99th percentile URL.

[b]Post-mortem demonstration of a procedure-related thrombus in the culprit artery, or a macroscopically large circumscribed area of necrosis with or without intra-myocardial haemorrhage meets the type 4a MI criteria.

Criteria for CABG-related MI ≤ 48 h after the index procedure (type 5 MI)

CABG-related MI is arbitrarily defined as elevation of cTn values > 10 times the 99th percentile URL in patients with normal baseline cTn values. In patients with elevated pre-procedure cTn in whom cTn levels are stable (≤ 20% variation) or falling, the post-procedure cTn must rise by > 20%. However, the absolute post-procedural value still must be > 10 times the 99th percentile URL. In addition, one of the following elements is required:

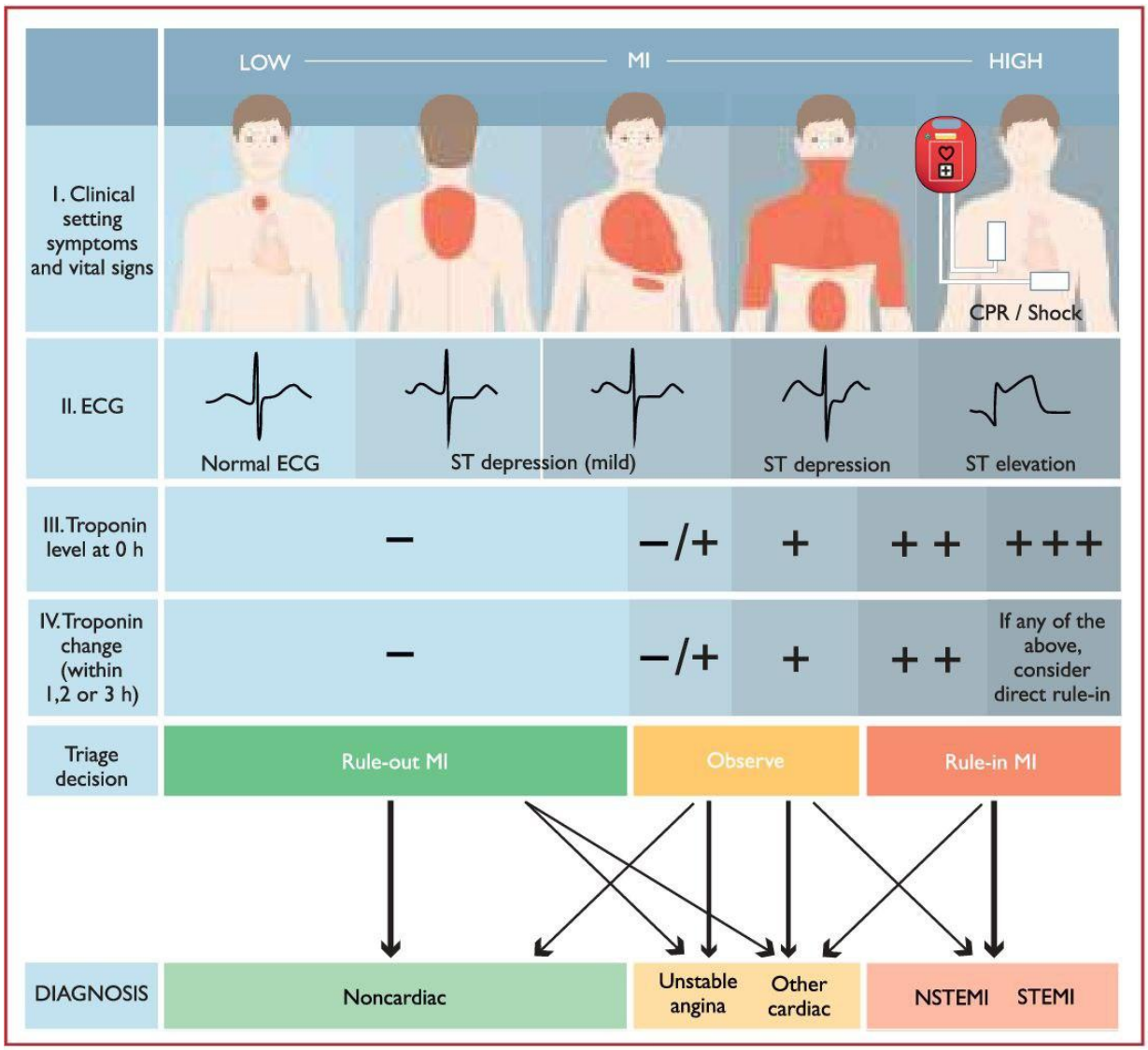
- Development of new pathological Q waves;[a]
- Angiographic documented new graft occlusion or new native coronary artery occlusion;
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic aetiology.

[a]Isolated development of new pathological Q waves meets the type 5 MI criteria if cTn values are elevated and rising but < 10 times the 99th percentile URL.[5]

How is acute MI diagnosed?

The diagnosis is secured when there is a rise and/or fall of troponin (high sensitivity assays are preferred) along with supportive evidence in the form of typical

symptoms, suggestive electrocardiographic (ECG) changes, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.



The gold standard for diagnosing myocardial infarction has been the World Health Organization definition, which requires any 2 of 3 criteria: ischemic symptoms, electrocardiographic changes, and elevated creatine kinase-MB levels. Recently, the American College of Cardiology and the European Society of Cardiology published a new definition that for the first time includes elevated troponin levels. (See Eur Heart J 2000; 21:1502.) The new criteria are elevated troponin or CK-MB levels and either ischemic symptoms or electrocardiographic changes. These authors evaluated the clinical implications of the new definition.[6]

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