Diagnosis of acute myocardial infarction

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INTRODUCTION

Myocardial infarction (MI) is defined as a clinical (or pathologic) event in the setting of myocardial ischemia in which there is evidence of myocardial injury [1,2]. 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.

A related issue is the evaluation of a patient who presents with chest pain suggestive of an acute coronary syndrome in whom the initial evaluation (ECG, cardiac enzymes) is not diagnostic. This issue is discussed separately. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Observation'.)

A discussion of the diagnosis and management of patients with suspected or documented MI during the coronavirus 2019 (COVID-19) pandemic is found elsewhere. (See "COVID-19: Myocardial infarction and other coronary artery disease issues", section on 'Acute coronary syndrome patients'.)

DEFINITIONS

This section defines the terms acute coronary syndrome (ACS), acute MI, ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina (UA). The term ACS is applied to patients in whom there is a suspicion or confirmation of myocardial ischemia. There are three types of ACS: STEMI, NSTEMI, and UA. The first two are characterized by a typical rise and/or fall in troponin with at least one value >99th percent upper reference limit (URL) [3].

UA is considered to be present in patients with ischemic symptoms suggestive of an ACS without elevation in biomarkers with or without ECG changes indicative of ischemia [4]. (See 'Troponin' below.)

UA and NSTEMI are frequently indistinguishable at initial evaluation. ST-segment and/or T wave ECG changes are often persistent in NSTEMI, while, if they occur in UA, they are usually transient. Regardless of the category, ST-segment change defines a higher-risk group [5]. (See "Acute coronary syndrome: Terminology and classification".)

Joint Task Force definitions — The Joint Task Force of the European Society of Cardiology, American College of Cardiology Foundation, the American Heart Association, and the World Heart Federation defined acute MI (2018) as the presence of acute myocardial injury detected by abnormal cardiac biomarkers in the setting of evidence of acute myocardial ischemia [2].

The joint task force refines the definition of MI by developing a clinical classification according to the assumed proximate cause of the myocardial ischemia:

- **Type 1** – MI caused by acute atherothrombotic coronary artery disease and usually precipitated by atherosclerotic plaque disruption (rupture or erosion).
- **Type 2** – MI consequent to a mismatch between oxygen supply and demand. This includes a multiplicity of potential mechanisms including coronary dissection, vasospasm, emboli, microvascular dysfunction, as well as increases in demand with or without underlying coronary artery disease.
- **Type 3** – Patients with a typical presentation of myocardial ischemia/infarction, such as presumed new ischemic ECG changes or ventricular fibrillation, with unexpected death before blood samples for biomarkers could be drawn or before their appearance in the blood.
- **Type 4a** – MI associated with percutaneous coronary intervention (PCI) is arbitrarily defined. Type 4a MI requires an elevation of cardiac troponin (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 percent variation) or falling, the post-procedure cTn must rise >20 percent to an absolute value more than five times the 99th percentile URL. In addition, there should be evidence of new myocardial ischemia, 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.

- **Type 4b** – A subcategory of PCI-related MI is stent/scaffold thrombosis. This is documented by angiography or autopsy using the same criteria utilized for type 1 MI.

- **Type 5** – Coronary artery bypass graft surgery (CABG) MI is defined as an 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 percent variation) or falling, the post-procedure cTn must rise by >20 percent. However, the absolute postprocedural 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.

  - 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 ischemic etiology.

After revascularization — The Fourth Universal Definition of MI in the setting of PCI or CABG are presented above. (See 'Definitions' above.)

Following revascularization with either CABG or PCI, troponin may rise. Transient elevations in troponins may represent necrosis, although the mechanism is unknown. Higher elevations are associated with worse prognosis after CABG. MI following PCI or CABG is discussed elsewhere. (See "Periprocedural myonecrosis following percutaneous coronary intervention" and "Troponin testing: Clinical use", section on 'Coronary artery revascularization'.)

Prior MI — According to the Fourth Universal Definition, any one of the following three criteria satisfies the diagnosis for a prior or silent/unrecognized MI [2]:

- Abnormal Q waves with or without symptoms in the absence of nonischemic causes.

- Imaging evidence of loss of viable myocardium in a pattern consistent with ischemic etiology. (See "Role of echocardiography in acute myocardial infarction", section on

'Indications for echocardiography in MI'.)

- Pathoanatomical findings of a prior MI.

MI after noncardiac surgery — Myocardial injury or infarction occurs frequently after noncardiac surgery in patients at increased risk [6]. This issue is discussed elsewhere. (See "Perioperative myocardial infarction or injury after noncardiac surgery" and "Perioperative myocardial infarction or injury after noncardiac surgery", section on 'Diagnosis'.)

WHEN TO SUSPECT ACUTE MI

Outcomes in patients with acute MI are significantly improved with very early treatment. Thus, early suspicion of the diagnosis is central to patient management. Every patient who presents for evaluation with chest pain, shortness of breath, new heart failure, sudden cardiac arrest, or new changes on an ECG should have the diagnosis considered. If another diagnosis is more likely, then an evaluation specific for MI can be altered. (See 'Differential diagnosis' below.)

CLINICAL MANIFESTATIONS

For a patient presenting with a suspected acute MI, an abbreviated history and physical examination, an ECG, and troponin should be obtained within 10 minutes of patient arrival [7]. This section will summarize important characteristics of the presentation of MI. A detailed discussion is found elsewhere. (See "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Clinical presentation' and "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Electrocardiogram assessment' and "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department", section on 'Physical examination'.)

History and physical examination — Patients with acute MI may present with chest pain or other symptoms of myocardial ischemia. In addition, some patients may present with sudden cardiac arrest. (See "Cardiac evaluation of the survivor of sudden cardiac arrest", section on 'Initial evaluation' and "Cardiac evaluation of the survivor of sudden cardiac arrest", section on 'Evaluation for structural heart disease'.)

Chest pain or discomfort is likely the most common symptom of acute MI. While chest pain is not required for the diagnosis of MI, its presence, particularly if characteristic for myocardial

ischemia, may influence decision making about the likelihood of the presence of MI (table 1).

The history should be targeted toward characteristics of chest pain: duration, character, similarity to possible previous episodes, provoking factors, and past history of coronary disease risk factors. Classic MI chest pain is chest tightness or pressure, in the substernal area, with radiation to the left arm or jaw. Associated symptoms include shortness of breath, diaphoresis, weakness, and anxiety. It should be noted, however, that in women, patients with diabetes, or in older individuals, these typical symptoms may not be present. Indeed, in these patients, atypical presentations are common. Aggravating and alleviating factors should be also investigated to distinguish cardiac versus noncardiac sources for the symptoms.

A full discussion of the characteristic of ischemic chest pain is found elsewhere. (See "Outpatient evaluation of the adult with chest pain".)

Among patients with chest pain characteristic of myocardial ischemia (angina pectoris), there are three primary presentations that suggest a change in the anginal pattern as acute coronary syndrome (ACS) as opposed to stable or exertional angina:

- Rest angina, which is usually more than 20 minutes in duration
- New onset angina that markedly limits physical activity
- Angina that is more frequent, longer in duration, or occurs with less exertion than previous angina

In a review of over 430,000 patients with confirmed acute MI from the National Registry of Myocardial Infarction 2, one-third had no chest pain on presentation to the hospital [8]. These patients may present with dyspnea alone, nausea and/or vomiting, palpitations, syncope, or cardiac arrest. As mentioned above, they are more likely to be older (74 versus 67 years), diabetic, and women. (See "Clinical features and diagnosis of coronary heart disease in women".)

The physical examination should include auscultation of the heart and lungs, measurement of blood pressure in both arms, checking the presence of all major pulses, and assessment for heart failure or circulatory compromise, which are associated with a high early mortality.

ECG — An early (target of 10 minutes after first medical contact) ECG is essential in the evaluation and diagnosis of patients with suspected ACS. It allows initial categorization of the patient with a suspected MI into one of three groups based on the pattern:

- STEMI (ST elevation or new left bundle branch block [LBBB]).

- Non-ST-elevation ACS, with either NSTEMI or unstable angina (UA; ST depression, T-wave inversions, or transient ST elevation).

- Undifferentiated chest pain syndrome (nondiagnostic ECG).

ST elevation — In patients with acute STEMI, the ECG evolves through a typical sequence. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction" and "ECG tutorial: Myocardial ischemia and infarction".)

Although not frequently seen, the earliest change in an STEMI is the development of a hyperacute or peaked T wave that reflects localized hyperkalemia. Thereafter, the ST segment elevates in the leads recording electrical activity of the involved region of the myocardium; it has the following appearance:

- Initially, there is elevation of the J point and the ST segment retains its concave configuration.

- With time, the ST-segment elevation becomes more pronounced and the ST segment becomes more convex or rounded upward.

- The ST segment may eventually become indistinguishable from the T wave; the QRS-T complex can actually resemble a monophasic action potential.

The joint European Society of Cardiology, American College of Cardiology Foundation, the American Heart Association, and the World Heart Federation (ESC/ACCF/AHA/WHF) committee for the definition of MI established specific ECG criteria for the diagnosis of ST-elevation MI [2]:

- New ST-segment elevation at the J-point in two contiguous leads, with the cut-points ≥0.1 mV in all leads other than leads V2 to V3.

- For leads V2 to V3: ≥2 mm in men ≥40 years; ≥2.5 mm in men <40 years, or ≥1.5 mm in women regardless of age.

Over time, there is further evolution of these ECG changes; the ST segment gradually returns to the isoelectric baseline, the R wave amplitude becomes markedly reduced, and the Q wave deepens. In addition, the T wave becomes inverted. These changes generally occur within the first two weeks after the event, but may progress more rapidly, within several hours of presentation.

The ECG can be used to localize the MI, and at times, predict the infarct-related artery. These issues are discussed separately. If there is ECG evidence of inferior wall ischemia (ST- or T-wave

changes in leads II, III, and aVF), the right-sided leads V4R, V5R, and V6R should also be obtained to evaluate the possibility of right ventricular infarction. Posterior leads (V7 to V9) are also indicated for those who present with ST-segment depression in the inferior leads to evaluate the possibility of posterior infarction [9]. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction".)

Although ST-segment elevation is consistent with acute MI (particularly if new), it can be seen in other disorders (table 2). As examples, ST-segment elevation can occur in myocarditis, acute pericarditis, patients with old MI and persistent ST-segment elevation often associated with wall motion abnormalities, and with the early repolarization variant. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction" and "Clinical manifestations and diagnosis of myocarditis in adults".)

In addition to patients with ST elevation on the ECG, two other groups of patients with an ACS may have an STEMI: those with new or presumably new LBBB and those with a true posterior MI. (See 'Bundle branch block or paced rhythm' below and "Electrocardiogram in the diagnosis of myocardial ischemia and infarction", section on 'Posterior wall MI'.)

Non-ST-elevation ECG abnormalities — A non-ST-elevation ACS is manifested by ST depressions and/or T-wave inversions without ST-segment elevations or pathologic Q waves. These ST-T wave abnormalities may be present diffusely in many leads; more commonly, they are localized to the leads associated with the region of ischemic myocardium. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction".)

As noted above, the two forms of non-ST-elevation acute coronary syndrome (UA and NSTEMI) are frequently indistinguishable at initial evaluation (prior to biomarker elevation). In a patient with an NSTEMI, ST-segment depressions usually evolve over the subsequent few days to result in residual ST-segment depression and T-wave inversions, but not to the formation of pathologic Q waves. In patients with UA, ST-segment and T-wave changes usually resolve completely.

The joint ESC/ACCF/AHA/WHF committee for the Fourth Universal definition of MI established specific ECG criteria for the diagnosis of NSTEMI [2]: new horizontal or downsloping ST-depression ≥0.5 mm in two contiguous leads and/or T inversion >1 mm in two contiguous leads with prominent R wave or R/S ratio >1.

Nondiagnostic initial ECG — The initial ECG is often not diagnostic in patients who are ultimately diagnosed with acute MI. In two series, for example, the initial ECG was not diagnostic in 45 percent and normal in 20 percent of patients subsequently shown to have an acute MI [10,11]. In patients clinically suspected of having an acute MI in whom the ECG is

nondiagnostic, it is recommended that the ECG should be repeated at 20- to 30-minute intervals for any patient with ongoing pain in whom the suspicion of ACS remains high. In some patients, initial nondiagnostic ECG changes will evolve into ST elevation or ST depression [10,12].

Bundle branch block or paced rhythm — Both LBBB, which is present in approximately 7 percent of patients with an acute MI [13], and pacing can interfere with the ECG diagnosis of MI (particularly STEMI) or coronary ischemia. Of note, approximately one-half of patients with LBBB and an acute MI do not have chest pain [14]. New right bundle branch block, while generally not interfering with the ECG diagnosis of STEMI, connotes an adverse prognosis similar in degree to LBBB.

Careful evaluation of the ECG may show some evidence of coronary ischemia in patients with LBBB or a paced rhythm. However, the clinical history and cardiac enzymes are of primary importance in suspecting or diagnosing MI in this setting. (See "Electrocardiographic diagnosis of myocardial infarction in the presence of bundle branch block or a paced rhythm".)

Troponin — Cardiac troponin (cTn) I and T are specific and sensitive biomarkers of cardiac injury. They are the preferred serologic tests for the evaluation of patients with suspected acute MI. The use of these tests is discussed in detail elsewhere, but the general principles will be briefly reviewed here. Values ≥99 percentile of the upper reference limit should be considered abnormal [2,7]. This value for troponin will vary depending on the assay used. (See "Troponin testing: Clinical use".)

An elevation in cTn must be interpreted in the context of the clinical history and ECG findings since it can be seen in a variety of clinical settings and is therefore not specific for an ACS. With an elevation of cTn in a situation where ischemia is not present, the term "cardiac injury" should be used. (See 'Other causes of biomarker elevation' below and "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome".)

As there can be chronic elevations of troponin in patients who do not have acute events, a rise and fall of troponin should be documented in acute MI [2,15]. The magnitude of change needed to operationalize this recommendation varies from assay to assay, so it is optimal when the clinical laboratory helps to make these distinctions [16].

Three points should be kept in mind when using troponin to diagnose acute MI:

- With highly sensitive troponin assays, most patients can be diagnosed within two to three hours of presentation [17].

- A negative test at the time of presentation, especially if the patient presents early after the onset of symptoms, does not exclude myocardial injury. Serial cTn testing is indicated in these patients.

- Acute MI can be excluded in most patients by six hours, but the guidelines suggest that if there is a high degree of suspicion of an ACS, a 12-hour sample should be obtained [2,7]. However, very few patients become positive after eight hours [18].

Isolated cTn increases in the absence of myocardial ischemia should be considered "myocardial injury" and a cause specified for its presence per the Fourth Universal Definition of MI. Troponin is also important for risk stratification after STEMI and NSTEMI. (See "Risk stratification after acute ST-elevation myocardial infarction" and "Risk stratification after non-ST elevation acute coronary syndrome" and "Risk factors for adverse outcomes after non-ST elevation acute coronary syndromes".)

Other causes of biomarker elevation — Elevations of troponin diagnose cardiac injury, not MI caused by acute coronary thrombus [19]. In patients with troponin elevation but without a clinical MI, other mechanisms for cardiac injury must be considered (eg, heart failure, rapid atrial fibrillation, myocarditis, anthracycline cardiotoxicity, subendocardial wall stress, myopericarditis, sepsis, etc). As an example, small amounts of cardiac injury can occur in critically ill patients, which may or may not represent an acute MI [20,21]. Troponin elevations also occur in chronic kidney disease. (See "Elevated cardiac troponin concentration in the absence of an acute coronary syndrome".)

In the emergency department setting, life-threatening causes of chest pain with troponin elevation not due to coronary artery disease are acute pulmonary embolism, in which troponin release may result from acute right heart overload, aortic dissection, myocarditis [19], and stress-induced cardiomyopathy. (See "Clinical presentation, evaluation, and diagnosis of the nonpregnant adult with suspected acute pulmonary embolism" and "Clinical manifestations and diagnosis of myocarditis in adults", section on 'Cardiac biomarkers' and "Clinical features and diagnosis of acute aortic dissection", section on 'Diagnosis'.)

Timing of measurement — We recommend the following approach to the timing of troponin measurement:

- Measure cTnI or cTnT at first presentation.

- If the troponin is not elevated, repeat at three to six hours. It is not uncommon to measure a second troponin earlier than six hours in patients who are highly suspected of having ongoing NSTEMI, since 80 percent of patients who rule in will do so in two to three hours

[17]. In an occasional patient in whom the index of suspicion for acute MI is high, but the first two troponin measurements are not elevated, a repeat measurement at 12 to 24 hours may be necessary.

- Patients who present late after the onset of symptoms may not manifest a changing pattern of high sensitivity cTn (hs-cTn) values over a short period of time. Thus, those who present late or in whom there is an unclear timing of the event and who have elevated hs-cTn may require a longer period of evaluation to document a changing pattern of values. This can occur in up to 26 percent of patients with MI.

In patients with suspected STEMI, appropriate care should not be delayed if the first troponin is not elevated.

DIAGNOSIS

According to the Fourth Universal Definition (see 'Definitions' above), the term acute myocardial infarction (MI) should be used when there is acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of cardiac troponin (cTn) values with at least one value above the 99th percentile URL and at least one of the following [2]:

- Symptoms of myocardial ischemia.
- New ischemic 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 ischemic etiology.
- Identification of a coronary thrombus by angiography or autopsy (not for type 2 or 3 MIs).

In addition, post-mortem demonstration of acute atherothrombosis in the artery supplying the infarcted myocardium meets criteria for type 1 MI. Evidence of an imbalance between myocardial oxygen supply and demand unrelated to acute atherothrombosis meets criteria for type 2 MI. Cardiac death in patients with symptoms suggestive of myocardial ischemia and presumed new ischemic ECG changes before cTn values become available or abnormal meets criteria for type 3 MI.

For some groups of patients such as the older individuals, individuals with diabetes, and women, symptoms may be atypical and ECG changes nonspecific. In those types of situations where ischemic heart disease is suspected, and a typical pattern of troponin is present, additional evaluations with imaging may be necessary.

COMPARING TYPE 1 AND 2 MYOCARDIAL INFARCTION

In addition to a different underlying pathology, type 1 and 2 MI have different patient characteristics and outcomes (see 'Joint Task Force definitions' above). In a 2021 database study comparing 216,657 patients with type 1 MI with 37,765 patients with type 2 MI, the following statistically significant differences were noted [22]:

- Patients with type 2 MI were older (71 versus 69 years) and more likely to be women (47.3 versus 40 percent).
- Type 2 MI patients had a higher prevalence of heart failure (27.9 versus 10.9 percent), kidney disease (35.7 versus 25.7 percent), and atrial fibrillation (31 versus 21 percent).
- Rates of coronary angiography (10.9 versus 57.3 percent), percutaneous coronary intervention (1.7 versus 38.5 percent), and coronary artery bypass grafting (0.4 versus 7.8 percent) were lower among type 2 MI patients.
- Patients with type 2 MI had lower risk of in-hospital mortality (adjusted odds ratio 0.57, 95% CI 0.54-0.60) and 30-day MI readmission (adjusted odds ratio 0.46, 95% CI 0.35-0.59). There was no difference in risk of 30-day all-cause or heart failure readmission.

DIFFERENTIAL DIAGNOSIS

Patients without features of typical angina are more likely to have another cause of chest pain. Common causes include other cardiovascular, pulmonary, and gastrointestinal disorders (table 3). (See 'History and physical examination' above.)

Life-threatening causes of chest pain that might suggest MI include pulmonary embolism, aortic dissection, pericarditis, and ruptured viscus.

Stress cardiomyopathy should also be considered. (See "Clinical manifestations and diagnosis of stress (takotsubo) cardiomyopathy".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Non-ST-elevation acute coronary syndromes (non-ST-elevation myocardial infarction)" and "Society guideline links: ST-elevation myocardial infarction (STEMI)".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Beyond the Basics topic (see "Patient education: Heart attack (Beyond the Basics)")

SUMMARY

- **Clinical definition** – Myocardial infarction (MI) is defined as a clinical (or pathologic) event caused by myocardial ischemia in which there is evidence of myocardial injury or necrosis. The diagnosis is secured when there is a rise and/or fall of cardiac biomarkers, along with supportive evidence in the form of typical symptoms, suggestive ECG changes, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality. (See 'Introduction' above.)

- **Types of MI** – The criteria used to define MI differ somewhat depending upon the particular clinical circumstance of the patient (eg, acute presentation with chest discomfort, presentation after percutaneous coronary intervention (PCI), unexplained cardiac arrest). (See 'Definitions' above.)

The two most common types of MI are:

- **Type 1** – MI caused by acute atherothrombotic coronary artery disease and usually precipitated by atherosclerotic plaque disruption (rupture or erosion).
- **Type 2** – MI consequent to a mismatch between oxygen supply and demand.

  This includes a multiplicity of potential mechanisms including coronary dissection, vasospasm, emboli, microvascular dysfunction, as well as increases in demand with or without underlying coronary artery disease.

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Topic 52 Version 49.0

GRAPHICS

Coronary artery surgery study definitions of angina

Definite angina
Substernal discomfort precipitated by exertion, with a typical radiation to the shoulder, jaw or inner aspect of the arm relieved by rest or nitroglycerin in less than 10 minutes
Probable angina
Has most of the features of definite angina but may not be entirely typical in some aspects
"Probably not" angina
Defined as an atypical overall pattern of chest pain that does not fit the description of definite angina
"Definitely not" angina
Chest pain is unrelated to activity, appears to be clearly of non-cardiac origin and is not relieved by nitroglycerin

Adapted from CASS Investigators. National Heart Lung and Blood Institute Coronary Artery Study, Circulation 1981; 63(Suppl I):I-81.

Graphic 59572 Version 1.0

Causes of ST-segment elevation

Myocardial ischemia/infarction
Noninfarction, transmural ischemia (eg vasospastic angina and takotsubo cardiomyopathy)
Acute MI especially due to acute epicardial coronary occlusion; may be due to takotsubo cardiomyopathy and other nonatherosclerotic coronary occlusion or severe hypoperfusion syndromes
Post-MI (ventricular aneurysm pattern)
Acute pericarditis
Abnormal early repolarization syndromes
Normal variants (including benign early repolarization)
Left ventricular hypertrophy or left bundle branch block (V1 to V2 or V3)
Other
Myocarditis (including COVID-19 infections)
Massive pulmonary embolism (leads V1 to V2 in occasional cases)
Brugada-type patterns (V1 to V3 with right bundle branch block-appearing morphology)
Myocardial tumor
Myocardial trauma
Hyperkalemia (only leads V1 and V2)
Hypothermia (J wave/Osborn wave)
Hypercalcemia (rarely)
Post-DC cardioversion (rarely)
Neurogenic stress cardiomyopathy

MI: myocardial infarction; COVID-19: coronavirus disease 2019; DC: direct current.

Graphic 81757 Version 8.0

Diagnoses other than cardiac ischemia for patients with chest pain

Cardiovascular not caused by epicardial CAD
Aortic dissection*
Aortic aneurysm*
Myocarditis*
Pericarditis
Stress (takotsubo) cardiomyopathy*
Chest wall
Cervical disc disease
Costochondritis
Fibrositis
Herpes zoster (before the rash)
Neuropathic pain
Rib fracture
Sternoclavicular arthritis

Pulmonary
Pleuritis
Pneumonia
Pulmonary embolus*
Tension pneumothorax*
Psychiatric
Affective disorders (eg, depression)
Anxiety disorders
Hyperventilation
Panic disorder
Primary anxiety
Somatoform disorders
Thought disorders (eg, fixed delusions)

Gastrointestinal
Biliary
Cholangitis
Cholecystitis
Choledocholithiasis
Colic
Esophageal
Esophagitis
Spasm
Reflux
Rupture*
Pancreatitis
Peptic ulcer disease
Nonperforating
Perforating*

* Potentially life-threatening conditions.

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