

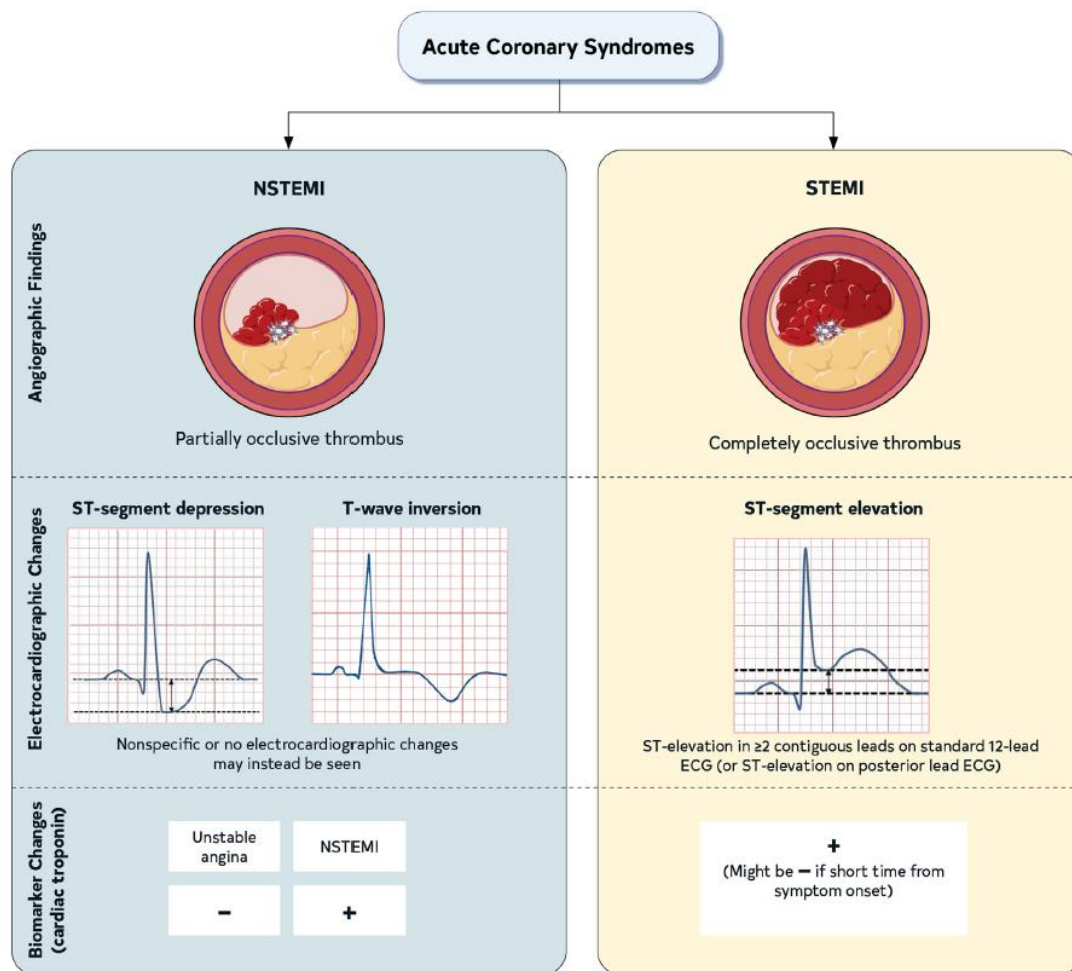


Figure 1. 2025 ACC/AHA GL on ACS. Types and Classification of Acute Coronary Syndromes

Table 1. 2025 ACC/AHA GL on ACS. Electrocardiographic Interpretation for Patients With Suspected Acute Coronary Syndromes

	NSTE-ACS	STEMI
Electrocardiographic evidence of ischemia	New or presumed new and usually dynamic horizontal or down-sloping ST-segment depression ≥ 0.5 mm in ≥ 2 contiguous leads and/or T-wave inversion >1 mm in ≥ 2 contiguous leads with prominent R wave or R/S ratio >1 or transient ST-segment elevation.	New or presumed new ST-segment elevation of ≥ 1 mm in ≥ 2 anatomically contiguous leads (measured at the J-point) in all leads other than V_2 - V_3 and ≥ 2 mm in men ≥ 40 y, ≥ 2.5 mm in men <40 y, and ≥ 1.5 mm in women regardless of age in leads V_2 - V_3 .*
Other observed electrocardiographic changes	Many patients with NSTE-ACS have either nonspecific ST-segment or T-wave changes or a normal ECG. The absence of electrocardiographic evidence of ischemia does not exclude ACS.	Posterior leads (V_7 - V_9) should be obtained in patients with suspected left circumflex occlusion particularly in the setting of isolated ST-segment depression ≥ 0.5 mm in leads V_1 - V_3 .

*ST-segment changes may be observed in other conditions including acute pericarditis, left ventricular hypertrophy, LBBB, Brugada syndrome, right ventricular pacing, Takotsubo syndrome, and early repolarization that may obscure the diagnosis of STEMI. New or presumably new LBBB at presentation occurs infrequently and should not be considered diagnostic of AMI in isolation; clinical correlation is required.¹³ A new LBBB in an asymptomatic patient does not constitute a STEMI equivalent.

ACS indicates acute coronary syndromes; AMI, acute myocardial infarction, LBBB, left bundle branch block; NSTE-ACS, non-ST-segment elevation ACS; and STEMI, ST-segment elevation MI.

2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol.* 2025

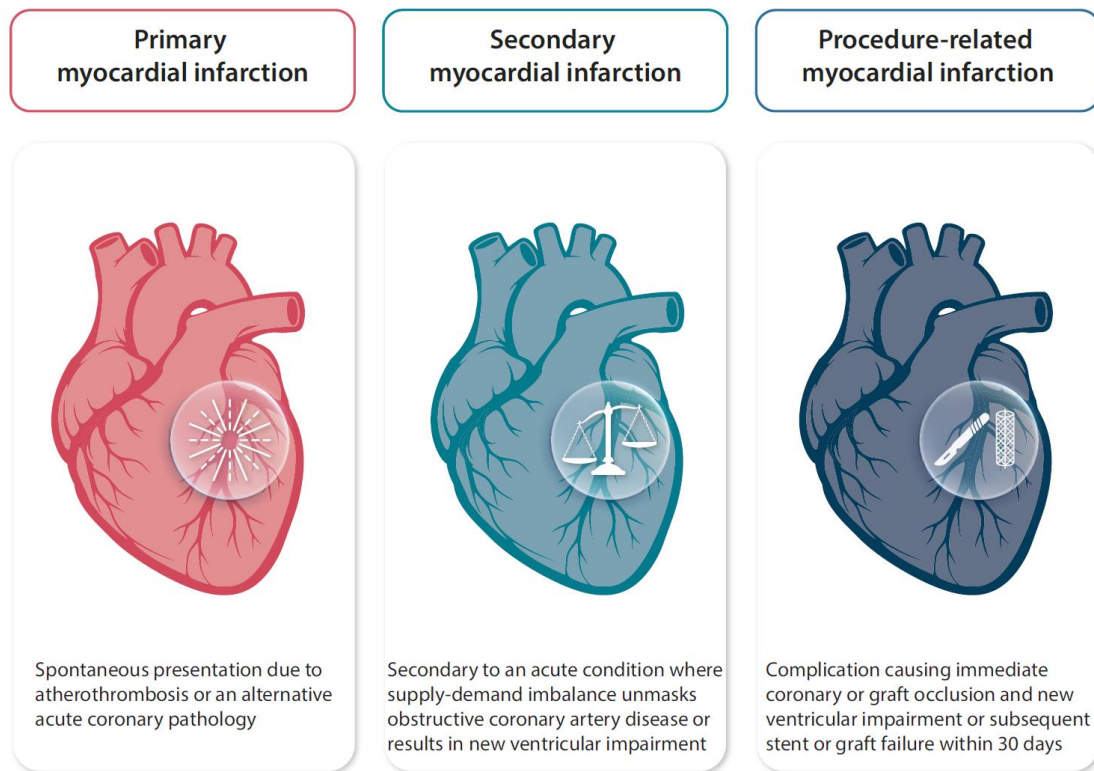


Figure 2. Fifth universal definition of myocardial infarction

Myocardial infarction can occur in three clinical settings: spontaneously due to a primary acute coronary pathology, secondary to an acute condition, or following a cardiac procedure

Table 2. ICD-11 codes for the classification of myocardial infarction

Final diagnosis	ICD-11	Sixth digit stem code/extension code
Acute ST-segment elevation myocardial infarction (STEMI) ^a	BA41.0	Primary myocardial infarction BA41..1 atherothrombosis
Acute non-ST-segment elevation myocardial infarction (NSTEMI)	BA41.1	BA41..2 spontaneous coronary artery dissection BA41..3 coronary embolism BA41..4 coronary vasospasm BA41..5 restenosis >30 days from procedure BA41..6 stent thrombosis >30 days from procedure BA41..7 graft failure >30 days from procedure BA41..8 undetermined aetiology BA41..9 unknown aetiology (no coronary imaging) Secondary myocardial infarction BA41..A/ICD-11 code for alternative acute condition Procedure-related myocardial infarction BA41..B due to a complication of any percutaneous cardiac procedure within 30 days BA41..C due to a complication of any open cardiac surgical procedure within 30 days
Other diagnoses		
Unstable angina	BA40	
Unspecified myocardial infarction	BA41.Z	Sudden death without evaluation ^b
Unrecognized myocardial infarction	BA50	

ICD-11, 11th edition of the International Classification of Diseases.

^a Including electrocardiographic findings of acute coronary occlusion

Table 3. Definitions and differential diagnosis of chest pain

AHA/ACC 2021 GL on Chest Pain.

Defining Chest Pain

An initial assessment of chest pain to triage patients effectively on the basis of the likelihood that symptoms may be attributable to myocardial ischemia. 1 B-NR

Chest pain should not be described as atypical, because it is not helpful in determining the cause and can be misinterpreted as benign in nature. Instead, chest pain should be described as cardiac, possibly cardiac, or noncardiac because these terms are more specific to the potential underlying diagnosis. 1 C-LD

History

A focused history that includes characteristics and duration of symptoms relative to presentation as well as associated features, and cardiovascular risk factor assessment should be obtained. 1 C-LD

Uniqueness of Chest Pain in Women

Women who present with chest pain are at risk for underdiagnosis, and potential cardiac causes should always be considered. 1 C-LD

In women presenting with chest pain, obtain a history that emphasizes accompanying symptoms that are more common in women with ACS 1 B-NR

Older Patients With Chest Pain

In patients who are ≥ 75 years of age, ACS should be considered when accompanying symptoms such as shortness of breath, syncope, or acute delirium are present, or when an unexplained fall has occurred. 1 C-LD

Diverse Patient Populations

Cultural competency training to help achieve the best outcomes in patients of diverse racial and ethnic backgrounds who present with chest pain. 1 C-LD

Among patients of diverse race and ethnicity in whom English may not be their primary language, addressing language barriers with the use of formal translation services is recommended. 1 C-LD

Patient-Centric Considerations

In patients with acute chest pain, 9-1-1 should be activated by patients or bystanders to initiate transport to the closest ED by emergency medical services (EMS). 1 C-LD

Chest Pain Characteristics and Corresponding Causes

Nature
Anginal symptoms are perceived as retrosternal chest discomfort (eg, pain, discomfort, heaviness, tightness, pressure, constriction, squeezing) (See Section 1.4.2, Defining Chest Pain, in the full guideline ¹).
Sharp chest pain that increases with inspiration and lying supine is unlikely related to ischemic heart disease (eg, these symptoms usually occur with acute pericarditis).
Onset and duration
Anginal symptoms gradually build in intensity over a few minutes.
Sudden onset of ripping chest pain (with radiation to the upper or lower back) is unlikely to be anginal and is suspicious of an acute aortic syndrome.
Fleeting chest pain—of few seconds' duration—is unlikely to be related to ischemic heart disease.
Location and radiation
Pain that can be localized to a very limited area and pain radiating to below the umbilicus or hip are unlikely related to myocardial ischemia.
Severity
Ripping chest pain ("worst chest pain of my life"), especially when sudden in onset and occurring in a hypertensive patient, or with a known bicuspid aortic valve or aortic dilation, is suspicious of an acute aortic syndrome (eg, aortic dissection).
Precipitating factors
Physical exercise or emotional stress are common triggers of anginal symptoms.
Occurrence at rest or with minimal exertion associated with anginal symptoms usually indicates ACS.
Positional chest pain is usually nonischemic (eg, musculoskeletal).
Relieving factors
Relief with nitroglycerin is not necessarily diagnostic of myocardial ischemia and should not be used as a diagnostic criterion.
Associated symptoms
Common symptoms associated with myocardial ischemia include, but are not limited to, dyspnea, palpitations, diaphoresis, lightheadedness, presyncope or syncope, upper abdominal pain, or heartburn unrelated to meals and nausea or vomiting.
Symptoms on the left or right side of the chest, stabbing, sharp pain, or discomfort in the throat or abdomen may occur in patients with diabetes, women, and elderly patients.

ACS indicates acute coronary syndrome.

Differential Diagnosis of Noncardiac Chest Pain

Respiratory

Pulmonary embolism
Pneumothorax/hemothorax
Pneumomediastinum
Pneumonia
Bronchitis
Pleural irritation
Malignancy

Gastrointestinal

Cholecystitis
Pancreatitis
Hiatal hernia
Gastroesophageal reflux disease/gastritis/esophagitis
Peptic ulcer disease
Esophageal spasm
Dyspepsia

Chest wall

Costochondritis
Chest wall trauma or inflammation
Herpes zoster (shingles)
Cervical radiculopathy
Breast disease
Rib fracture
Musculoskeletal injury/spasm

Psychological

Panic disorder
Anxiety
Clinical depression
Somatization disorder
Hypochondria

Other

Hyperventilation syndrome
Carbon monoxide poisoning
Sarcoidosis
Lead poisoning
Prolapsed intervertebral disc
Thoracic outlet syndrome
Adverse effect of certain medications (e.g., 5-fluorouracil)
Sickle cell crisis

ESC 2023 GL on ACS. Differential diagnoses of acute coronary syndrome in the setting of acute chest pain

Cardiac	Pulmonary	Vascular	Gastrointestinal	Orthopaedic	Other
Myocarditis/pericarditis, cardiomyopathies ^a	Pulmonary embolism	Aortic dissection	Oesophagitis, reflux, or spasm	Musculoskeletal disorders	Anxiety disorders
Tachyarrhythmias	(Tension) Pneumothorax	Symptomatic aortic aneurysm	Peptic ulcer, gastritis	Chest trauma	Herpes zoster
Acute heart failure	Bronchitis, pneumonia	Stroke	Pancreatitis	Muscle injury/ inflammation	Anaemia
Hypertensive emergencies	Pleuritis		Cholecystitis	Costochondritis	
Aortic valve stenosis				Cervical spine pathologies	
Takotsubo syndrome					
Coronary spasm					
Cardiac trauma					

^aDilated, hypertrophic, and restrictive cardiomyopathies may cause angina or chest discomfort.

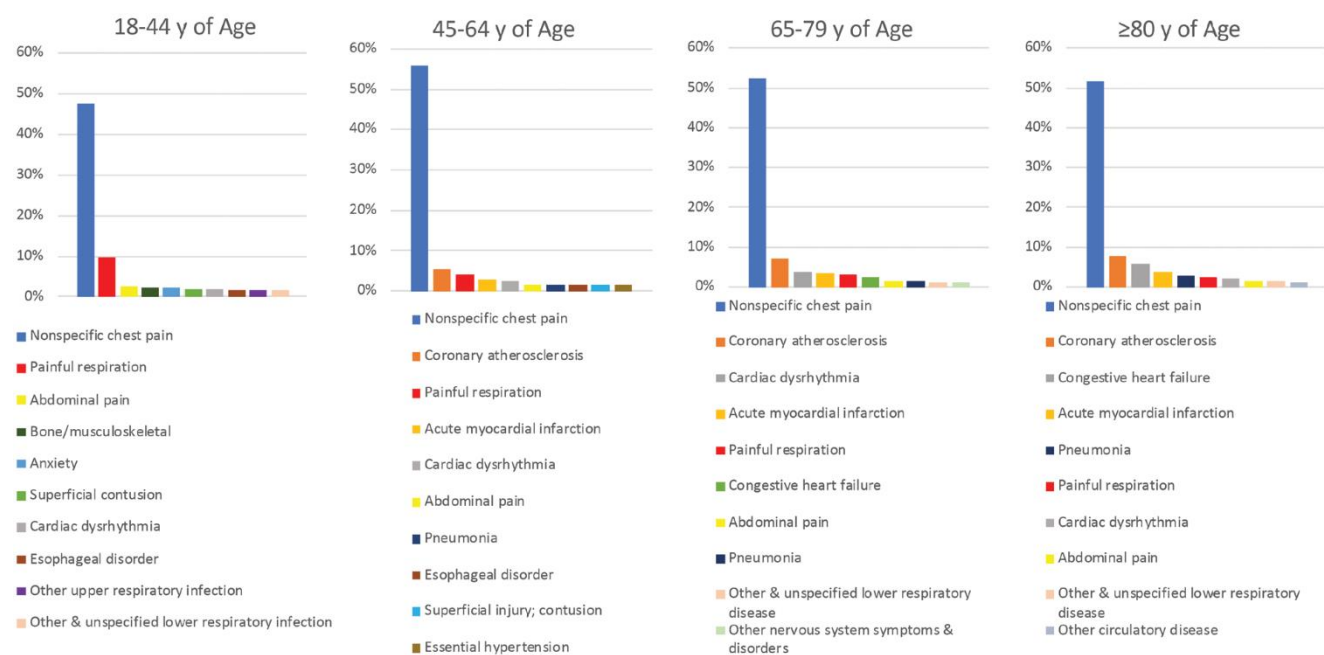


Figure 3. AHA/ACC 2021 GL on Chest Pain. Top 10 Causes of Chest Pain in the ED Based on Age (Weighted Percentage)

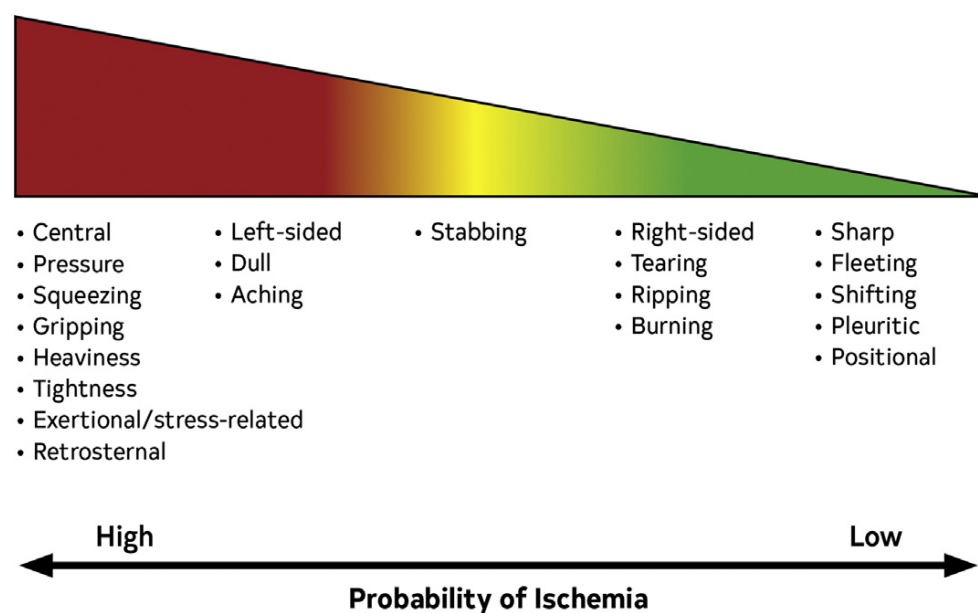


Figure 4. AHA/ACC 2021 GL on Chest Pain. Index of Suspicion That Chest "Pain" Is Ischemic in Origin on the Basis of Commonly Used Descriptors.

Table 4. AHA/ACC 2021 GL on Chest Pain. Physical examination**Physical Examination**

In patients presenting with chest pain, perform a focused cardiovascular examination to aid in the diagnosis of ACS or other potentially serious causes of chest pain (eg, aortic dissection, pulmonary embolism (PE), or esophageal rupture) and to identify complications. 1 C-EO

Physical Examination in Patients With Chest Pain

Clinical Syndrome	Findings
Emergency	
ACS	Diaphoresis, tachypnea, tachycardia, hypotension, crackles, S3, MR murmur (2); examination may be normal in uncomplicated cases
PE	Tachycardia + dyspnea—>90% of patients; pain with inspiration (7)
Aortic dissection	Connective tissue disorders (e.g., Marfan syndrome), extremity pulse differential (30% of patients, type A>B) (8) Severe pain, abrupt onset + pulse differential + widened mediastinum on CXR >80% probability of dissection (9) Frequency of syncope >10% (8), AR 40%–75% (type A) (10)
Esophageal rupture	Emesis, subcutaneous emphysema, pneumothorax (20% patients), unilateral decreased or absent breath sounds
Other	
Noncoronary cardiac: AS, AR, HCM	AS: Characteristic systolic murmur, tardus or parvus carotid pulse AR: Diastolic murmur at right of sternum, rapid carotid upstroke HCM: Increased or displaced left ventricular impulse, prominent a wave in jugular venous pressure, systolic murmur
Pericarditis	Fever, pleuritic chest pain, increased in supine position, friction rub
Myocarditis	Fever, chest pain, heart failure, S3
Esophagitis, peptic ulcer disease, gall bladder disease	Epigastric tenderness Right upper quadrant tenderness, Murphy sign
Pneumonia	Fever, localized chest pain, may be pleuritic, friction rub may be present, regional dullness to percussion, egophony
Pneumothorax	Dyspnea and pain on inspiration, unilateral absence of breath sounds
Costochondritis, Tietze syndrome	Tenderness of costochondral joints
Herpes zoster	Pain in dermatomal distribution, triggered by touch; characteristic rash (unilateral and dermatomal distribution)

ACS indicates acute coronary syndrome; AR, aortic regurgitation; AS, aortic stenosis; CXR, chest x-ray; LR, likelihood ratio; HCM, hypertrophic cardiomyopathy; MR, mitral regurgitation; PE, pulmonary embolism; and PUD, peptic ulcer disease.

Table 5. ACC/AHA 2025 GL on ACS. Initial Assessment**Prehospital Assessment and Management Considerations for Suspected ACS**

In patients with suspected ACS, 12-lead ECG should be acquired and interpreted within 10 minutes of first medical contact (FMC)* to identify patients with STEMI 1 B-NR

In patients with suspected ACS in which the initial ECG is nondiagnostic of STEMI, serial ECGs to detect potential ischemic changes should be performed, especially when clinical suspicion of ACS is high, symptoms are persistent, or the clinical condition deteriorates 1 C-LD

In patients with suspected STEMI, immediate emergency medical services (EMS) Transport to a PCI-capable hospital for primary PCI (PPCI[†]) is the recommended triage strategy, with an FMC–to-first-device time system goal of ≤90 minutes. 1 B-NR

In patients with suspected STEMI, early advance notification of the receiving PCI-capable hospital by EMS personnel and activation of the cardiac catheterization team is recommended to reduce time to reperfusion. 1 B-NR

*FMC indicates the time point when the patient is initially assessed by a health care professional who can obtain and interpret the ECG and deliver initial interventions (eg, defibrillation).

‡PPCI refers to emergency PCI in the setting of STEMI to achieve reperfusion in patients without previous fibrinolytic treatment.

Initial In-Hospital Assessment of Patients With Confirmed or Suspected ACS

Acquisition and interpretation of an ECG within 10 minutes is recommended to help guide patient management. 1 B-NR

If the initial ECG is nondiagnostic, serial 12-lead ECGs should be performed to detect potential ischemic changes, especially when clinical suspicion of ACS is high, symptoms are persistent, or clinical condition deteriorates. 1 B-NR

cTn should be measured as soon as possible, preferably using a high-sensitivity cTn (hs-cTn) assay 1 B-NR

With an initial hs-cTn or cTn that is nondiagnostic, the recommended time intervals for repeat measurements after the initial sample collection (time zero) are 1 to 2 hours for hs-cTn and 3 to 6 hours for conventional cTn assays. 1 B-NR

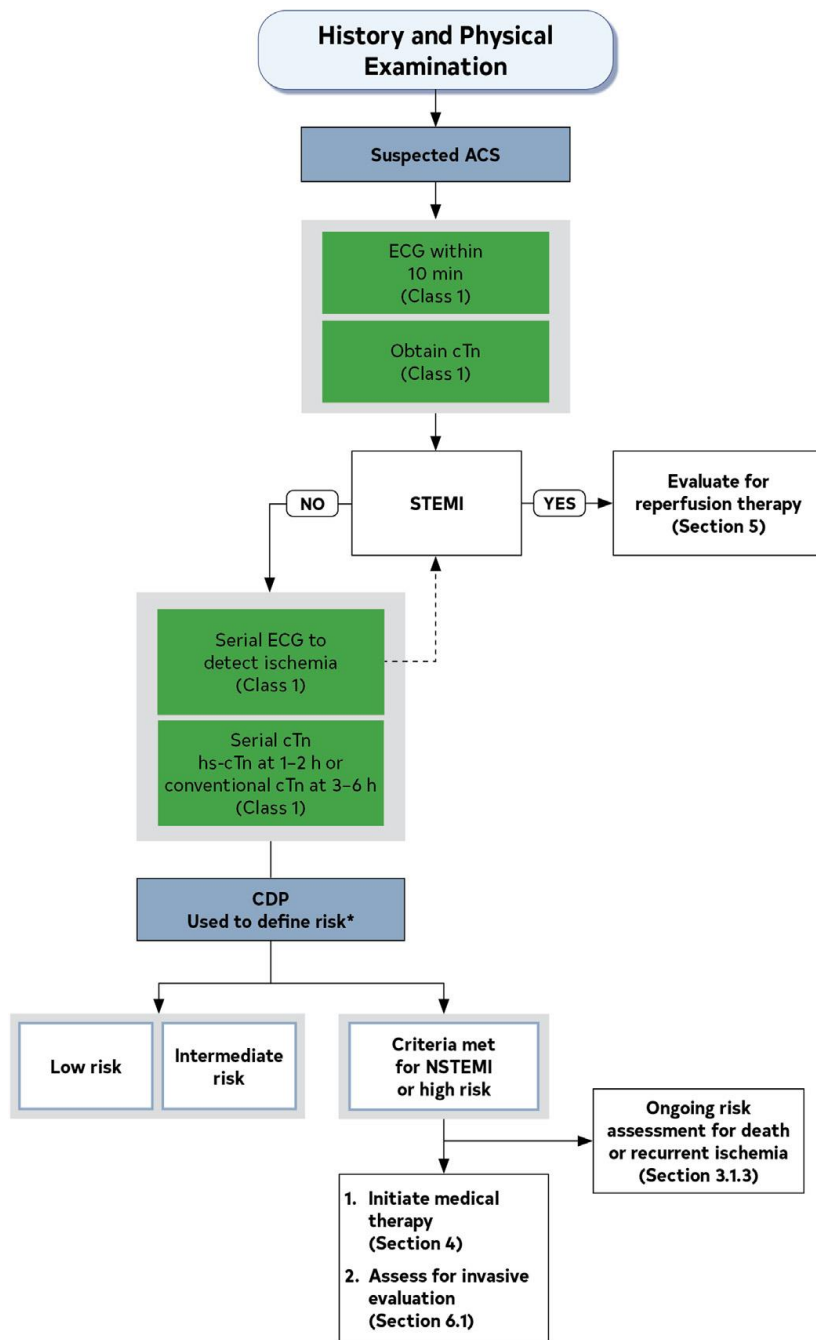


Figure 4. ACC/AHA 2025 GL on ACS. Initial Assessment of Patients With Suspected ACS

*Examples of evidence-based CDPs and definition of low, intermediate, and high risk as defined in the 2021 AHA/ACC/Multisociety Chest Pain Guideline.

ACS indicates acute coronary syndromes; CDP, clinical decision pathway; cTn, cardiac troponin; hs-cTn, high-sensitivity cardiac troponin; NSTEMI, non–ST-segment elevation myocardial infarction; and STEMI, ST-segment elevation myocardial infarction.

Table 6. ESC 2023 guidelines for ACS. Recommendations for clinical and diagnostic tools for patients with suspected acute coronary syndrome

Base the diagnosis and initial short-term risk stratification of ACS on a combination of clinical history, symptoms, vital signs, other physical findings, ECG, and hs-cTn.1 I B

ECG

Twelve-lead ECG recording and interpretation as soon as possible at the point of FMC, with a target of <10 min. I B

Continuous ECG monitoring and the availability of defibrillator capacity as soon as possible in all patients with suspected STEMI, in suspected ACS with other ECG changes or ongoing chest pain, and once the diagnosis of MI is made. I B

Use of additional ECG leads (V3R, V4R, and V7–V9) in cases of inferior STEMI or if total vessel occlusion is suspected and standard leads are inconclusive. I B

An additional 12-lead ECG in cases with recurrent symptoms or diagnostic uncertainty. I C

Blood sampling

Measure cardiac troponins with high-sensitivity assays immediately after presentation and obtain the results within 60 min of blood sampling I B

Use an ESC algorithmic approach with serial hs-cTn measurements (0 h/1 h or 0 h/2 h) to rule in and rule out NSTEMI I B

Additional testing after 3 h if the first two hs-cTn measurements of the 0 h/1 h algorithm are inconclusive and no alternative diagnoses explaining the condition have been made I B

Use of established risk scores (e.g. GRACE risk score) for prognosis estimation IIa B

Triage for emergency reperfusion strategy

Patients with suspected STEMI are immediately triaged for an emergency reperfusion strategy I A

ACS, acute coronary syndrome; ECG, electrocardiogram; ESC, European Society of Cardiology; FMC, first medical contact; GRACE, Global Registry of Acute Coronary Events; hs-cTn, high-sensitivity cardiac troponin; MI, myocardial infarction; NSTEMI, non-ST-elevation myocardial infarction; STEMI, ST-elevation myocardial infarction.

Table 7. Electrocardiographic findings of myocardial ischaemia or infarction

Feature	Description of electrocardiographic findings of myocardial ischaemia or infarction
ST-segment elevation	New ST-segment elevation at the J-point in two contiguous leads: ≥ 1 mm in all leads other than leads V_2 - V_3 where the following apply: ≥ 2.5 mm in males <40 years, ≥ 2 mm in males ≥ 40 years, or ≥ 1.5 mm in females regardless of age, in the absence of bundle branch block and/or left ventricular hypertrophy
ST-segment depression	New horizontal or down sloping ST-segment depression ≥ 0.5 mm at the J-point in two or more contiguous leads
Hyperacute T waves	Symmetrical, broad T waves disproportionately large to the preceding QRS complex in two contiguous leads
de Winter T waves	Tall, prominent, symmetrical T waves with upsloping ST segment depression in the precordial leads
Biphasic T waves	Initial positive deflection followed by a negative deflection
T-wave inversion	New or dynamic T-wave inversion ≥ 1 mm in two contiguous leads
Wellens syndrome	Biphasic or deeply inverted T waves in leads V_2 and V_3
Pathologic Q waves	Q-wave duration ≥ 40 ms and/or a depth of $\geq 25\%$ of the R wave in two contiguous leads
Sgarbossa criteria	In the presence of left bundle branch block or ventricular pacing; ST-segment elevation ≥ 1 mm concordant (in the same direction) with the QRS complex; ST-segment depression ≥ 1 mm in lead V_1 , V_2 , or V_3 ; ST-segment elevation ≥ 5 mm not concordant with the QRS complex
Ventricular arrhythmia	Ventricular fibrillation or ventricular tachycardia

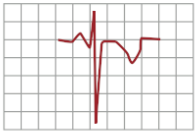
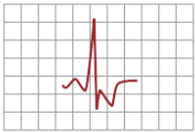
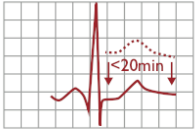
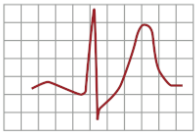
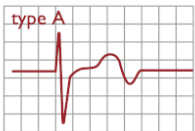
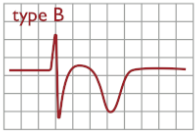
ECG pattern	Criteria	Signifying	Figure
a Isolated T-wave inversion	T-wave inversion >1 mm in ≥ 5 leads including I, II, aVL, and V2–V6	Only mildly impaired prognosis	 I, II, aVL, or V2 to V6
b ST-segment depression	J point depressed by ≥ 0.05 mm in leads V2 and V3 or ≥ 1 mm in all other leads followed by a horizontal or downsloping ST-segment for ≥ 0.08 s in ≥ 1 leads (except aVR)	More severe ischaemia	 ≥ 1 leads  ≥ 1 leads
c Transient ST-segment elevation	ST segment elevation in ≥ 2 contiguous leads of ≥ 2.5 mm in men <40 years, ≥ 2 mm in men ≥ 40 years, or ≥ 1.5 mm in women regardless of age in leads V2–V3 and/or ≥ 1 mm in the other leads lasting <20 min	Only mildly impaired prognosis	 ≥ 2 contiguous leads
d De Winter ST-T	1–3 mm upsloping ST-segment depression at the J point in leads V1–V6 that continue into tall, positive, and symmetrical T waves	Proximal LAD occlusion/ severe stenosis	 V1–V6
e Wellens sign	Isoelectric or minimally elevated J point (<1 mm) + biphasic T wave in leads V2 and V3 (type A) or symmetric and deeply inverted T waves in leads V2 and V3, occasionally in leads V1, V4, V5, and V6 (type B)	Proximal LAD occlusion/ severe stenosis	 type A (V1-)V2-V3(-V4)  type B (V1-)V2-V3(-V4)

Figure 5. ESC 2023 GL on ACS. Electrocardiographic abnormalities in patients with non-ST-segment elevation acute coronary syndrome.

ECG, electrocardiogram; LAD, left anterior descending artery. This figure highlights some of the electrocardiographic abnormalities that may be present in patients with NSTEMI-ACS.

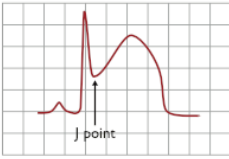
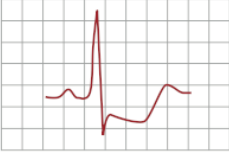
ECG pattern	Criteria	Signifying	Figure
i STEMI	New ST-elevation at the J-point in ≥ 2 contiguous leads ^a ≥ 2.5 mm in men <40 years, ≥ 2 mm in men ≥ 40 years, or ≥ 1.5 mm in women regardless of age in leads V2–V3 and/or ≥ 1 mm in the other leads (in the absence of LV hypertrophy or left bundle branch block) ^a Including V3R and V4R	Ongoing acute coronary artery occlusion	
ii Posterior STEMI	ST-segment depression in leads V1–V3, especially when the terminal T-wave is positive (ST-segment elevation equivalent), and concomitant ST-segment elevation ≥ 0.5 mm recorded in leads V7–V9	Posterior STEMI	
iii LCx occlusion/ right ventricular MI	ST-segment elevation in V7–V9 and V3R and V4R, respectively	Left circumflex (LCX) artery occlusion or right ventricular MI	
iv Multivessel ischaemia/ left main obstruction	ST depression ≥ 1 mm in six or more surface leads (inferolateral ST depression), coupled with ST-segment elevation in aVR and/or V1	Multivessel ischaemia or left main coronary artery obstruction, particularly if the patient presents with haemodynamic compromise	
v Left bundle branch block/ paced rhythm	QRS duration greater than 120 ms Absence of Q wave in leads I, V5 and V6 Monomorphic R wave in I, V5 and V6 ST and T wave displacement opposite to the major deflection of the QRS complex	Patients with a high clinical suspicion of ongoing myocardial ischaemia should be managed in a similar way to STEMI patients	
vi Right bundle branch block	QRS duration greater than 120 ms rsR' "bunny ear" pattern in the anterior precordial leads (leads V1–V3) Slurred S waves in leads I, aVL and frequently V5 and V6	Patients with a high clinical suspicion of ongoing myocardial ischaemia should be managed in a similar way to STEMI patients	

Figure 6. ESC 2023 GL on ACS. Electrocardiographic abnormalities in patients with STEMI and ECG findings that, if present, may prompt triage for immediate reperfusion therapy.

ECG, electrocardiogram; STE-ACS, ST-elevation acute coronary syndrome; STEMI, ST-elevation myocardial infarction.

Table 8. Causes of ST elevation other than myocardial infarction

ST elevation of normal variant	V ₁ -V ₅ with inverted T waves
LV hypertrophy	Concave, other features of LV hypertrophy
Acute pericarditis	Diffuse ST segment depression but <5 mm Reciprocal ST segment depression in aVR, not in aVL PR segment depression
Hyperkalaemia	Widened QRS and tall, peaked, tented T waves Low-amplitude or absent P waves ST segment usually downsloping
Brugada syndrome (type 1)	rSR' in V ₁ and V ₂
Wolff-Parkinson-White syndrome	ST elevation with J point notching in leads I, aVL, V ₄ -V ₆
Pulmonary embolism	ST elevation in both inferior and anteroseptal leads
Cardioversion	Transient ST elevation (1-2 min after direct shock)
Hiatus hernia and stomach compression	Concave elevation in anterior chest leads without reciprocal inferior ST depression
Cardiac tumors	Localized, convex ST elevation and T wave inversion
Propofol-related infusion syndrome	Downsloping ST segment elevation

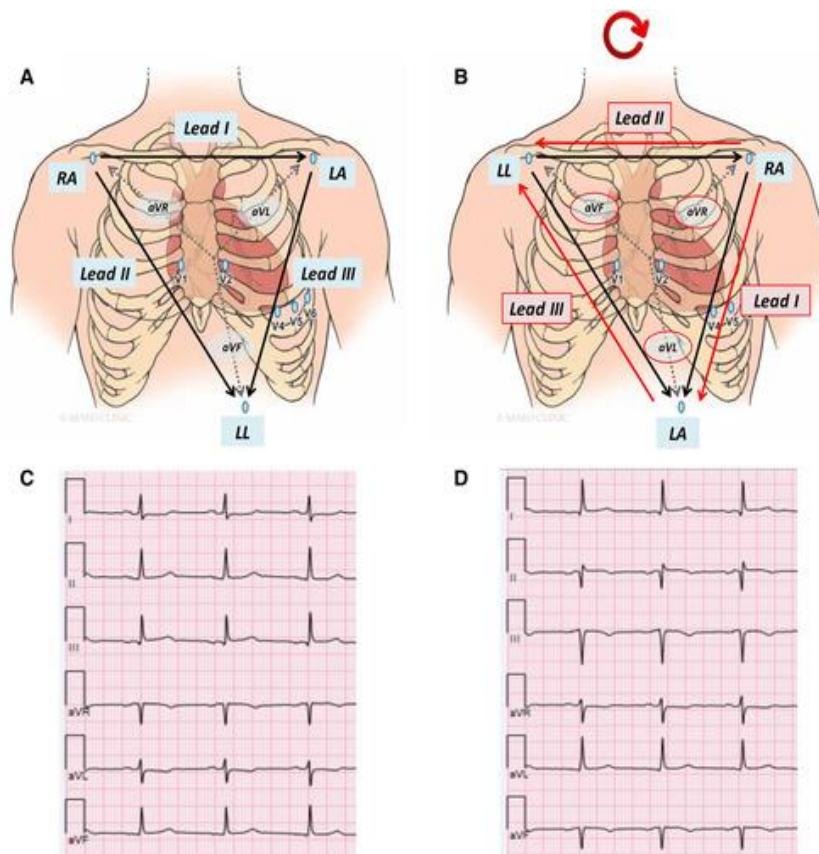


Figure 7. The “carousel” rotation.

A, Normal electrode placement and how the vectors of limb leads are obtained. B, Lead misplacement in a clockwise “carousel” rotation and the new vectors of the misplaced leads. Side-by-side comparison of the baseline (C) and misplaced (D) ECGs. Notice the shift in identical P and QRS complex patterns among the augmented limb leads (ie, lead aVR in C is identical to lead aVF in D). The bipolar limb leads vectors of the erroneously recorded ECG (D) are deducted based on the anatomic location of the misplaced electrodes (ie, lead II in D records the voltage difference between left leg and right arm electrodes, which, as shown in B, is the opposite vector of the normal lead I; therefore the P wave and QRS morphology in the misplaced lead II is the negative reflection of normal lead I). LA indicates left arm; LL, left leg; and RA, right arm.

Tapia-Orihuela RKA, et al. A “Carousel” ECG Confusion. *Circulation*. 2021;143:1824–1827

Table 9. Criteria for identification of the infarct-related artery in anterior and inferior MI

Anterior MI	
Proximal LAD occlusion	ST elevation in V_1 to V_3 (aVL) and ST depression in II, III, aVF
Proximal LAD occlusion	ST elevation >2.5 mm in V_1 or RBBB with initial Q wave (not sensitive but very specific criteria)
LAD occlusion distal to first diagonal	ST elevation in V_1 to V_3 without ST depression in II, III, aVF
Distal LAD occlusion	ST elevation in V_1 to V_3 (aVL) and ST elevation in II, III, aVF (inferoapical LAD extension)
Inferior MI	
RCA occlusion	ST elevation in III > ST elevation in II, and ST depression in I or aVL
Left Cx occlusion	ST elevation in III not greater than ST elevation in II, and ST elevation or isoelectric in I, aVL, V_5 , V_6

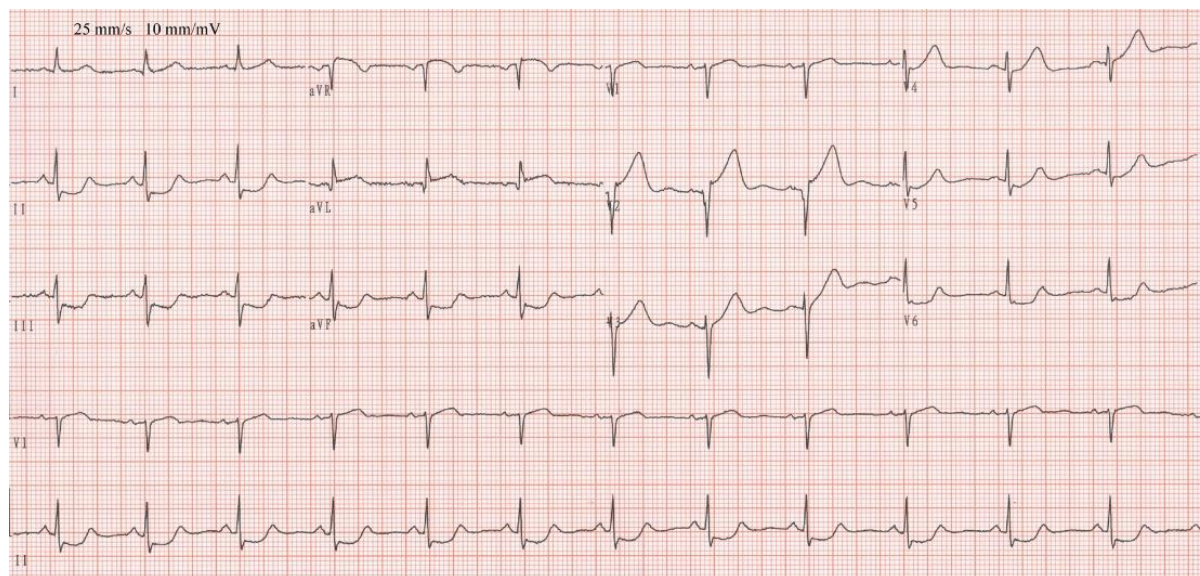


Figure 8. Q waves and ST-segment elevations in leads aVL and V_2 with ST-segment depressions in leads II, III, aVF, and V_4 to V_6 . Lead V_2 is electrically continuous with lead aVL (approximately -30°); therefore, the concomitant ST-segment elevation in both leads supports the diagnosis of high lateral wall ischemia.

Chang Q et al. An Atypical Presentation of an Occlusion Myocardial Infarction Requiring Urgent Revascularization. *Circulation*. 2026.

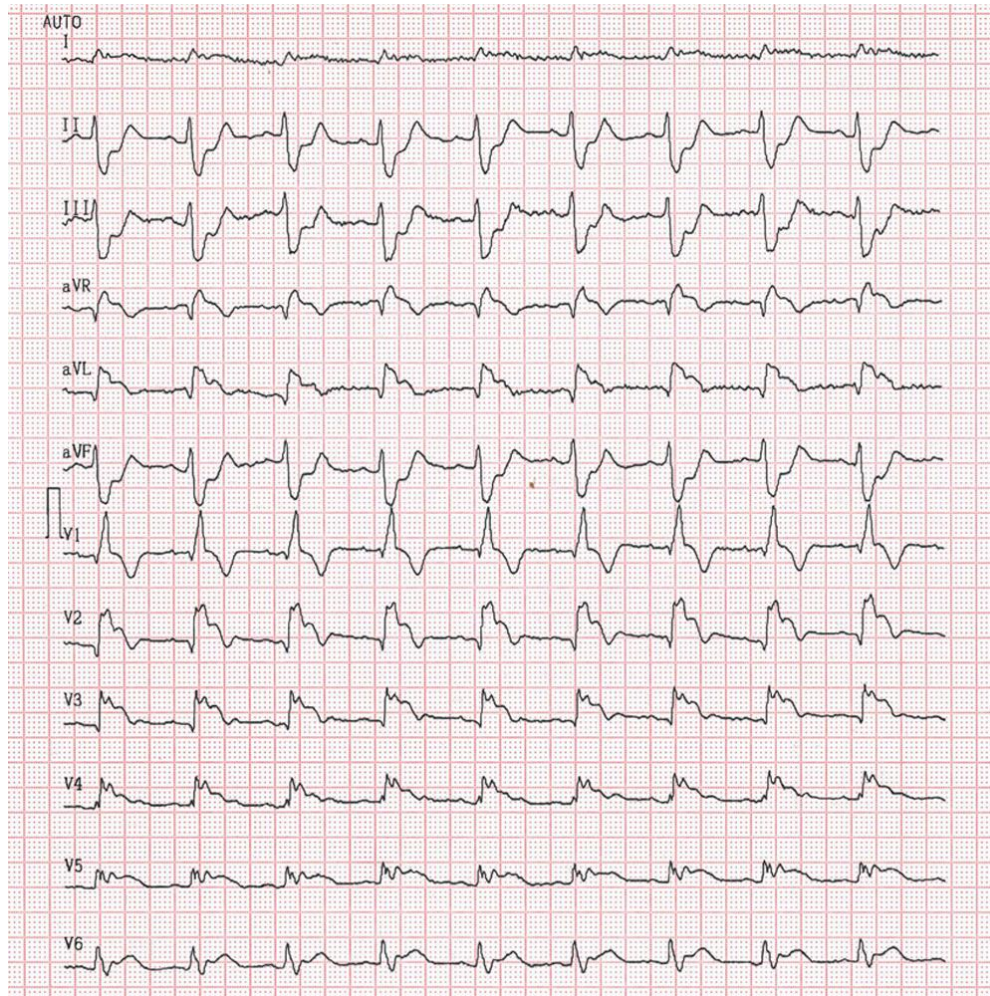


Figure 9. Left main stem occlusion.

Zhou H, et al. Anterior ST-Segment Elevation Myocardial Infarction Without ST Elevation in Lead V1: Where Is the Culprit Lesion? *Circulation*. 2020;141:1351-1353.

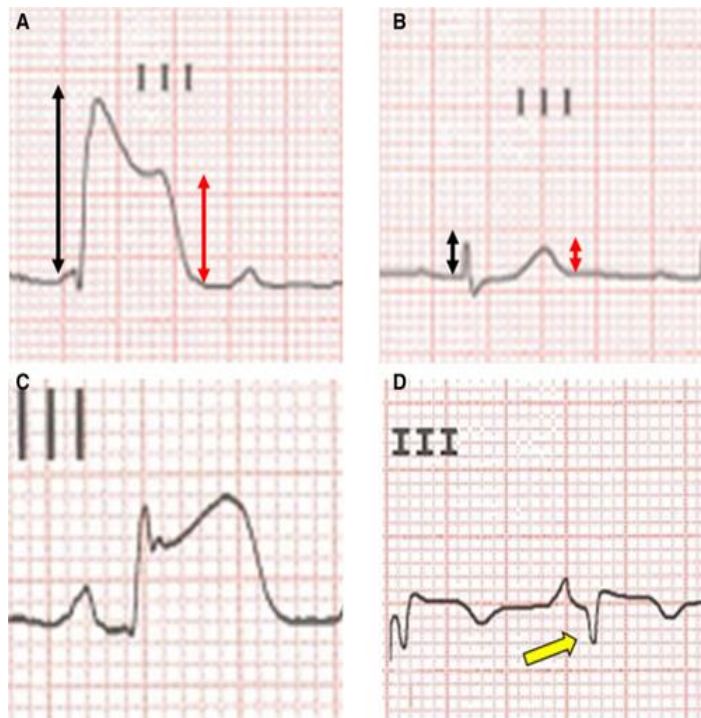


Figure 10. Changes observed in QRS complex and ST-segment changes (lead III) during (A) and after (B) epicardial spasm and during (C) and after (D) myocardial infarction attributable to plaque rupture, both involving the proximal right coronary artery.

A significant increase in amplitude (black arrow) and duration, along with a delay in the peak of the R wave, was observed during right coronary spasm. ST-segment elevation, blurring of J point, and increased amplitude of T wave (red arrow) resulted in a characteristic monophasic curve. A nonconducted P wave (complete atrioventricular block) contributed to an additional deformation of this curve. This QRS-T monophasic curve is not seen in acute myocardial infarction (C). The development of Q waves is usually seen after myocardial infarction (yellow arrow; D) and not after coronary spasm.

Real C and al. e. An Unusual Call From the Urology Ward. . *Circulation* 2021;144:324-327.

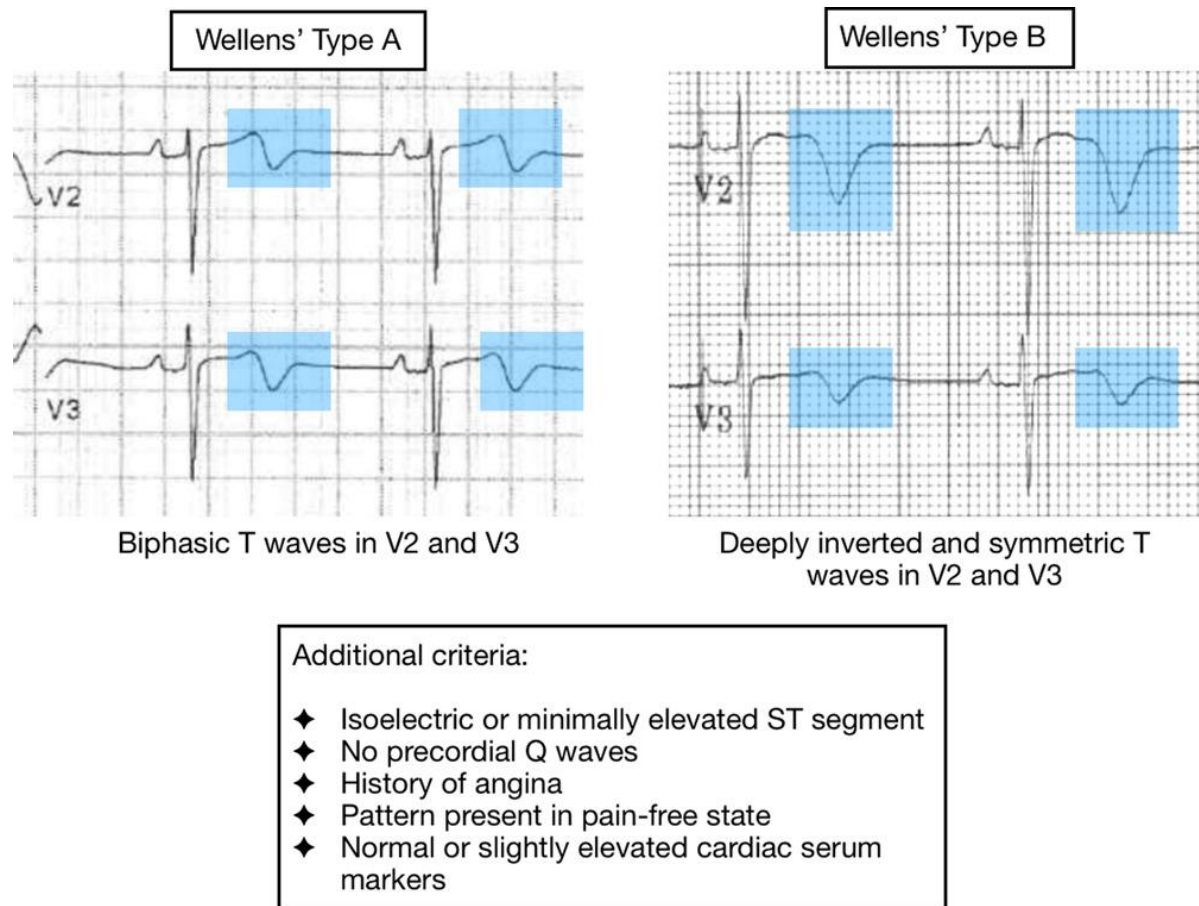


Figure 11. Wellens patterns and syndrome criteria. The upper left image depicts Wellens type A pattern with biphasic T waves in V2 and V3. The upper right image depicts Wellens type B pattern with deeply inverted and symmetrical T waves in V2 and V3. The lower box shows the additional criteria present that characterize Wellens syndrome.

Lutz K, et al. Flash Pulmonary Edema in a 70-Year-Old Man. *Circulation*. 2021;143:1331-1334.

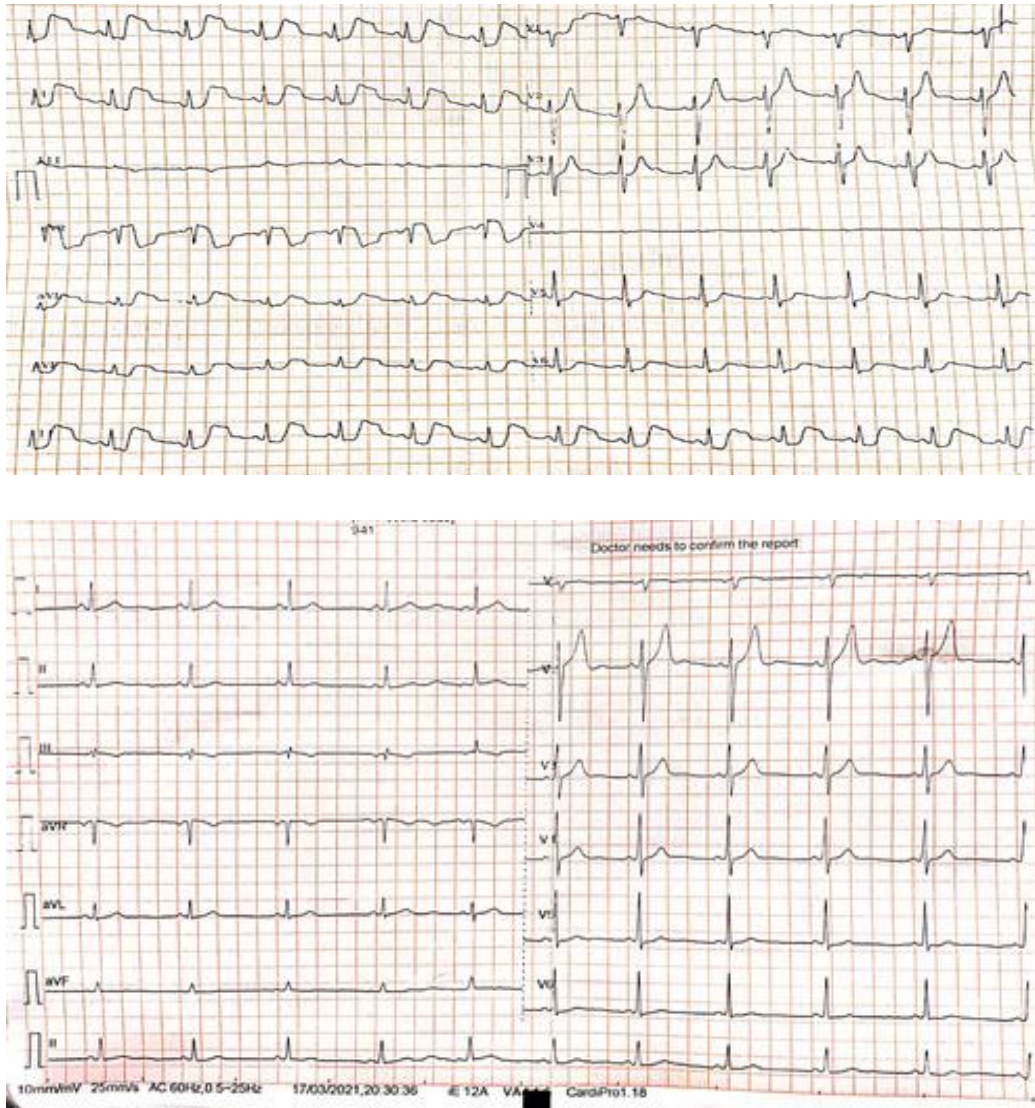


Figure 12. Electromechanical association artifact.

Upper panel: 12-lead ECG showing bizarre morphologies of ST-segments and T waves not evident in lead III.

Lower panel: Twelve-lead ECG taken after 15 minutes with the right arm clip placed proximally.

Rajendran G, et al. Not All Waves Are Factual. *Circulation*. 2021;144:751-753.

Table 10. Definition of the BARCELONA Algorithm to Diagnose AMI in the Presence of LBBB

The BARCELONA algorithm is positive if any of the following criteria are present:

(1) ST deviation ≥ 1 mm (0.1 mV) concordant with QRS polarity in any ECG lead, thus including either:

ST depression ≥ 1 mm (0.1 mV) concordant with QRS polarity, in any ECG lead.

ST elevation ≥ 1 mm (0.1 mV) concordant with QRS polarity, in any ECG lead

(2) ST deviation ≥ 1 mm (0.1 mV) discordant with QRS polarity, in any lead with max (R|S) voltage ≤ 6 mm (0.6 mV).

Di Marco A, et al. New Electrocardiographic Algorithm for the Diagnosis of Acute Myocardial Infarction in Patients With Left Bundle Branch Block. *Journal of the American Heart Association*. 2020;9:e015573.

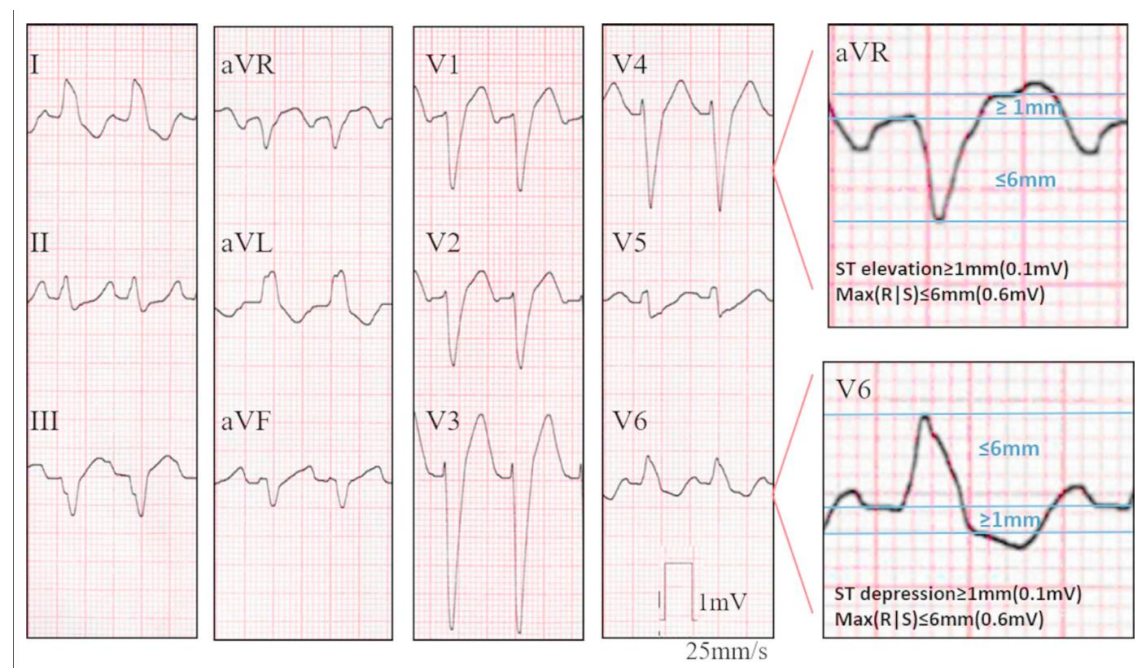


Figure 13. ECG from a patient with acute myocardial infarction and culprit lesion in the left circumflex artery, showing discordant ST deviation ≥ 1 mm (0.1 mV) in 2 leads with a QRS voltage ≤ 6 mm (0.6 mV).

Di Marco A, et al. New Electrocardiographic Algorithm for the Diagnosis of Acute Myocardial Infarction in Patients With Left Bundle Branch Block. *Journal of the American Heart Association*. 2020;9:e015573.

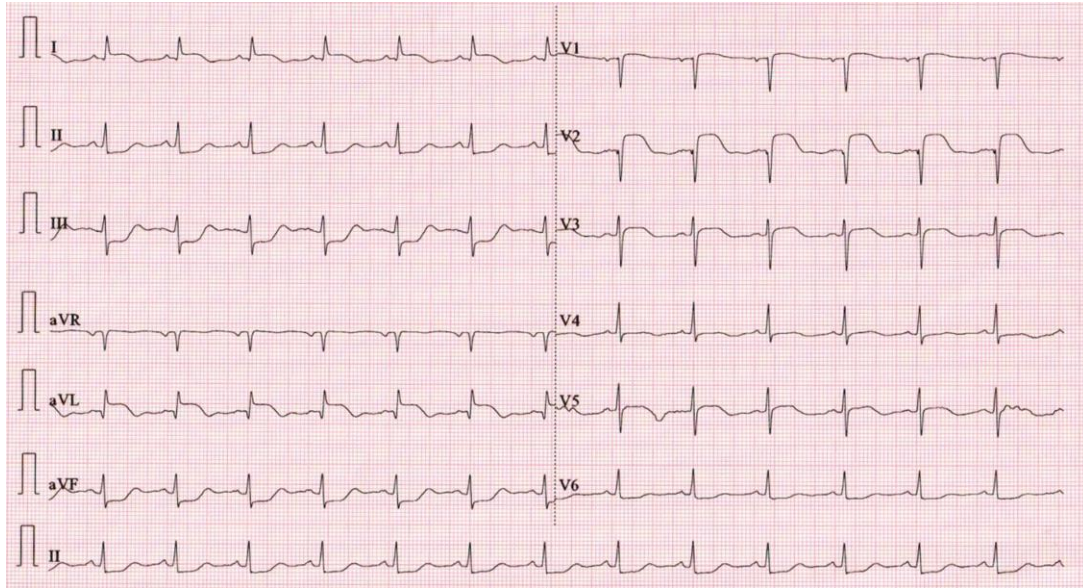


Figure 14. Anterior STEMI in dextrocardia.

ECG repeated with precordial leads on the right side of chest and right and left arm electrode reversal showed anterolateral ST-segment–elevation myocardial infarction with ST elevation in V1–V3, I, and aVL and reciprocal ST depression in the inferior leads.

Akkineni KP, et al. Deceptive Clues: When the Obvious Isn't the Answer. *Circulation*. 2025;152:560-562.

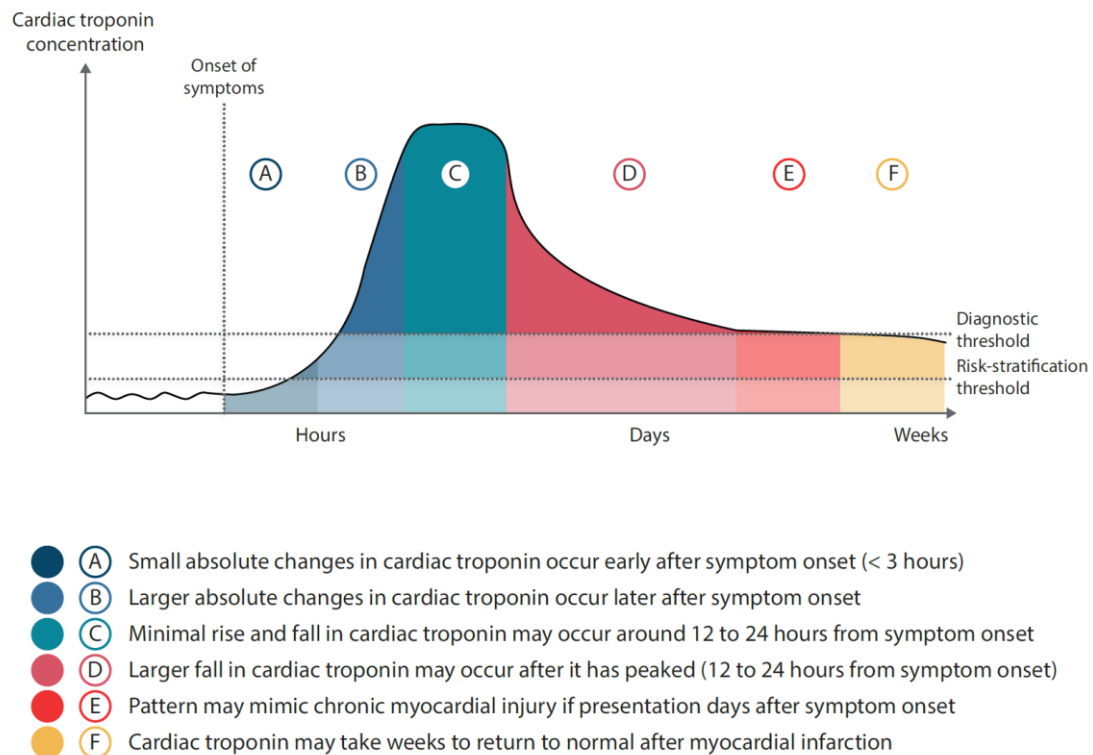


Figure 15. Rise and fall in cardiac troponin over time from symptom onset.

Following the onset of acute myocardial infarction, changes in cardiac troponin vary over time: (A) Small absolute changes in cardiac troponin occur early after symptom onset (<3 h). (B) Larger absolute changes in cardiac troponin occur later after symptom onset. (C) Minimal rise and fall in cardiac troponin may occur around 12 to 24 h from symptom onset. (D) Larger fall in cardiac troponin may occur after it has peaked (12 to 24 h from symptom onset). (E) Pattern may mimic chronic myocardial injury if presentation days after symptom onset. (F) Cardiac troponin may take weeks to return to normal after myocardial infarction and some patients may develop chronic myocardial injury following myocardial infarction.

Mills NL, et al. Fifth Universal Definition of Myocardial Infarction (2026). *Eur Heart J.* 2026.

Table 11. ESC 2023 GL on ACS. Conditions other than acute Type 1 myocardial infarction associated with cardiomyocyte injury (i.e. cardiac troponin elevation)

Myocardial injury related to acute myocardial ischaemia because of oxygen supply/demand imbalance (Type 2 MI)	
Reduced myocardial perfusion, e.g.:	
• Coronary artery spasm, microvascular dysfunction	
• Coronary embolism	
• Non-atherosclerotic coronary artery dissection	
• Sustained bradyarrhythmia	
• Hypotension or shock	
• Respiratory failure	
• Severe anaemia	
Increased myocardial oxygen demand, e.g.:	
• Sustained tachyarrhythmia	
• Severe hypertension with or without left ventricular hypertrophy	
Other causes of myocardial injury	
Cardiac conditions:	
• Heart failure	
• Myocarditis ^a	
• Cardiomyopathy (any type)	
• Takotsubo syndrome	
• Cardiac contusion or cardiac procedures (CABG, PCI, valvular interventions, ablation, pacing, cardioversion, or endomyocardial biopsy)	
Systemic conditions:	
• Sepsis, infectious disease	
• Chronic kidney disease	
• Stroke, subarachnoid haemorrhage	
• Pulmonary embolism, pulmonary hypertension	
• Infiltrative diseases (e.g. amyloidosis, sarcoidosis, haemochromatosis, scleroderma)	
• Myocardial drug toxicity or poisoning (e.g. doxorubicin, 5-fluorouracil, trastuzumab, snake venoms)	
• Critically ill patients	
• Hypo- and hyper-thyroidism	
• Strenuous exercise	
• Rhabdomyolysis	

CABG, coronary artery bypass grafting; MI, myocardial infarction; PCI, percutaneous coronary intervention.

^aIncludes myocardial extension of endocarditis or pericarditis.

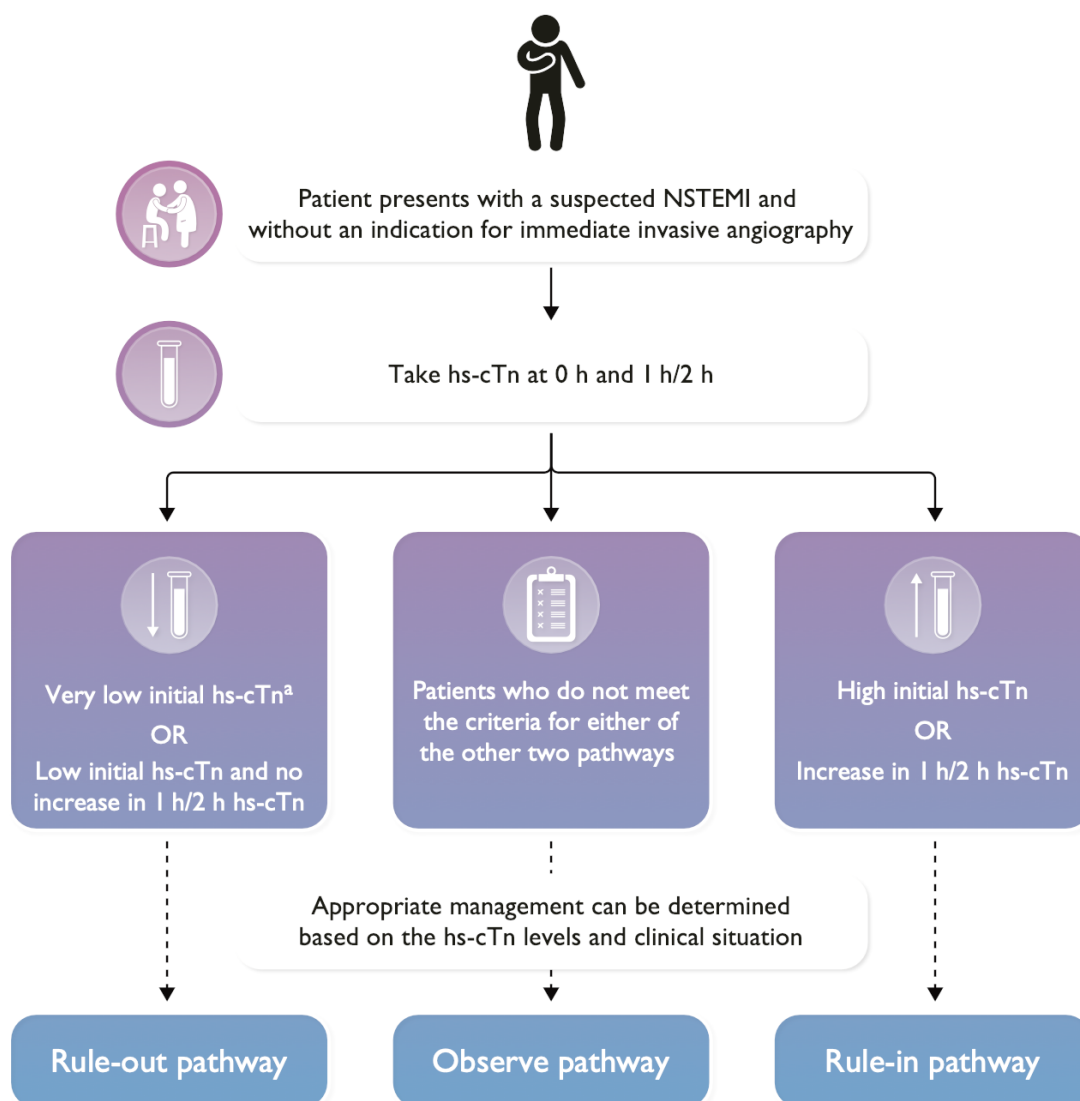


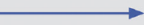
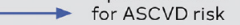
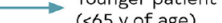
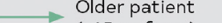
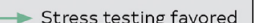
Figure 16. ESC 2023 GL on ACS. The 0 h/1 h or 0 h/2 h rule-out and rule-in algorithms using high-sensitivity cardiac troponin assays in patients presenting to the emergency department with suspected NSTEMI and without an indication for immediate invasive angiography.

Patients are classified into one of three pathways as per the results of their hs-cTn values at 0 h (time of initial blood test) and 1 h or 2 h later. Patients with a very low initial hs-cTn value or patients with a low initial value and no 1 h/2 h change in hs-cTn are assigned to the ‘rule-out’ pathway. Patients with a high initial hs-cTn value or a 1 h/2 h change in hs-cTn are assigned to the ‘rule-in’ pathway. Patients who do not meet the criteria for the rule-out or rule-in strategies are assigned to the ‘observe’ pathway, and these patients should have hs-cTn levels checked at 3 h ± echocardiography in order to decide on further management. Cut-offs are assay specific and derived to meet pre-defined criteria for sensitivity and specificity for NSTEMI. Potential management and testing options for each of the three strategies are provided in the relevant sections of the main text.

^aOnly applicable if the chest pain onset was >3 h prior to the 0 h hs-cTn measurement.
hs-cTn, high-sensitivity cardiac troponin; NSTEMI, non-ST-elevation myocardial infarction.

Table 12. AHA/ACC 2021 GL on Chest Pain. Diagnostic Tests

Choosing the Right Diagnostic Test

Pretest likelihood of CAD	Low   No testing necessary   Option for CAC for ASCVD risk stratification		
	Intermediate-high   Younger patient (<65 y of age)	OR	Less obstructive CAD suspected   CCTA favored
	Intermediate-high   Older patient (≥65 y of age)	OR	More obstructive CAD suspected   Stress testing favored

	Favors use of CCTA	Favors use of stress imaging
Goal	• Rule out obstructive CAD • Detect nonobstructive CAD	• Ischemia-guided management
Availability and expertise	• High-quality imaging and expert interpretation routinely available	• High-quality imaging and expert interpretation routinely available
Likelihood of obstructive CAD	• Age <65 y	• Age ≥65 y
Prior test results	• Prior functional study inconclusive	• Prior CCTA inconclusive
Other compelling indications	• Anomalous coronary arteries • Require evaluation of aorta or pulmonary arteries	• Suspect scar (especially if PET or stress CMR available) • Suspect coronary microvascular dysfunction (when PET or CMR available)

Stress testing information					
	ETT	Stress echocardiography	SPECT MPI	PET MPI	Stress CMR MPI
Patient capable of exercise	✓	✓	✓		
Pharmacologic stress indicated		✓	✓	✓	✓
Quantitative flow				✓	✓
LV dysfunction/scar		✓	✓	✓	✓

ASCVD indicates atherosclerotic cardiovascular disease; CAD, coronary artery disease; CAC, coronary artery calcium; CCTA, coronary computed tomography angiography; CMR, cardiovascular magnetic resonance; ETT, exercise tolerance test; LV, left ventricular; MPI, myocardial perfusion imaging; PET, positron emission tomography and SPECT, single-photon emission computed tomography.

Contraindication by Type of Imaging Modality and Stress Protocol

Exercise ECG	Stress Nuclear	Stress Echocardiography	Stress CMR	CCTA
■ Abnormal ST changes on resting ECG, digoxin, left bundle branch block, Wolff-Parkinson-White pattern, ventricular paced rhythm (unless test is performed to establish exercise capacity and not for diagnosis of ischemia) ■ Unable to achieve ≥ 5 METs or unsafe to exercise ■ High-risk unstable angina or AMI (< 2 d) i.e., active ACS ■ Uncontrolled heart failure ■ Significant cardiac arrhythmias (e.g., VT, complete atrioventricular block) or high risk for arrhythmias caused by QT prolongation ■ Severe symptomatic aortic stenosis ■ Severe systemic arterial hypertension (e.g., $\geq 200/110$ mm Hg) ■ Acute illness (e.g., acute PE, acute myocarditis/pericarditis, acute aortic dissection)	■ High-risk unstable angina, complicated ACS or AMI (< 2 d) ■ Contraindications to vasodilator administration ■ Significant arrhythmias (e.g., VT, second- or third-degree atrioventricular block) or sinus bradycardia < 45 bpm ■ Significant hypotension (SBP < 90 mm Hg) ■ Known or suspected bronchoconstrictive or bronchospastic disease ■ Recent use of dipyridamole or dipyridamole-containing medications ■ Use of methylxanthines (e.g., aminophylline, caffeine) within 12 h ■ Known hypersensitivity to adenosine, regadenoson ■ Severe systemic arterial hypertension (e.g., $\geq 200/110$ mm Hg)	■ Limited acoustic windows (e.g., in COPD patients) ■ Inability to reach target heart rate ■ Uncontrolled heart failure ■ High-risk unstable angina, active ACS or AMI (< 2 d) ■ Serious ventricular arrhythmia or high risk for arrhythmias attributable to QT prolongation ■ Respiratory failure ■ Severe COPD, acute pulmonary emboli, severe pulmonary hypertension ■ Contraindications to dobutamine (if pharmacologic stress test needed) ■ Atrioventricular block, uncontrolled atrial fibrillation ■ Critical aortic stenosis† ■ Acute illness (e.g., acute PE, acute myocarditis/pericarditis, acute aortic dissection) ■ Hemodynamically significant LV outflow tract obstruction ■ Contraindications to atropine use: ■ Narrow-angle glaucoma ■ Myasthenia gravis ■ Obstructive uropathy ■ Obstructive gastro intestinal disorders ■ Severe systemic arterial hypertension (e.g., $\geq 200/110$ mm Hg) <u>Use of Contrast Contraindicated in:</u> ■ Hypersensitivity to perflutren ■ Hypersensitivity to blood, blood products, or albumin (for Optison only)	■ Reduced GFR (< 30 mL/min/1.73 m²) ■ Contraindications to vasodilator administration ■ Implanted devices not safe for CMR or producing artifact limiting scan quality/interpretation ■ Significant claustrophobia ■ Caffeine use within past 12 h	■ Allergy to iodinated contrast ■ Inability to cooperate with scan acquisition and/or breath-hold instructions ■ Clinical instability (e.g., acute respiratory distress, severe hypotension, unstable arrhythmia) ■ Renal impairment as defined by local protocols ■ Contraindication to beta blockade in the presence of an elevated heart rate and no alternative medications available for achieving target heart rate ■ Heart rate variability and arrhythmia ■ Contraindication to nitroglycerin (if indicated)

For all the imaging modalities, inability to achieve high-quality images should be considered, in particular for obese patients

Readers should also review each imaging society's guidelines for more details on test contraindications (1-14).

*Screening for potential pregnancy by history and/or pregnancy testing should be performed according to the local imaging facilities policies for undertaking radiological examinations that involve ionizing radiation in women of child-bearing age.

†Low-dose dobutamine may be useful for assessing for low-gradient AS.

ACS indicates acute coronary syndrome; AMI, acute myocardial infarction; AS, aortic stenosis; CCTA, coronary computed tomography angiography; CMR, cardiovascular magnetic resonance imaging; COPD, chronic obstructive pulmonary disease; GFR, glomerular filtration rate; LV, left ventricular; MET, metabolic equivalent; MRI, magnetic resonance imaging; PE, pulmonary embolism; SBP, systolic blood pressure; and VT, ventricular tachycardia.

Warranty Period for Prior Cardiac Testing

Test Modality	Result	Warranty Period
Anatomic	Normal coronary angiogram CCTA with no stenosis or plaque	2 y
Stress testing	Normal stress test (given adequate stress)	1 y

CCTA indicates coronary computed tomographic angiography.

Table 13. ESC 2023 guidelines for ACS. Recommendations for non-invasive imaging in the initial assessment of patients with suspected acute coronary syndrome

Emergency TTE in patients with suspected ACS presenting with cardiogenic shock or suspected mechanical complications.	I C
In patients with suspected ACS, non-elevated (or uncertain) hs-cTn levels, A no ECG changes and no recurrence of pain, incorporating CCTA or a non-invasive stress imaging test as part of the initial workup	IIa
Emergency TTE at triage in cases of diagnostic uncertainty but this should not result in delays C in transfer to the cardiac catheterization laboratory if there is suspicion of an acute coronary artery occlusion.	IIa
Routine, early CCTA in patients with suspected ACS is not recommended. B	III

ACS, acute coronary syndrome; CCTA, coronary computed tomography angiography; ECG, electrocardiogram; hs-cTn, high-sensitivity cardiac troponin; TTE, transthoracic echocardiography.

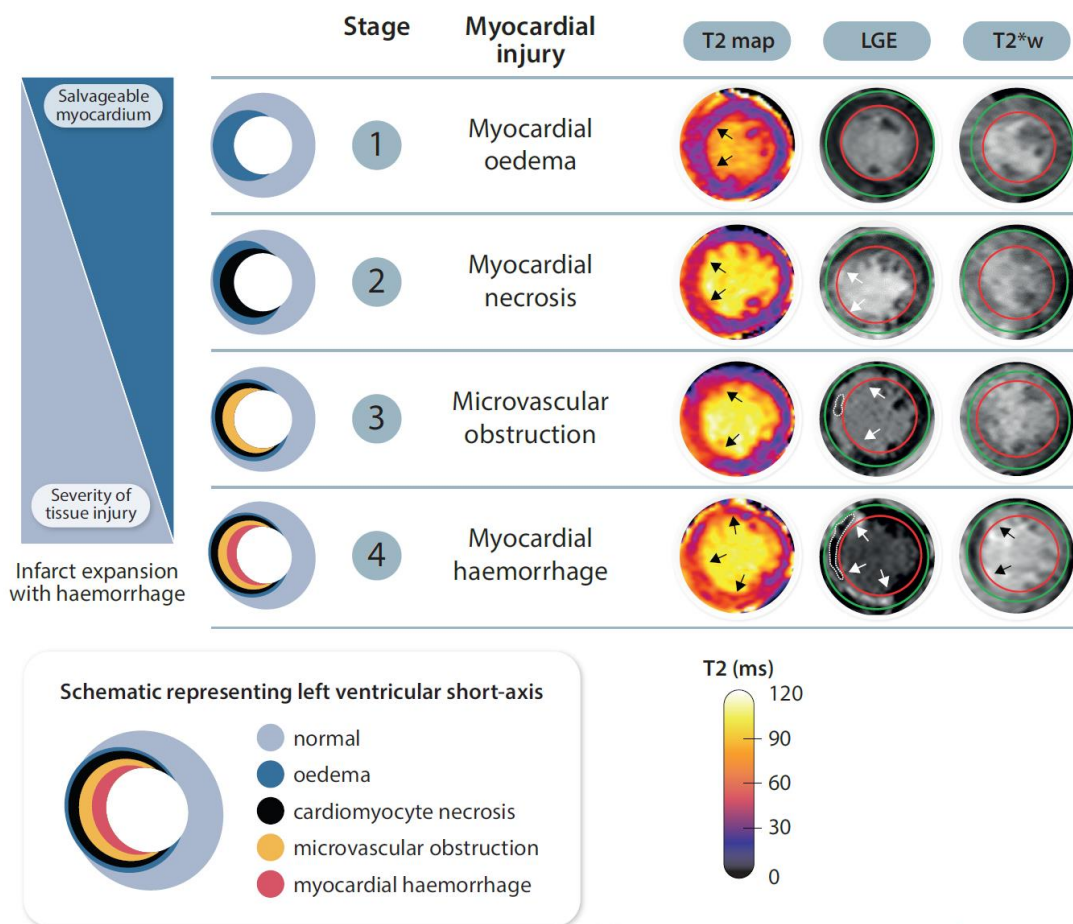


Figure 17. Stages of primary myocardial infarction by cardiac magnetic resonance imaging.

LGE, late gadolinium enhancement. Each stage of myocardial injury is progressive and is discernible from aggregate evidence derived from T2 maps (colour bar provided), LGE, and T2*-weighted (T2*w) short-axis cardiac magnetic resonance images along each row. *Stage 1* is marked by oedema only (high signal on T2 map), absence of myocardial necrosis (lack of signal enhancement on LGE image), or myocardial haemorrhage (absence of low signal on T2*w images). *Stage 2* demonstrates both myocardial necrosis (high signal on LGE image) and oedema (high signal on T2 map) but absence of persistent microvascular obstruction (absence of low signal within the zone of LGE image) or myocardial haemorrhage. *Stage 3* highlights the presence of microvascular obstruction (presence of low signal within the zone of high signal on LGE image) along with myocardial necrosis and oedema. Note that myocardial haemorrhage is also absent at Stage 3 as evidenced by lack of low signal within the zone of infarction on T2*w image. *Stage 4* is characterized by myocardial haemorrhage (low signal within the zone of high signal in the T2*w image), along with microvascular obstruction, myocardial necrosis, and oedema. High signal (brighter than surrounding tissue) and low signal (darker than surrounding tissue). Black arrowheads point to high signal on T2 maps, white arrow heads point to high signal on LGE, and blue arrow heads point to low signal on T2*-w image. Note that low signal zones on Stage 3 and Stage 4 within LGE are demarcated by contoured zones within high signal zones in LGE. Red and green contours on LGE and T2w images are endocardial and epicardial contours segmenting the left ventricular myocardium.

Mills NL, et al. Fifth Universal Definition of Myocardial Infarction (2026). *Eur Heart J.* 2026.

Table 14. AHA/ACC 2021 GL on Chest Pain. Patient-centric algorithms

Patients With Acute Chest Pain and Suspected ACS (Not Including STEMI)

Clinical decision pathways (CDPs) should categorize patients into low-, intermediate-, and high-risk strata to facilitate disposition and subsequent diagnostic evaluation. 1 B-NR

Recommended time intervals for serial troponins after the initial troponin sample collection (time zero) for repeat measurements are: 1 to 3 hours for high-sensitivity troponin and 3 to 6 hours for conventional troponin assays. 1 B-NR

To standardize the detection and differentiation of myocardial injury in patients presenting with acute chest pain and suspected ACS, institutions should implement a CDP that includes a protocol for troponin sampling based on their particular assay. 1 C-LD

Orevious testing when available should be considered and incorporated into CDPs. 1 C-LD

For patients with acute chest pain, a normal ECG, and symptoms suggestive of ACS that began at least 3 hours before ED arrival, a single hs-cTn concentration that is below the limit of detection on initial measurement (time zero) to exclude myocardial injury 2a B-NR

Definition Used for Low-Risk Patients With Chest Pain

Low Risk (<1% 30-d Risk for Death or MACE)	
hs-cTn Based	
T-0	T-0 hs-cTn below the assay limit of detection or "very low" threshold if symptoms present for at least 3 h
T-0 and 1- or 2-h Delta	T-0 hs-cTn and 1- or 2-h delta are both below the assay "low" thresholds (>99% NPV for 30-d MACE)
Clinical Decision Pathway Based	
HEART Pathway ⁴¹	HEART score ≤3, initial and serial cTn/hs-cTn < assay 99th percentile
EDACS ¹⁰⁶	EDACS score ≤16; initial and serial cTn/hs-cTn < assay 99th percentile
ADAPT ⁹⁰	TIMI score 0, initial and serial cTn/hs-cTn < assay 99th percentile
mADAPT	TIMI score 0/1, initial and serial cTn/hs-cTn < assay 99th percentile
NOTR ⁹⁴	0 factors

ADAPT indicates 2-hour Accelerated Diagnostic Protocol to Access Patients with Chest Pain Symptoms Using Contemporary Troponins as the Only Biomarkers; cTn, cardiac troponin; EDACS, Emergency Department Acute Coronary Syndrome; HEART Pathway, History, ECG, Age, Risk Factors, Troponin; hs-cTn, high-sensitivity cardiac troponin; MACE, major adverse cardiovascular events; mADAPT, modified 2-hour Accelerated Diagnostic Protocol to Access Patients with Chest Pain Symptoms Using Contemporary Troponins as the Only Biomarkers; NOTR, No Objective Testing Rule; NPV, negative predictive value; and TIMI, Thrombolysis in Myocardial Infarction.

Low-Risk Patients With Acute Chest Pain

Patients with a 30-day risk of death or major adverse cardiovascular events (MACE) <1% should be designated as low risk. 1 B-NR

Discharge home without admission or urgent cardiac testing patients with suspected ACS who are deemed low-risk 2a B-NR

(<1% 30-day risk of death or MACE).

Intermediate-Risk Patients With Acute Chest Pain

Transthoracic echocardiography (TTE) as a rapid, bedside test to establish baseline ventricular and valvular function, evaluate for wall motion abnormalities, and to assess for pericardial effusion. 1 C-EO

Management in an observation unit to shorten length of stay and lower cost relative to an inpatient admission 2a A

Intermediate-Risk Patients With No Known CAD

Index Diagnostic Testing

Anatomic Testing

For patients eligible for diagnostic testing after a negative or inconclusive evaluation for ACS, coronary computed tomography angiography (CCTA) for exclusion of atherosclerotic plaque and obstructive CAD. 1 A

For patients with moderate-severe ischemia on current or prior (≤ 1 year) stress testing, and no known CAD established by prior anatomic testing, invasive coronary angiography (ICA). 1 C-EO

For patients with evidence of previous mildly abnormal stress test results (≤ 1 year), CCTA is reasonable for diagnosing obstructive CAD. 2a C-LD

Stress Testing

For patients are eligible for cardiac testing, either exercise ECG, stress echocardiography, stress positron emission tomography (PET)/single-photon emission computed tomography (SPECT) myocardial perfusion imaging (MPI), or stress CMR for the diagnosis of myocardial ischemia. 1 B-NR

Sequential or Add-on Diagnostic Testing

For patients with a coronary artery stenosis of 40% to 90% in a proximal or middle coronary artery on CCTA, fractional flow reserve computed tomography (FFR-CT) for the diagnosis of vessel-specific ischemia and to guide decision-making regarding the use of coronary revascularization. 2a B-NR

For patients with an inconclusive prior stress test, CCTA for excluding the presence of atherosclerotic plaque and obstructive CAD. 2a C-EO

For patients with an inconclusive CCTA, stress imaging (with echocardiography, PET/SPECT MPI, or CMR) for the diagnosis of myocardial ischemia 2a C-EO

Intermediate-Risk Patients With Known CAD

Optimize GDMT before additional cardiac testing is performed, in patients with new onset or worsening symptoms I A

ICA in patients with worsening symptoms significant left main, proximal left anterior descending stenosis or multivessel CAD on prior anatomic testing I A

or history of prior coronary revascularization

CCTA to determine progression of atherosclerotic plaque in patients with known nonobstructive CAD I Ia B-NR

FFR-CT for the diagnosis of vessel-specific ischemia and to guide decision-making regarding the use of coronary revascularization, in patients with a coronary artery stenosis of 40% to 90% in a proximal or middle coronary artery on CCTA I Ia B-NR

Stress imaging (PET/SPECT MPI, CMR, or stress echocardiography) in patients with new onset or worsening symptoms. I Ia B-NR

High-Risk Patients With Acute Chest Pain

Patients with new ischemic changes on electrocardiography, troponin-confirmed acute myocardial injury, new-onset left ventricular systolic dysfunction (ejection fraction <40%), newly diagnosed moderate-severe ischemia on stress testing, hemodynamic instability, and/or a high clinical decision pathway (CDP) risk score should be designated as high risk for short-term MACE. I B-NR

ICA for patients designated as high risk I C-EO

CMR or echocardiography for establishing alternative diagnoses in high-risk patients with acute chest pain who are troponin positive in whom obstructive CAD has been excluded by CCTA or ICA I Ia B-NR

AHA/ACC 2021 GL on Chest Pain.

Low-Risk Patients With Stable Chest Pain and No Known CAD

For patients presenting to the outpatient clinic, a model to estimate pretest probability of obstructive CAD is effective to identify patients at low risk for obstructive CAD and favorable prognosis in whom additional diagnostic testing can be deferred. 1 B-NR

For patients categorized as low risk, CAC testing as a first-line test for excluding calcified plaque and identifying patients with a low likelihood of obstructive CAD. 2a B-R

For patients categorized as low risk, exercise testing without imaging as a first-line test for excluding myocardial ischemia and determining functional capacity in patients with an interpretable ECG. 2a B-NR

Intermediate-High Risk Patients With Stable Chest Pain and No Known CAD

Anatomic Testing

CCTA for diagnosis of CAD, for risk stratification, and for guiding treatment decisions. 1 A

Stress Testing

Stress imaging (stress echocardiography, PET/SPECT MPI or CMR) for diagnosis of myocardial ischemia and for estimating risk of MACE. 1 B-R

For patients for whom rest/stress nuclear MPI is selected, PET in preference to SPECT, if available to improve diagnostic accuracy and decrease the rate of nondiagnostic test results.	2a B-R
Exercise electrocardiography for patients with an interpretable ECG and ability to achieve maximal levels of exercise (≥ 5 METs).	2a B-R
In patients selected for stress MPI using SPECT, use of attenuation correction or prone imaging may be reasonable to decrease the rate of false-positive findings.	2b B-R
Assessment of Left Ventricular Function	
In patients with pathological Q waves, symptoms or signs suggestive of heart failure, complex ventricular arrhythmias, or a heart murmur with unclear diagnosis, TTE for diagnosis of resting left ventricular systolic and diastolic ventricular function and detection of myocardial, valvular, and pericardial abnormalities.	1 B-NR
Sequential or Add-on Testing: What to Do if Index Test Results are Positive or Inconclusive	
For patients with known coronary stenosis of 40% to 90% in a proximal or middle coronary segment on CCTA, FFR-CT for diagnosis of vessel-specific ischemia and to guide decision-making regarding the use of coronary revascularization.	2a B-NR
CCTA after an inconclusive or abnormal exercise ECG or stress imaging study.	2a B-NR
Addition of CAC testing to stress testing.	2a B-NR
Stress imaging after inconclusive CCTA.	2a B-2
CCTA or ICA after a negative stress test but with high clinical suspicion of CAD.	2b C-EO
Acute Chest Pain in patients with prior CABG surgery	
Stress imaging to evaluate for myocardial ischemia or CCTA for graft stenosis or occlusion in patients who do not have ACS	1 C-LD
ICA in patients who do not have ACS or who have an indeterminate/nondiagnostic stress test	1 C-LD
Evaluation of Patients With Acute Chest Pain Receiving Dialysis	
In patients who experience acute unremitting chest pain while undergoing dialysis, transfer by EMS to an acute care setting.	1 B-NR
Evaluation of Acute Chest Pain in Patients With Cocaine and Methamphetamine Use	
In patients presenting with acute chest pain, consider cocaine and methamphetamine use as a cause of their symptoms.	2a B-NR
Shared Decision-Making in Patients With Acute Chest Pain	
For patients with suspected ACS who are deemed low risk by a CDP, patient decision aids to improve understanding and effectively facilitate risk communication.	1 B-R
For patients with suspected ACS who are deemed intermediate risk by a CDP, shared decision-making between the clinician and patient regarding the need for admission, for observation, discharge, or further evaluation in an outpatient setting for improving patient understanding and reducing low-value testing.	1 B-R

Table 15. AHA/ACC 2021 GL on Chest Pain. Patients with Stable Chest Pain

Low-Risk Patients With Stable Chest Pain and No Known CAD

For patients presenting to the outpatient clinic, 1 B-NR
a model to estimate pretest probability of obstructive CAD is effective to identify patients at low risk for obstructive CAD and favorable prognosis in whom additional diagnostic testing can be deferred.

For patients categorized as low risk, 2a B-R
CAC testing as a first-line test for excluding calcified plaque and identifying patients with a low likelihood of obstructive CAD.

For patients categorized as low risk, exercise testing without 2a B-NR
imaging as a first-line test for excluding myocardial ischemia and determining functional capacity in patients with an interpretable ECG.

Intermediate-High Risk Patients With Stable Chest Pain and No Known CAD

Anatomic Testing

CCTA for diagnosis of CAD, for risk stratification, and for guiding treatment decisions. 1 A

Stress Testing

Stress imaging (stress echocardiography, PET/SPECT MPI or CMR) 1 B-R
for diagnosis of myocardial ischemia and for estimating risk of MACE.

For patients for whom rest/stress nuclear MPI is selected, PET 2a B-R
in preference to SPECT, if available to improve diagnostic accuracy and decrease the rate of nondiagnostic test results.

Exercise electrocardiography for patients with an interpretable ECG and ability 2a B-R
to achieve maximal levels of exercise (≥ 5 METs).

In patients selected for stress MPI using SPECT, use of attenuation correction 2b B-R
or prone imaging may be reasonable to decrease the rate of false-positive findings.

Assessment of Left Ventricular Function

In patients with pathological Q waves, symptoms or signs suggestive of heart failure, 1 B-NR
complex ventricular arrhythmias, or a heart murmur with unclear diagnosis,
TTE for diagnosis of resting left ventricular systolic and diastolic ventricular
function and detection of myocardial, valvular, and pericardial abnormalities.

Sequential or Add-on Testing: What to Do if Index Test Results are Positive or Inconclusive

For patients with known coronary stenosis of 40% to 90% in a proximal or middle 2a B-NR
coronary segment on CCTA, FFR-CT for diagnosis of vessel-specific
ischemia and to guide decision-making regarding the use of coronary revascularization.

CCTA after an inconclusive or abnormal exercise ECG or stress imaging study. 2a B-NR

Addition of CAC testing to stress testing.	2a B-NR
Stress imaging after inconclusive CCTA.	2a B-2
CCTA or ICA after a negative stress test but with high clinical suspicion of CAD.	2b C-EO

Table 16. AHA/ACC 2021 GL on Chest Pain. Evaluation of Acute Chest Pain With Nonischemic Cardiac Pathologies

Acute Chest Pain With Nonischemic Cardiac Pathologies

TTE for diagnosis in patients in whom other potentially life-threatening nonischemic cardiac conditions are suspected (eg, aortic pathology, pericardial effusion, endocarditis). 1 C-EO

Acute Chest Pain With Suspected Acute Aortic Syndrome

In clinical concern for aortic dissection, computed tomography angiography (CTA) of the chest, abdomen, and pelvis for diagnosis and treatment planning. 1 C-EO

In clinical concern for aortic dissection, transesophageal echocardiography (TEE) or CMR if CT is contraindicated or unavailable. 1 C-EO

Acute Chest Pain With Suspected PE

In stable patients with acute chest pain with high clinical suspicion for PE, CTA using a PE protocol. 1 B-NR

Need for further testing should be guided by pretest probability. 1 C-EO

Acute Chest Pain With Suspected Myopericarditis

In patients with myocardial injury who have nonobstructive coronary arteries on anatomic testing, CMR with gadolinium contrast to distinguish myopericarditis from other causes, including myocardial infarction and nonobstructive coronary arteries (MINOCA). 1 B-NR

In patients with suspected acute myopericarditis, CMR if there is diagnostic uncertainty, or to determine the presence and extent of myocardial and pericardial inflammation and fibrosis. 1 B-NR

In patients with suspected myopericarditis, TTE to determine the presence of ventricular wall motion abnormalities, pericardial effusion, valvular abnormalities, or restrictive physiology. 1 C-EO

In patients with suspected acute pericarditis, noncontrast or contrast cardiac CT scanning to determine the presence and degree of pericardial thickening. 2b C-EO

Acute Chest Pain With Valvular Heart Disease (VHD)

In patients presenting with suspected or known history of VHD, TTE for determining the presence, severity, and cause of VHD. 1 C-EO

In patients with suspected or known VHD in whom TTE diagnostic quality is inadequate, TEE (with 3D imaging if available) for determining the severity and cause of VHD. 1 C-EO

In patients presenting with known or suspected VHD, CMR imaging as an alternative to TTE and/or TEE is nondiagnostic.	2a C-EO
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Table 17. AHA/ACC 2021 GL on Chest Pain. Evaluation of Acute Chest Pain With Suspected Noncardiac Causes

Acute Chest Pain With Suspected Noncardiac Causes

Evaluate patients for noncardiac causes if they have persistent or recurring symptoms despite a negative stress test or anatomic cardiac evaluation, or a low-risk designation by a CDP.	1 C-EO
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Evaluation of Acute Chest Pain With Suspected Gastrointestinal Syndromes

Evaluation for gastrointestinal causes in patients with recurrent acute chest pain without evidence of a cardiac or pulmonary .	2a C-LD
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Evaluation of Acute Chest Pain With Suspected Anxiety and Other Psychosomatic Considerations

Referral to a cognitive-behavioral therapist of patients with recurrent, similar presentations for acute chest pain with no evidence of a physiological cause on prior diagnostic evaluation including a negative workup for myocardial ischemia. 2a B-R

Evaluation of Acute Chest Pain in Patients With Sickle Cell Disease

Emergency transfer by EMS to an acute care setting of patients with sickle cell disease who report acute chest pain.	1 B-NR
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In patients with sickle cell disease who report acute chest pain, ACS should be excluded.	1 C-LD
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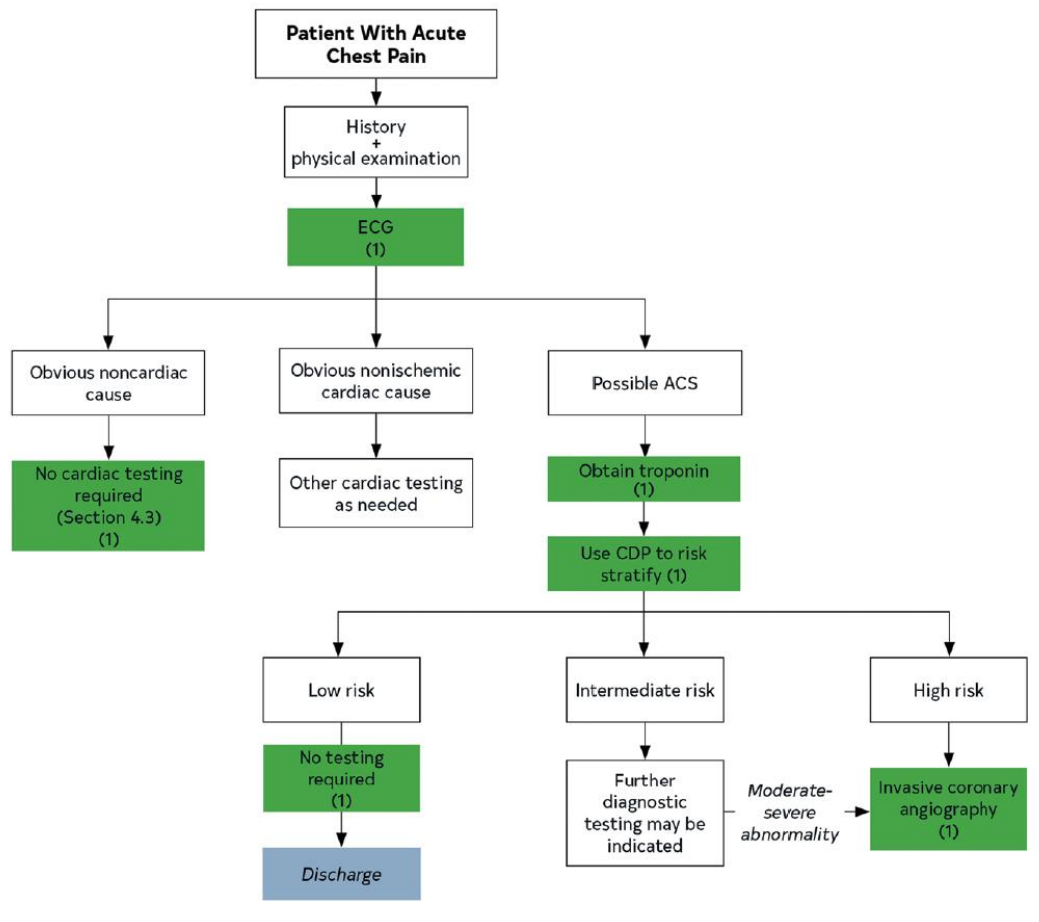


Figure 18. AHA/ACC 2021 GL on Chest Pain. General Approach to Risk Stratification of Patients With Suspected ACS

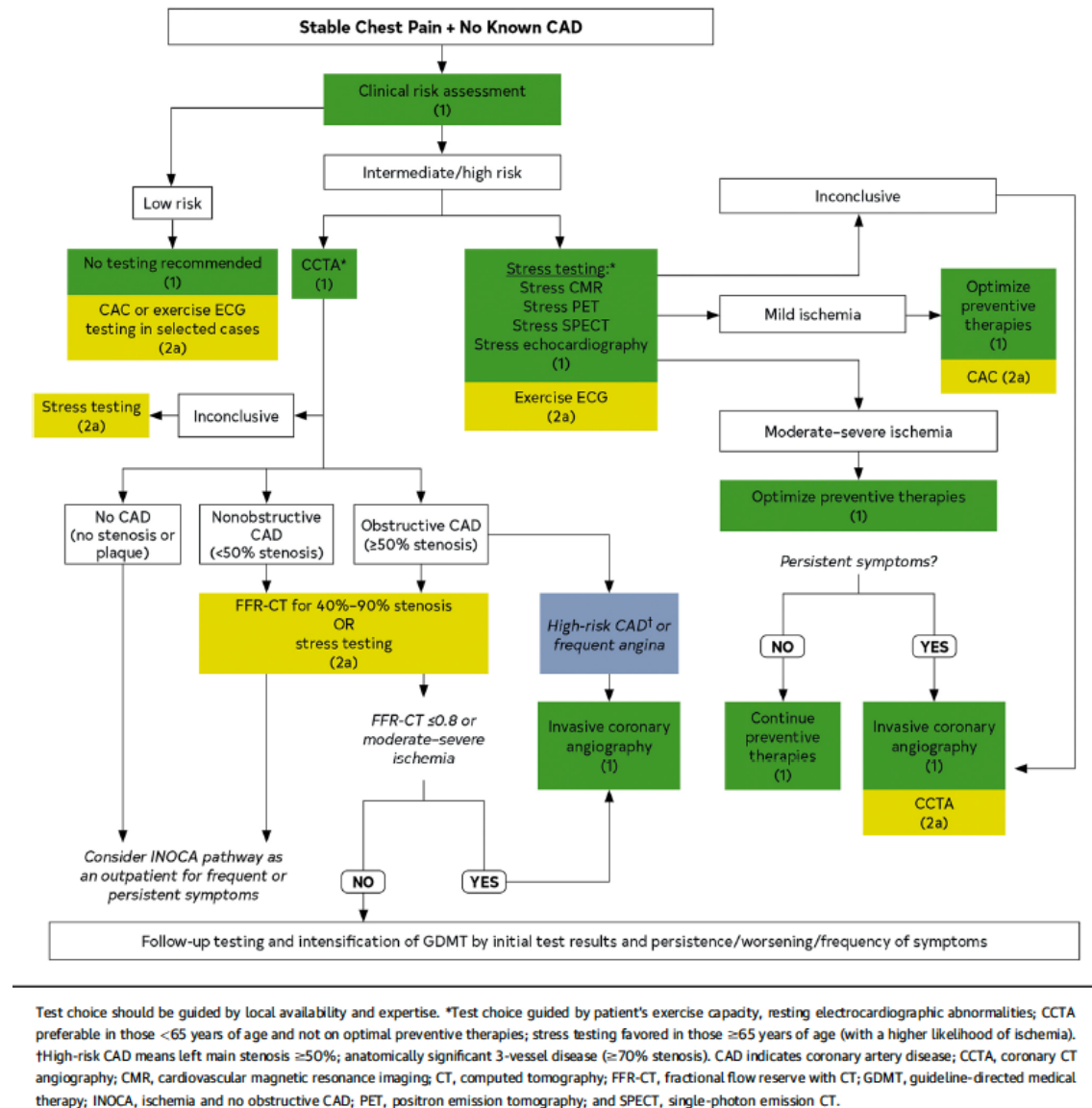
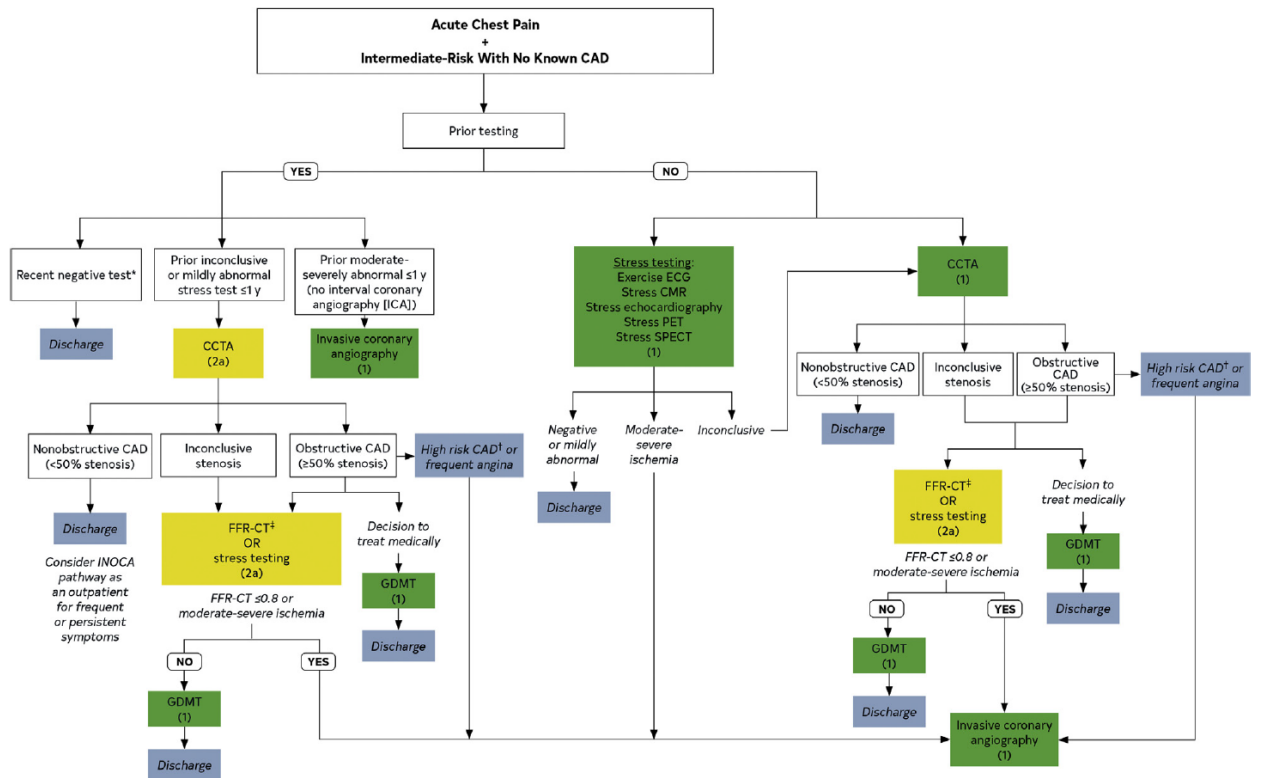


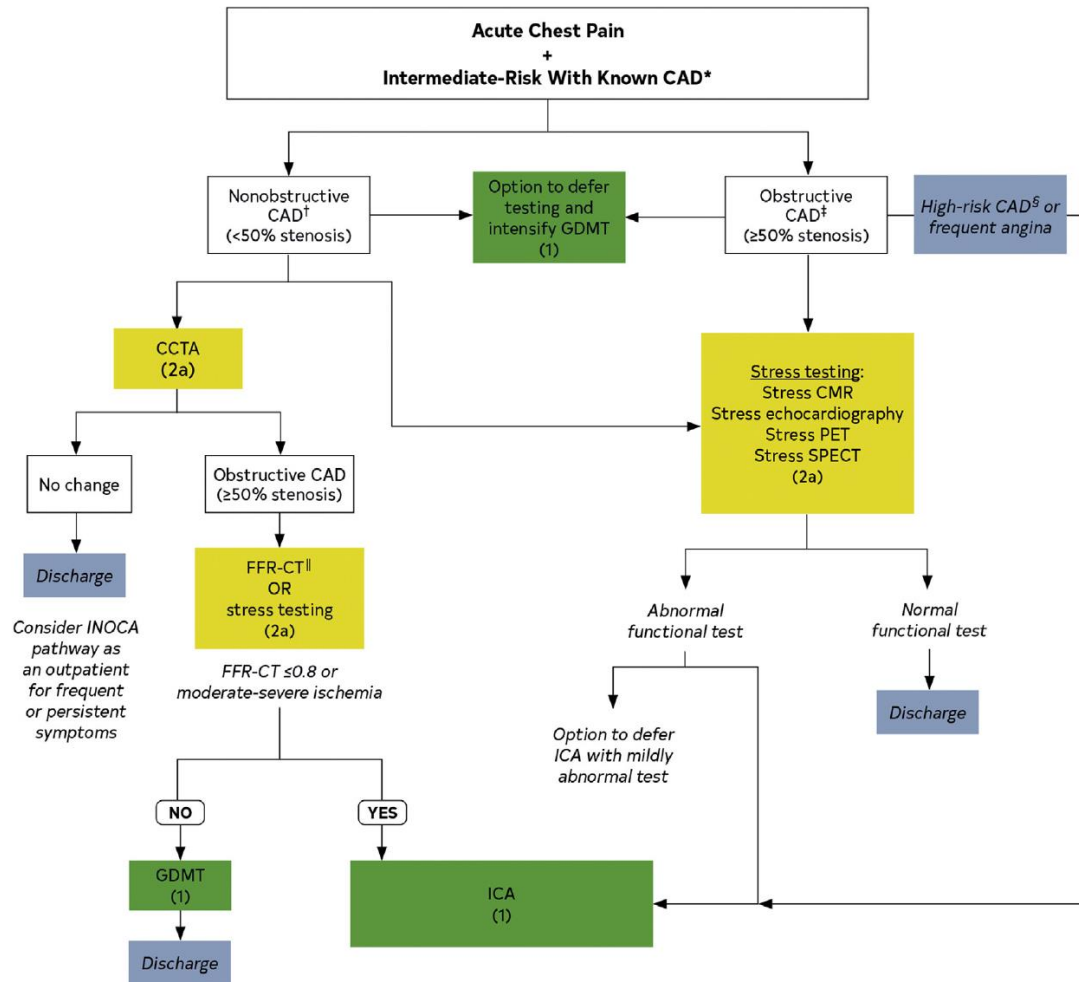
Figure 19. AHA/ACC 2021 GL on Chest Pain. Clinical Decision Pathway for Patients With Stable Chest Pain and No Known CAD

ACS indicates acute coronary syndrome; CDP, clinical decision pathway; and ECG, electrocardiogram.



Test choice should be guided by local availability and expertise. *Recent negative test: normal CCTA ≤ 2 years (no plaque/no stenosis) OR negative stress test ≤ 1 year, given adequate stress. †High-risk CAD means left main stenosis $\geq 50\%$; anatomically significant 3-vessel disease ($\geq 70\%$ stenosis). ‡For FFR-CT, turnaround times may impact prompt clinical care decisions. However, the use of FFR-CT does not require additional testing, as would be the case when adding stress testing. CAD indicates coronary artery disease; CCTA, coronary CT angiography; CMR, cardiovascular magnetic resonance imaging; CT, computed tomography; FFR-CT, fractional flow reserve with CT; GDMT, guideline-directed medical therapy; ICA, invasive coronary angiography; INOCA, ischemia and no obstructive coronary artery disease; PET, positron emission tomography; and SPECT, single-photon emission CT.

Figure 20. AHA/ACC 2021 GL on Chest Pain. Evaluation Algorithm for Patients With Suspected ACS at Intermediate Risk With No Known CAD



Test choice should be guided by local availability and expertise. *Known CAD is prior MI, revascularization, known obstructive or nonobstructive CAD on invasive or CCTA. †If extensive plaque is present a high-quality CCTA is unlikely to be achieved, and stress testing is preferred ‡Obstructive CAD includes prior coronary artery bypass graft/percutaneous coronary intervention. §High-risk CAD means left main stenosis $\geq 50\%$; anatomically significant 3-vessel disease ($\geq 70\%$ stenosis). ||FFR-CT turnaround times may impact prompt clinical care decisions. ACS indicates acute coronary syndrome; CAD, coronary artery disease; CCTA, coronary CT angiography; CMR, cardiovascular magnetic resonance; CT, computed tomography; FFR-CT, fractional flow reserve with CT; GDMT, guideline-directed medical therapy; ICA, invasive coronary angiography; INOCA, ischemia and no obstructive coronary artery disease; PET, positron emission tomography; and SPECT, single-photon emission CT.

Figure 21. AHA/ACC 2021 GL on Chest Pain. Evaluation Algorithm for Patients With Suspected ACS at Intermediate Risk With Known CAD

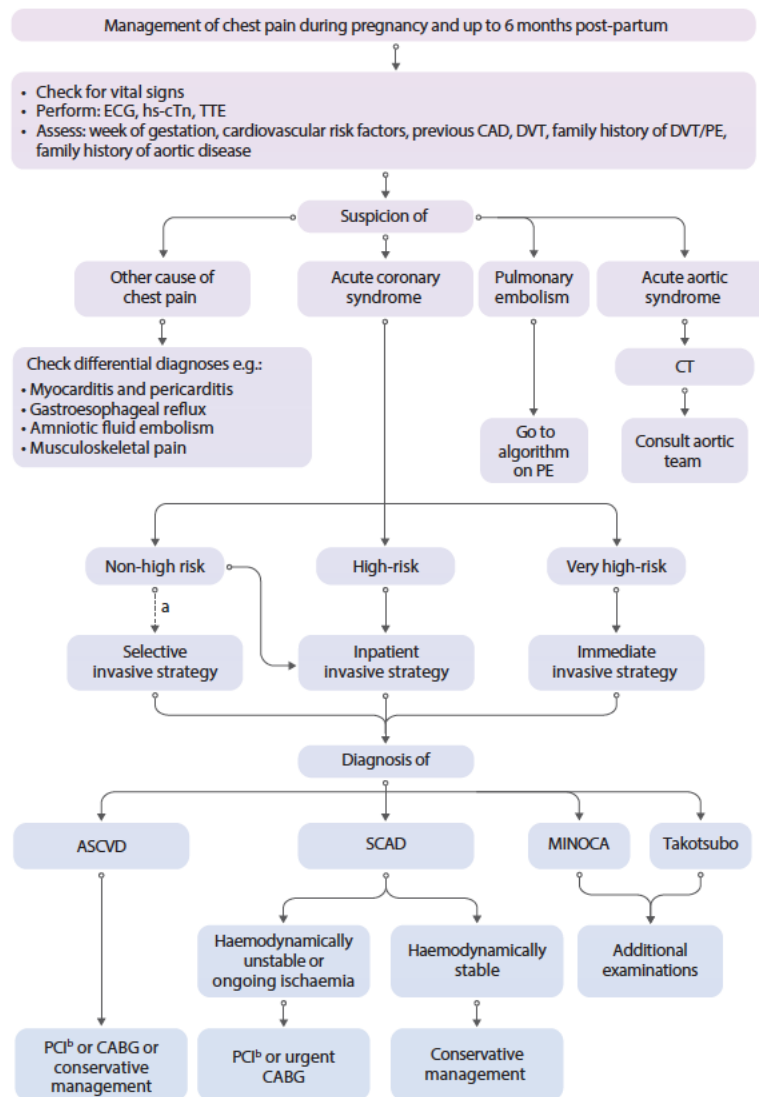


Figure 22. ESC 2025 GL on Pregnancy. Management of chest pain during pregnancy and within 6 months post-partum.

ASCVD, atherosclerotic cardiovascular disease; B-blocker, betablocker; CABG, coronary artery bypass graft; CAD, coronary artery disease; CT, computed tomography; DVT, deep venous thrombosis; ECG, electrocardiogram; EF, ejection fraction; hs-cTn, high sensitivity cardiac troponin; i.v., intravenous; MINOCA, myocardial infarction with non-obstructive coronary arteries; o.d., once a day; PCI, percutaneous coronary intervention; PE, pulmonary embolism; SCAD, spontaneous coronary artery dissection; TTE, transthoracic echocardiogram.

^aIn patients without very high or high-risk features and a low index of suspicion for unstable angina.

^bDual anti-platelet therapy:

clopidogrel: loading dose of 300–600 mg orally, followed by an oral maintenance dose of 75 mg o.d.
Aspirin: loading dose of 150–300 mg orally or 75–250 mg i.v. if oral ingestion is not possible, followed by an oral maintenance dose of 75–100 mg o.d.

Table 18. ACC/AHA 2025 GL on ACS. Selected Risk Stratification Tools for Patients With ACS

GRACE Risk Score (2.0)		TIMI Risk Score for Unstable Angina/NSTEMI*	TIMI Risk Score for STEMI
Target population	ACS	Unstable angina or NSTEMI	STEMI
Target outcome(s)	In-hospital, 6-mo, 1-y, 3-y, death or death/MI	14-d all-cause death, MI, or urgent revascularization	30-d all-cause death
Variables used	In-hospital risk score Age Killip class Systolic blood pressure Heart rate ST-segment deviation Cardiac arrest on admission Serum creatinine Elevated cardiac biomarkers	Age ≥ 65 y ≥ 3 risk factors for CAD Known coronary stenosis ($\geq 50\%$) ST-segment deviation ≥ 0.5 mm ≥ 2 anginal events in prior 24 h Aspirin use in prior 7 d Elevated cardiac biomarkers (CK-MB or troponin)	Age (65-74 y/ ≥ 75 y) (2-3 points) Killip class II-IV (2 points)* Systolic blood pressure (<100 mm Hg, 3 points) Heart rate >100 bpm (2 points) Anterior ST-segment elevation or LBBB (1 point) Diabetes/hypertension/angina (1 point) Weight (<67 kg, 1 point) Time to treatment >4 h (1 point)

*Score 1 point for each characteristic that is present.

ACS indicates acute coronary syndromes; bpm, beats per minute; CAD, coronary artery disease; CK-MB, creatine kinase-myocardial band; GRACE, Global Registry of Acute Coronary Events; LBBB, left bundle branch block; MI, myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction; and TIMI, Thrombolysis In Myocardial Infarction.

Table 19. AHA/ACC 2021 GL on Chest Pain. Low-Risk Patients With Acute Chest Pain

1	B-NR	1. Patients with acute chest pain and a 30-day risk of death or MACE $<1\%$ should be designated as low risk. ¹⁻¹¹
2a	B-R	2. In patients with acute chest pain and suspected ACS who are deemed low-risk ($<1\%$ 30-day risk of death or MACE), it is reasonable to discharge home without admission or urgent cardiac testing. ¹²⁻¹⁶