Diagnosis of acute myocardial infarction and patient risk stratification

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A 12-lead ECG is crucial in identifying patients with ST-elevation myocardial infarction (STEMI), who require rapid reperfusion therapy. Measurement of cardiac enzymes contributes to risk stratification in those with non-STEMI acute coronary syndrome to help determine appropriate management.

Key points

- **Acute myocardial infarction (MI) can be grouped into ST-elevation and non ST-elevation MI.**
- **Diagnosis of MI requires review of the patient's symptoms and signs, ECG patterns and troponin levels.**
- **Some patients do not present with the chest pain characteristic of MI; in these patients, angina-equivalent criteria need to be considered.**
- **Simple risk scores such as the TIMI and HEART scores can help risk-stratify patients and determine the most appropriate treatment strategy; these scores may be useful for GPs as well as in the emergency department.**

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Acute myocardial infarction (MI) remains the leading cause of death in Australia and worldwide. It is estimated that over 350,000 Australians have had a heart attack at some time in their lives. Each year, around 54,000 Australians have a heart attack. This equates to one heart attack every nine minutes.[1]

GPs are often the first point of contact for a person experiencing a heart attack. Expeditious diagnosis and prompt ambulance transfer to hospital can improve the prognosis of patients with this potentially fatal event.

Definition of myocardial infarction

Acute MI refers to cardiomyocyte necrosis in a clinical setting consistent with acute myocardial ischaemia.[2] Recent definitions of MI are based heavily on the presence of cardiac biomarkers (troponins). A combination of criteria must be met for the diagnosis of acute MI (Box).[2] Definitions of MI include:

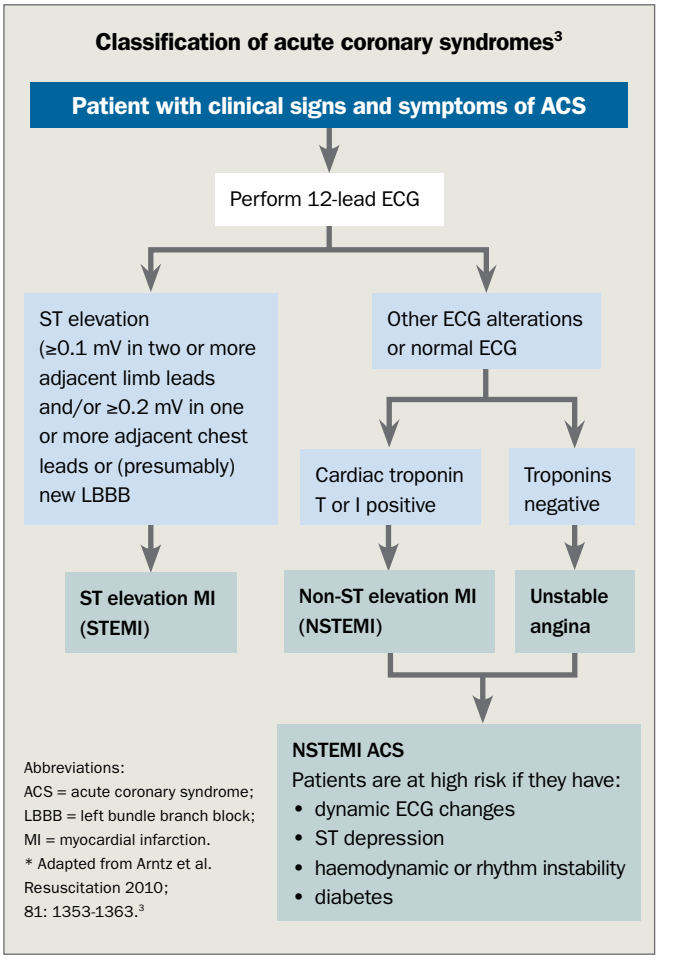
- detection of an increase and/or decrease in a cardiac biomarker, preferably high-sensitivity cardiac troponin, with at least one value above the 99th percentile of the upper reference limit plus
- at least one of the following:
  - symptoms of ischaemia
  - new, or presumed new, significant ST segment–T wave (ST–T) changes or new left bundle branch block
  - development of pathological Q waves on an ECG
  - imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
  - intracoronary thrombus detected on angiography or autopsy.

In patients with a raised troponin level, it is important to consider acute coronary syndrome (ACS) – spontaneous MI related to ischaemia caused by a primary coronary event such as plaque erosion, rupture, fissuring or dissection. However, other MI types have been defined. For example, in patients with a myocardial demand–supply mismatch, a troponin increase can be seen (signifying MI) in the absence of

Universal definition of myocardial infarction*

- **Detection of an increase and/or decrease in a cardiac biomarker,** preferably high-sensitivity cTn, with at least one value above the 99th percentile of the URL and at least one of the following:
  - symptoms of ischaemia
  - new, or presumed new, significant ST segment–T wave changes or new LBBB
  - development of pathological Q waves on an ECG
  - imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
  - intracoronary thrombus detected on angiography or autopsy.
- **Cardiac death** with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or new LBBB, but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would have been increased.
- **Percutaneous coronary intervention-related MI** is arbitrarily defined by elevation of cTn values (>5 x 99th percentile URL) in patients with normal baseline values (≤99th percentile URL), or a rise of cTn values >20% if the baseline values are elevated and are stable or falling. In addition, either (i) symptoms suggestive of myocardial ischaemia, or (ii) new ischaemic ECG changes, or (iii) angiographic findings consistent with a procedural complication, or (iv) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required.
- **Stent thrombosis-associated with MI** when detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac biomarker values with at least one value above the 99th percentile URL.
- **Coronary artery bypass grafting-related MI** is arbitrarily defined by elevation of cardiac biomarker values (>10 x 99th percentile URL) in patients with normal baseline cTn values (≤99th percentile URL). In addition, either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Abbreviations: cTn = cardiac troponin; LBBB = left bundle branch block; MI = myocardial infarction; URL = upper reference limit.
* According to the European Society of Cardiology, American College of Cardiology Foundation, American Heart Association and World Heart Federation.[2]



plaque rupture; this is defined as type 2 MI. An example is in patients with sepsis, where the oxygen demand of the heart increases. If the blood supply to the myocardium cannot keep up with this demand then the troponin level rises. Other types of MI in the absence of ACS are described in the Box: cardiac death (type 3 MI), percutaneous coronary intervention-related MI (type 4a), stent thrombus-associated MI (type 4b) and coronary artery bypass grafting-related MI (type 5).

Clinical classification of myocardial infarction

The term ACS refers to a spectrum of conditions compatible with acute myocardial ischaemia and/or infarction that are usually caused by an abrupt reduction in coronary blood flow. These conditions include unstable angina, ST-elevation MI (STEMI) and non-ST-elevation MI (NSTEMI).

There are many different ways to classify acute MIs. A common clinical classification system based on ECG findings is shown in the flowchart.[3] This system distinguishes two MI types:

- STEMI, which is marked by persistent (over 20 minutes) ST elevation or new left bundle branch block
- NSTEMI, which lacks persistent ST elevation or new left bundle branch block; ECG changes may include transient ST-segment elevation, ST-segment depression or T-wave changes, or the ECG may be normal.

STEMI generally results from red, fibrin-rich, occlusive thrombus. Restoring coronary patency as promptly as possible by primary angioplasty or fibrinolytic therapy is a key determinant of short-term and long-term outcome. In patients with STEMI, 'time equals muscle', and a delay in treatment can often lead to death or heart failure and other complications that adversely affect prognosis and quality of life.

In contrast, NSTEMI is generally associated with white, platelet-rich partially occlusive thrombus. The clinical spectrum of NSTEMI varies, and the treatment strategy, including the timing of coronary angiography or revascularisation, depends on risk

assessment using common clinical risk assessment scores.[4-9]

Unstable angina is defined as myocardial ischaemia at rest or on minimal exertion in the absence of cardiomyocyte necrosis. Patients with unstable angina have a better prognosis than those with NSTEMI and are less likely to benefit from early invasive angiography or revascularisation.

Diagnosis of myocardial infarction

Symptoms and signs

Chest pain or discomfort

Important characteristics of chest pain that help to differentiate cardiac and noncardiac pain include the location, character, duration and relationship of the pain to exertion and other aggravating or alleviating factors. The characteristic chest discomfort associated with MI is often termed 'prolonged ischaemic-sounding chest pain'. The pain is usually severe, with a duration longer than 10 minutes and a limited response to nitrates and commonly used analgesics.

The usual chest pain associated with myocardial ischaemia can be described with the following mnemonic:

- P = provocative and palliative (alleviative) factors: worse with exercise, better with rest or glyceryl trinitrate (sublingual nitrate)
- Q = quality and quantity of the pain: pressure, tightness, crushing, poorly localised, lasting more than 10 minutes. Pain that is short-lived (less than a minute) is unlikely to be cardiac
- R = region and radiation of the pain: usually retrosternal, radiating to the jaw or the left arm or both arms; however, any pain between the umbilicus and the oral cavity could raise concern for cardiac pain in the appropriate clinical setting
- S = severity of the pain: usually rated as moderate to severe by the patient
- T = together with shortness of breath, sweating, nausea, syncope or dizziness.

Angina equivalents

It is important to remember that not all patients with MI have pain with the usual characteristics. In particular, women, elderly patients, patients with dementia and those with diabetes may not describe typical ischaemic-sounding chest pain. In addition, patients with distractive pain (e.g. a concurrent hip fracture) may underestimate or not report chest pain. The diagnosis of MI should be considered with presenting symptoms termed 'angina equivalents', especially in the above groups of patients.

Angina equivalents can include but are not limited to:

- shortness of breath
- any discomfort or pain between the umbilicus and the mouth (including epigastric pain or indigestion-like symptoms)
- exertional throat pain or pain in the arms, more commonly the medial aspect of the left arm
- nausea
- extreme fatigue
- dizziness
- belching on exertion.[10]

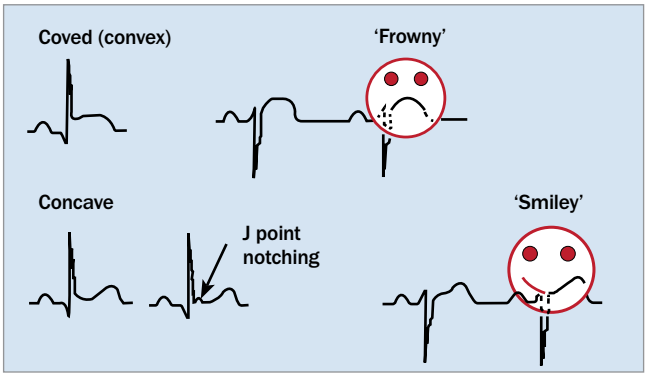


Figure 1. Comparison of ST elevation between myocardial infarction and more benign causes such as pericarditis or early repolarisation. a (top). An elevated ST segment that is coved (convex), resembling a 'frowny' face, is more likely to be caused by the acute injury of myocardial infarction. b (bottom). ST elevation that is concave, resembling a 'smiley' face, is usually benign, especially when J-point notching is seen.

Although the history of presenting symptoms is very important in diagnosing ACS, it lacks both sensitivity and specificity. Sometimes a patient strongly denies the presence of chest pain but admits to having 'indigestion' that worsens on exertion. Consequently, it is safer to use the term 'chest discomfort' than 'chest pain' when interviewing patients. For patients with angina-equivalent symptoms, the ECG and measurement of cardiac enzymes (including troponin levels) may be more reliable diagnostic tools than the history alone to diagnose ACS.

ECG diagnostic criteria

For patients with possible ACS, the 12-lead ECG obtained within 10 minutes of arrival at medical contact quickly identifies patients with STEMI. These patients require rapid reperfusion by percutaneous coronary intervention in the cardiac catheterisation laboratory, or the administration of fibrinolytic therapy if cardiac catheterisation will be delayed.

Delay in the diagnosis of MI can cause the window of opportunity for immediate thrombolysis or primary angioplasty to be missed. Although identifying ST segment elevation in a standard 12-lead ECG is the important criterion for the immediate reperfusion strategy, a number of clinical conditions can result in ST elevation, mimicking acute MI (see below).

STEMI

ST elevation in acute MI is typically confined to a vascular territory and is convex upward (Figure 1).[11] Often there is reciprocal ST depression in the opposite leads. In the early stages of acute MI, the ECG may show tall peaked T waves followed by ST segment elevation, generally defined as more than 1 mm in limb leads (leads I, II, III, aVL and aVF) or more than 2 mm in precordial leads (leads V1 to

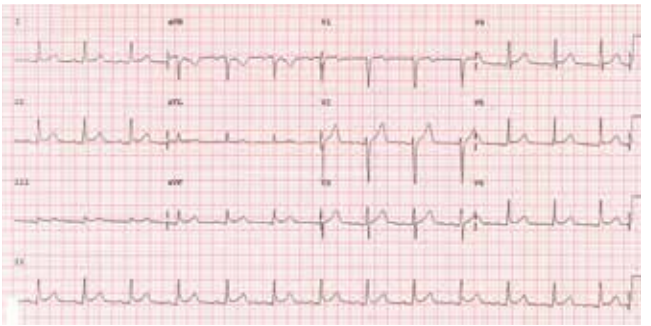


Figure 2. ECG in a patient with pericarditis, showing widespread concave ('saddleback') ST segments (leads I, II avF and V6 in this instance), PR depression (more pronounced in inferior leads) and PR elevation (aVR).
ECG courtesy of Dr Haris Haqqani, Prince Charles Hospital, Brisbane, Qld.

V6). As time progresses, the ECG may show Q waves and T-wave inversion. Of note, the pathological Q wave is defined as a Q wave with a depth more than one-quarter of the height of the R wave in the same QRS complex.

NSTEMI

In a patient with an NSTEMI, the ECG changes may be more subtle. In fact, these patients may have no apparent ECG changes. ST changes such as ST depression, symmetrical T-wave inversion or (usually in more prolonged symptoms) the presence of Q waves in any leads other than aVR can increase the suspicion of an NSTEMI but are not required for the diagnosis. T-wave inversion in the V1 lead can be normal.

Posterior STEMI

A posterior STEMI can sometimes be hard to identify. It should be suspected when the ECG shows:

- ST depression greater than 1 mm in the V1 to V3 leads
- a tall R wave in the V1 or V2 lead
- a tall, upright T wave in the V1 or V2 lead.

An easy way to recognise a posterior MI is to 'flip up' the ECG and look at leads V2 to V4. If ST elevation can be seen in these leads while looking through the reverse side of the ECG then a posterior STEMI is diagnosed. The rationale is that as leads V2 to V4 lie opposite the posterior wall of the heart, ST depression in these leads is equivalent to ST elevation in the posterior leads.

A posterior MI could also be diagnosed by recording an ECG with leads V7 to V9 on the posterior chest wall. In practice this means running the precordial leads onto the back of the chest and placing the electrodes in the same plane as V6 (V7 = left posterior axillary line, V8 = tip of left scapula, V9 = left paraspinal region). A posterior MI exists if there is ST elevation in any of leads V7 to V9.

Non-MI causes of elevated ST segments

Not all patterns of elevated ST segments signify MI. Other conditions that can cause ST elevation and mimic acute MI include the following.

Pericarditis

A typical history of pericarditis is a sharp stabbing pain that is worse when the patient is lying down and lessens when they are sitting up and leaning forward. In patients with acute pericarditis, the ST segment is diffusely elevated, with upward concavity in the precordial leads as well as the limb leads (Figures 1 and 2).[11] The ST elevation usually involves more than one coronary vascular territory, and usually there are no reciprocal ST changes. In pericarditis, the PR segment can also be depressed, but this finding is not specific for acute pericarditis.[12,13]

Early repolarisation

Early repolarisation is characterised by ST elevation with a concave morphology and notching of the J point (the junction where the QRS complex ends and the ST segment begins). It is seen in leads with a tall R wave and is most marked in leads V2 to V5 (Figure 3). Rarely, it can involve inferior leads (II, III and avF). Early repolarisation occurs in 2 to 5% of the population, predominantly in young males. The reciprocal changes classically seen in acute MI are generally absent, unless the early repolarisation is associated with ACS.[14] Early repolarisation is less common after the age of 50 years and

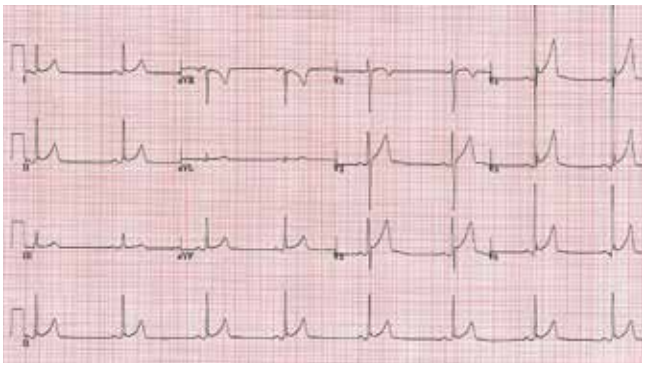


Figure 3. ECG in a patient with early repolarisation, showing J-point notching and concave upwards ST elevation in the precordial leads, in the absence of reciprocal ST depression.

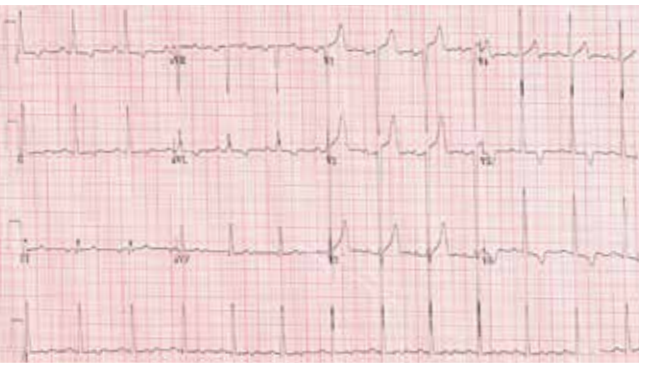


Figure 4. ECG showing typical changes of left ventricular hypertrophy, including deep S waves (marked in leads V1 and V2) with concave ST elevation and tall R waves (especially in leads V5 and V6).

Table 1. Thrombolysis in Myocardial Infarction (TIMI) risk score for patients with unstable angina and NSTEMI[19*]

Item	Score
History	
Age 65 years or older	1
Three or more risk factors	1
Known coronary artery disease (stenosis ≥50%)	1
Aspirin use in past 7 days	1
Presentation	
Recent (<24 hours) severe angina	1
Increased cardiac markers	1
ST deviation ≥0.5 mm	1
Maximum	7

Abbreviation: NSTEMI = non-ST elevation myocardial infarction.
* Risk stratification: rates of all-cause mortality or new or recurrent myocardial infarction or severe recurrent anginal chest pain requiring urgent revascularisation in 14 days are 4.7% (for score of 0 to 1), 8.3% (2), 13.2% (3), 19.9% (4), 26.2% (5), at least 40.9% (6 to 7).

rare after the age of 70 years. Therefore it should not be considered as a diagnosis in elderly patients unless there is evidence of previous longstanding ECG changes.

Left ventricular hypertrophy

Left ventricular hypertrophy is frequently associated with secondary ST-segment or T-wave abnormalities. The ST segments and T waves are directed opposite to the QRS complex, causing discordance between the QRS complex and the ST–T abnormalities. Typically there is ST segment elevation in the precordial leads VI to V3, where the QRS complex predominates (Figure 4). Patients may have a history of hypertension or a family history of hypertrophic cardiomyopathy.

Takotsubo cardiomyopathy

Takotsubo cardiomyopathy mimics acute MI in presentation and may be considered in any postmenopausal woman who presents with chest pain following an intense emotional or physical stress. The most common presenting symptom of takotsubo cardiomyopathy is acute chest pain. The most common acute ECG findings are ST-segment elevation in the precordial leads and T-wave inversions in most leads. The ECG changes in takotsubo cardiomyopathy, in contrast to those in acute MI, are not limited to a single vascular territory.[15] Importantly, takotsubo cardiomyopathy is a diagnosis of exclusion that should be made only after sufficient investigation. If there is any suspicion of STEMI then these patients should be treated as if they have STEMI until proven otherwise.

Table 2. Global Registry of Acute Coronary Events (GRACE) risk score[19*]

Item	Score	Item	Score
Killip (CHF) class		**Age (years)**	
I	0	≤30	0
II	20	30–39	8
III	39	40–49	25
IV	59	50–59	41
Systolic blood pressure (mmHg)		60–69	58
≤80	58	70–79	75
80–99	53	80–89	91
100–119	43	≥90	100
120–139	34	**Creatinine level (mg/dL)**	
140–159	24	0–0.39	1
160–199	10	0.40–0.79	4
≥200	0	0.80–1.19	7
Heart rate (beats per min)		1.20–1.59	10
≤50	0	1.60–1.99	13
50–69	3	2.00–3.99	21
70–89	9	≥4.00	28
90–109	15	**Other risk factors**	
110–149	2	Cardiac arrest at admission	39
150–199	38	ST-segment deviation	28
≥200	46	Elevated cardiac enzymes	14

Abbreviation: CHF = congestive heart failure.
* Total score = sum of scores for Killip class + systolic blood pressure + heart rate + age + creatinine level + cardiac arrest at admission + ST-segment deviation + elevated cardiac enzyme levels (score range, 1 to 372). Interpretation of the score is complex; for more information refer to the GRACE website (http://www.outcomes-umassmed.org/GRACE/grace_risk_table.aspx).

Cardiac enzymes

Cardiac troponin is a highly sensitive biomarker of myocardial injury and is used in the diagnosis of acute MI and for risk stratification of patients with acute coronary symptoms. In 2000, a joint committee of the European Society of Cardiology and the American College of Cardiology issued new criteria that acknowledged that elevations in biomarkers were fundamental to the diagnosis of acute MI.[16] The serum troponin level is very sensitive for detecting myocardial injury

Table 3. HEART score for patients with chest pain*[19]

Item	Score
History	
Highly suspicious	2
Moderately suspicious	1
Slightly suspicious	0
ECG	
Significant ST depression	2
Nonspecific repolarisation disturbance	1
Normal	0
Age	
≥65 years	2
45–65 years	1
<45 years	0
Risk factors	
≥3 risk factors or history of atherosclerotic disease	2
1 or 2 risk factors	1
No risk factors known	0
Troponin	
≥3 x normal limits	2
1–3 x normal limits	1
≤normal limit	0
Maximum	**10**
Risk stratification†	
Low risk	1 to 3
Medium risk	4 to 6
High risk	7+

* Adapted from Backus et al. Curr Cardiol Rev 2011; 7: 2-8.[19]
† Risk of major adverse coronary event (MACE) within the next 6 weeks (defined as all-cause mortality, myocardial infarction or coronary revascularisation).

from any cause, including acute MI, and in most situations a raised troponin level indicates an adverse prognosis. However, troponin lacks specificity for the diagnosis of ACS as the troponin level is also elevated in a number of other conditions, including sepsis, heart failure, renal failure, pulmonary embolism and pericarditis (termed 'nonischaemic troponin rise').[16-18] In general, an increased serum troponin level warrants further investigations and an expert opinion.

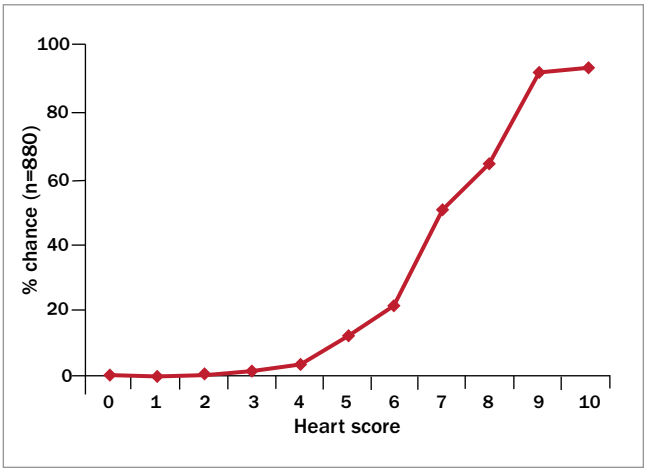


Figure 5. Probability of experiencing a major adverse coronary event (MACE) within the next six weeks by HEART score. Risk of MACE was 0.9% for patients with a HEART score of 1 to 3, 12% for a score of 4 to 6 and 65% for a score of 7 or more.[19]

Risk stratification in patients with MI

Patients with suspected ACS who have no ST-segment elevation on the ECG should undergo further observation and investigation to rule out other diagnoses, enable risk stratification and determine the most appropriate treatment strategy.

Risk assessment scores and clinical prediction algorithms that use clinical history, physical examination, ECG and cardiac troponin levels have been developed to help identify patients with ACS at increased risk of an adverse outcome.

TIMI and GRACE scores

Several scoring methods are commonly used to determine the risk of short-term and longer-term events in patients hospitalised with suspected ACS.[19] The Thrombolysis in Myocardial Infarction score (TIMI) and the Global Registry of Acute Coronary Events score (GRACE) are commonly known tools (Tables 1 and 2). Patients can be stratified into low, intermediate and high risk groups, helping determine appropriate in-hospital management (e.g. coronary angiography for those with high risk).

HEART score

The diagnosis of NSTEMI typically causes uncertainty. Classic considerations for risk stratification are the patient's history, ECG, age, risk factors and troponin level, which are combined in the HEART score. This is a prospectively studied scoring system to help emergency departments risk-stratify patients with chest pain and identify those who are likely to have a major adverse coronary event (MACE) within the next six weeks (defined as all-cause mortality, MI or coronary revascularisation). Advantages of the

HEART score for GPs are that all of the items are readily available in the GP surgery and it is easy to use.

The HEART score involves a one-time troponin measurement, at the point of care. The remainder of the score is based on age, history, risk factors and ECG results (Table 3). A score of 0 to 3 is defined as low risk, and patients have a less than 1% risk of MACE at six weeks (Figure 5). With higher scores the risk increases exponentially, and patients with a score of 4 or above (medium or high risk) require further management and hospital admission.

The HEART score is a strong predictor of event-free survival on the one hand and potentially life-threatening cardiac events on the other. Although the HEART score is sometimes compared with the older TIMI and GRACE scores, the latter measure risk of death for patients with ACS and are not as good at initially identifying those who have ACS. Additionally, major advantages of the HEART system over the other scoring systems are that it allows clinicians to reassure low-risk patients and to intervene more quickly for higher-risk patients. The HEART score mirrors clinical decision-making and does not require complicated calculations such as those needed for the GRACE score. Well-known markers of increased risk, such as older age, presence of risk factors and a history of coronary atherosclerosis, are all incorporated in the HEART score. This scoring system is recommended as it is easily and simply calculated by GPs working in emergency care or the community.

Conclusions

We present here the key diagnostic tools to aid in the diagnosis of STEMI and NSTEMI. In general, both presentations require prompt patient referral to the emergency department, preferably by ambulance. The GP is often the first medical contact for a patient with chest pain. The patient's symptoms and signs, ECG and, if possible, cardiac troponin level, can help differentiate acute MI from other conditions that mimic MI. The HEART score is a simple risk-stratification tool that may help GPs reassure low-risk patients and intervene more quickly for higher-risk patients. **CT**

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