Ischaemic heart disease: NSTE-ACS

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Abstract

Non-ST elevation acute coronary syndrome (NSTE-ACS) accounts for a substantial proportion of myocardial infarctions, with increasing incidence over the past two decades; this is partly attributed to the widespread use of high-sensitivity cardiac troponin assays, enabling earlier and more accurate detection of myocardial injury. NSTE-ACS predominantly affects older individuals, with a higher prevalence in those with co-morbid conditions such as hypertension, diabetes mellitus, dyslipidaemia and chronic kidney disease. While the short-term mortality is lower than in ST elevation ACS, individuals with NSTE-ACS exhibit more extensive coronary artery disease and co-morbidities, contributing to higher long-term mortality and recurrent ischaemic events. Diagnosis is primarily based on clinical presentation, electrocardiographic findings and elevated cardiac biomarkers, particularly high-sensitivity troponin, which confirms myocardial necrosis. Initial management involves stabilization with antiplatelet therapy, anticoagulation and prompt risk stratification, followed by invasive strategies such as percutaneous coronary intervention in high-risk patients. Secondary prevention focuses on pharmacological therapies including dual antiplatelet therapy, statins and β-adrenoceptor blockers, alongside lifestyle modifications. Despite favourable short-term outcomes with early intervention, the long-term prognosis remains dependent on effective management of the underlying risk factors and adherence to secondary prevention strategies, as recurrent cardiovascular events remain common.

Keywords Antiplatelet therapy; coronary angiography; coronary artery bypass surgery; GRACE score; non-ST elevation acute coronary syndrome; percutaneous coronary intervention; secondary prevention; troponin

Key points

- NSTE-ACS is the most common type of myocardial infarction (around 60–75%), predominantly affecting older adults with co-morbidities such as hypertension, diabetes and chronic kidney disease, and although it has lower early mortality than STEMI, it is associated with worse long-term outcomes due to more diffuse coronary disease
- It is caused by rupture or erosion of an atherosclerotic plaque leading to partial or intermittent coronary artery occlusion and formation of a non-occlusive thrombus, resulting in sub-endocardial ischaemia rather than full-thickness infarction
- Patients typically present with prolonged chest discomfort (pressure or tightness) that may radiate to the arm, neck or jaw, along with symptoms such as dyspnoea, nausea and diaphoresis, although atypical presentations (e.g. fatigue, epigastric pain or isolated dyspnoea) are common in elderly patients, women and those with diabetes or chronic kidney disease
- Diagnosis is based on clinical assessment, ECG changes such as ST segment depression or T wave inversion (which may be transient or initially absent, requiring serial ECGs), and a rise and/or fall in high-sensitivity cardiac troponin in the context of myocardial ischaemia, while imaging such as echocardiography and coronary angiography helps confirm the diagnosis and guide management
- Management involves rapid initial assessment and risk stratification (e.g. GRACE or TIMI score), early initiation of dual antiplatelet therapy and anticoagulation, and timely invasive management with coronary angiography and percutaneous coronary intervention in high-risk patients, followed by comprehensive secondary prevention including statins, β-blockers, angiotensin-converting enzyme inhibitors and lifestyle modification to reduce recurrent cardiovascular events

Epidemiology

Non-ST elevation acute coronary syndrome (NSTE-ACS) accounts for approximately 60–75% of acute myocardial infarctions (MIs) in developed countries. Its incidence has increased over recent decades, partly because of the widespread use of high-sensitivity cardiac troponin assays, enabling earlier detection.[1]

NSTE-ACS predominantly affects older adults and is commonly associated with co-morbidities such as hypertension, diabetes, dyslipidaemia and chronic kidney disease (CKD). Although it is more common in men, women have higher mortality rates, often related to atypical presentations and delayed diagnosis. Compared with ST-elevation ACS (STE-ACS), NSTE-ACS patients typically have more diffuse coronary disease and co-morbidities, leading to worse long-term outcomes despite lower early mortality.

Globally, the incidence of NSTE-ACS is increasing in low- and middle-income countries as risk factors such as obesity, diabetes and smoking become more prevalent, reflecting a shift in the global burden of coronary heart disease.

Pathophysiology

The pathophysiology of NSTE-ACS centres on partial or transient occlusion of a coronary artery, leading to subendocardial myocardial ischaemia and necrosis – without full-thickness (transmural) infarction as seen in STE-ACS.

Most cases arise from rupture or erosion of an atherosclerotic plaque, exposing thrombogenic material and triggering platelet

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activation and coagulation; this results in a non-occlusive thrombus. Reduced coronary blood flow, often combined with increased myocardial oxygen demand, preferentially affects the subendocardium because of its high metabolic requirements and reduced perfusion during systole. Unlike STE-ACS, occlusion is incomplete or intermittent, limiting the extent of infarction. Less commonly, NSTE-ACS occurs by non-atherosclerotic mechanisms.

Diagnosis

Clinical assessment

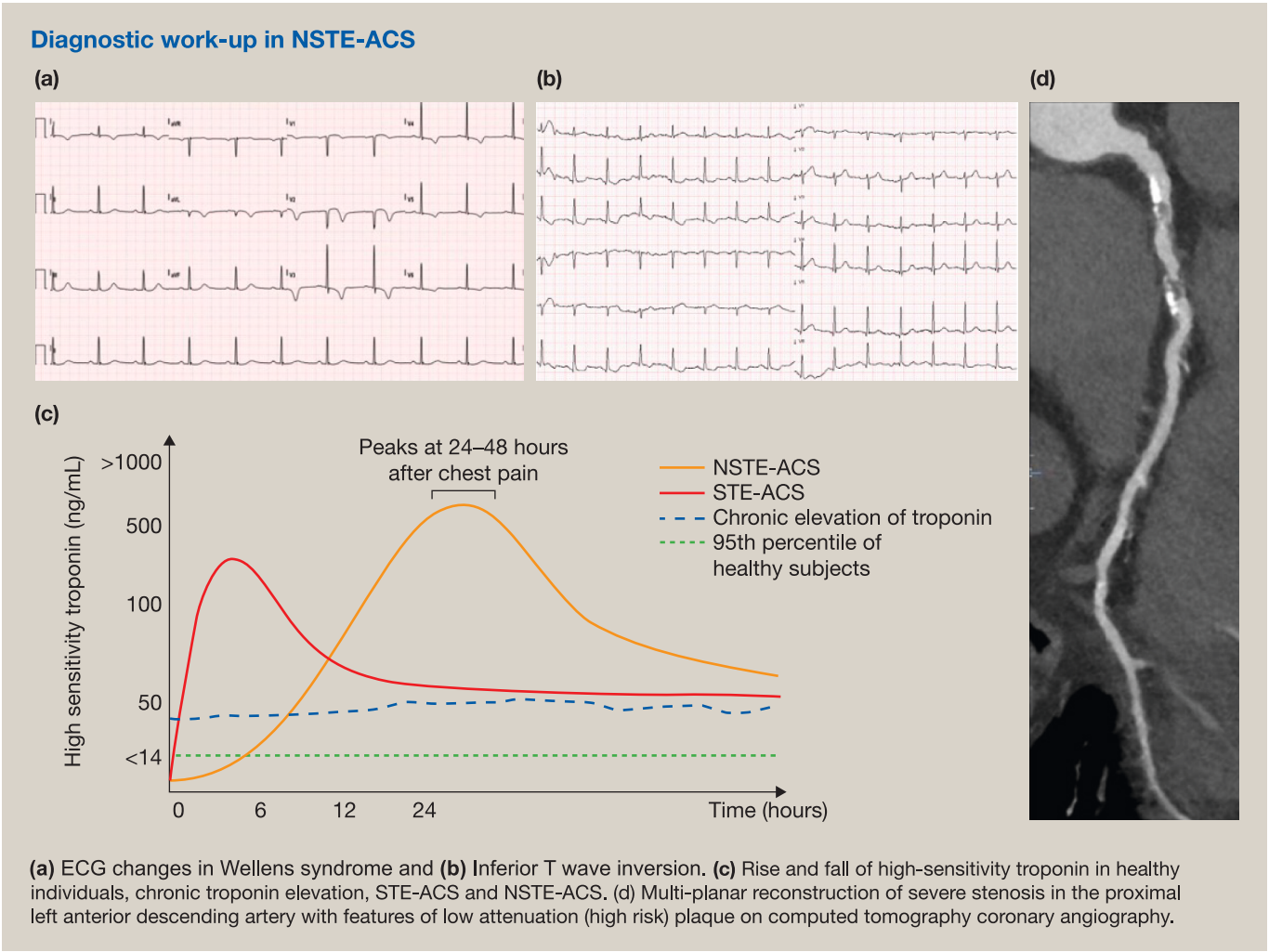
Symptoms are similar to other acute coronary syndromes (ACSs) but are often less severe or more variable than in STE-ACS. The typical presentation includes prolonged chest discomfort described as pressure or tightness, frequently radiating to the arm, neck, jaw or back, and occurring at rest or with minimal exertion. Associated features include dyspnoea, diaphoresis, nausea and palpitations.

Atypical presentations are common in older adults, women and individuals with diabetes or CKD, often manifesting as dyspnoea, fatigue or epigastric discomfort. These atypical manifestations often lead to delayed diagnosis and treatment, contributing to higher morbidity and mortality in these populations. Physical examination findings are frequently non-specific, although signs of heart failure can be present in more extensive ischaemia.

Investigations

Electrocardiogram (ECG) is the first-line and most urgent test and should be performed within 10 minutes of presentation. Typical findings included ST segment depression, T wave inversion or transient ST segment elevation (Figure 1). Initial ECGs can appear normal or non-specific so serial ECGs are usually necessary.

Cardiac biomarkers: the biochemical hallmark of NSTE-ACS is an elevation in high-sensitivity cardiac troponin (type I or T). Elevated troponin confirms myocardial necrosis and helps differentiate NSTE-ACS from unstable angina. High-sensitivity troponin assays allow for an earlier detection of these changes, with some algorithms allowing for diagnosis or exclusion within 1–3 hours.



(a) ECG changes in Wellens syndrome and **(b)** Inferior T wave inversion. **(c)** Rise and fall of high-sensitivity troponin in healthy individuals, chronic troponin elevation, STE-ACS and NSTE-ACS. (d) Multi-planar reconstruction of severe stenosis in the proximal left anterior descending artery with features of low attenuation (high risk) plaque on computed tomography coronary angiography.

Figure 1

Caution must be applied in that troponin can be elevated and can represent myocardial injury rather than myocardial ischaemia. This can be caused by critical illness (sepsis, acute renal failure, pulmonary embolism, severe burns), inflammatory and structural factors (myocarditis, infiltrative disease, aortic dissection) and neurological or traumatic events (stroke, seizure, trauma) (Figure 2). In clinical practice, distinguishing between injury and ischaemia can be difficult.

The fourth definition of MI emphasizes the role of high-sensitivity cardiac troponin assays and distinguishes between myocardial injury and MI.[2] Myocardial injury is defined by elevated cardiac troponin values >99th percentile upper reference limit; it is considered acute if there is a rise and/or fall in these values. For MI to be diagnosed, this must be in the context of myocardial ischaemia. The definition was updated to reflect the increased use of high-sensitivity troponin assays.

Typically, troponin concentrations rise within a few hours of symptom onset, peak around 18–24 hours and then gradually fall over the course of about 10 days (Figure 1). A rise or fall of at least 20% in troponin concentrations is needed for diagnosis, along with ischaemic symptoms and other evidence of ischaemic damage.[3]

Cardiac imaging: echocardiography is an essential early investigation in patients with NSTE-ACS to:

- quantify left ventricular function and regional wall-motion abnormalities supporting the diagnosis of ischaemia or infarction
- detect acute complications (papillary muscle dysfunction/acute mitral regurgitation, ventricular septal defect, free-wall rupture/tamponade)
- guide haemodynamic management when there is shock or new/worsening heart failure.

Echocardiography should be performed urgently at the bedside if the patient is haemodynamically unstable or has clinical signs of heart failure, a new systolic murmur or a suspected mechanical complication. In stable patients it is still recommended early during admission to inform risk stratification and decisions about the timing of angiography.

Routine resting echocardiography also provides a baseline for post-ACS care (ejection fraction-directed therapy, cardiac rehabilitation and follow-up), and specialized echocardiography (contrast, stress or transoesophageal) can be used selectively where image quality is poor or additional detail is required.

Computed tomography coronary angiography (CTCA) plays a supportive diagnostic role in NSTE-ACS, particularly in individuals with low to intermediate clinical risk when the diagnosis remains uncertain after ECG and high-sensitivity troponin testing.[4] It provides rapid, non-invasive visualization of the coronary anatomy and has a very high negative predictive value; this makes it especially useful for ruling out obstructive coronary artery disease in patients with atypical symptoms or equivocal biomarker results.

CTCA can help differentiate true ischaemic NSTE-ACS from non-coronary causes of chest pain, guide the need for invasive angiography and prevent unnecessary catheterization in low-risk patients. However, early CTCA does not affect primary outcomes in intermediate and high-risk patients who have a suspected NSTE-ACS.[5]

Coronary angiography is the gold standard technique to identify culprit coronary lesions and determine suitability for percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG). European Society of Cardiology guidelines recommend immediate invasive angiography (<2 hours) for very high-risk patients (haemodynamic instability or

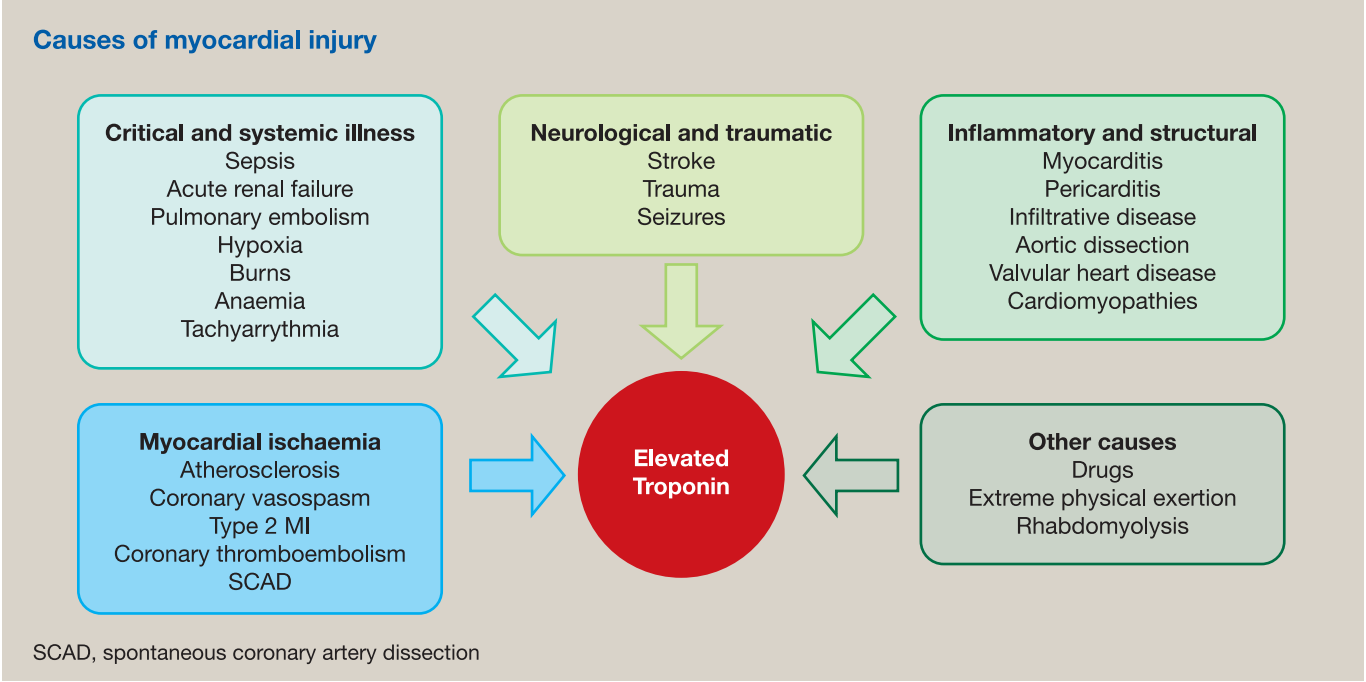


Figure 2

refractory pain) and an early invasive strategy (within 4 hours) for most other patients with a confirmed diagnosis of NSTE-ACS.

It should be noted that evidence for early invasive strategy is absent. Trials such as RAPID NSTEMI were not completed and previous randomized controlled trials had a narrow time differentiation between the early invasive group and standard care, so unsurprisingly there was no difference.

MI with non-obstructive coronary arteries (MINOCA) is a term used to describe patients with a provisional diagnosis of MI who are found to have non-obstructive or normal coronary arteries after coronary angiography. MINOCA is not a definitive diagnosis but an umbrella term for a heterogenous group of conditions that have caused, or initially been misdiagnosed as, MI (Table 1).

Risk stratification tools

The Global Registry of Acute Coronary Events 2.0 (GRACE 2.0), Thrombolysis In Myocardial Infarction (TIMI) and History, ECG, Age, Risk Factors and Troponin (HEART) scores are used to estimate mortality and guide the urgency of intervention. They comprise a combination of clinical, ECG and troponin indicators (Figure 3).

The purpose of these risk stratification tools is to identify high-risk patients, who benefit from an early invasive strategy (e.g. coronary angiography within 24 hours), guide antithrombotic and antiplatelet therapy and predict short- and long-term prognosis (mortality, reinfarction, bleeding risk).

Acute management

Acute management of NSTE-ACS aims to relieve ischaemia, prevent progression of infarction and reduce mortality. Initial assessment includes an ECG within 10 minutes, serial cardiac troponin concentrations, and risk stratification using the GRACE 2.0 or TIMI score. Early stabilization focuses on symptom relief with nitrates (±morphine), oxygen only if saturations are <90%, and prompt initiation of antiplatelet and anticoagulant therapy.

Pharmacotherapy

Anti-platelet therapy: dual antiplatelet therapy (DAPT) with aspirin and a $P2Y_{12}$ inhibitor is recommended for all patients unless contraindicated. Ticagrelor or prasugrel is preferred because of their superior efficacy, while clopidogrel is reserved for patients with an increased bleeding risk or intolerance. DAPT is typically continued for 12 months, with the duration individualized according to ischaemic and bleeding risk.[1]

In individuals undergoing PCI, DAPT reduces stent thrombosis and recurrent MI; in medically managed NSTE-ACS, it remains equally important for secondary prevention (Figure 4).

Anticoagulation: anticoagulation should be initiated early to reduce thrombotic complications and continued until revascularization or clinical stabilization. Unfractionated heparin is commonly used, particularly when early PCI is anticipated. Enoxaparin is an alternative. Fondaparinux is preferred in medically managed patients but requires additional unfractionated heparin during PCI. Dose adjustment is required in renal impairment (Figure 4).

Definitive treatment

Percutaneous coronary intervention plays a central therapeutic role in NSTE-ACS as part of an early invasive strategy aimed at restoring coronary blood flow, limiting myocardial damage and preventing recurrent ischaemic events. PCI is typically performed after diagnostic coronary angiography has identified a culprit lesion.

In individuals at high risk (e.g. elevated or rising troponin, dynamic ST segment or T wave changes, heart failure, haemodynamic compromise, high GRACE 2.0 score) early PCI within 24

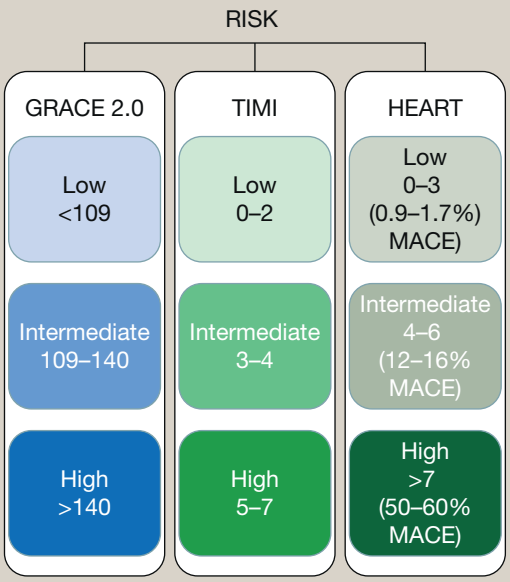
Non-atherosclerotic causes of NSTE-ACS

Aetiology	Pathophysiology
Coronary vasospasm	**Epicardial coronary vasospasm:** defined as intense vasoconstriction of an epicardial coronary artery resulting in compromised myocardial blood flow. Coronary vasospasm can occur in response to drugs, toxins or tumours (e.g. 5-fluorouracil, cocaine or phaeochromocytoma, respectively), from vascular smooth muscle hyperreactivity or spontaneously because of disorders in coronary vasomotor tone **Microvascular dysfunction:** often results from endothelial activation, increased oxidative stress and platelet and leucocyte adhesion, which reduce nitric oxide availability and promote vasoconstriction.
Coronary thromboembolism	First described by Pirizel et al. (See Further reading), and although uncommon, can cause MINOCA. Coronary thromboembolism in MINOCA has been reported in up to 2.9% of patients, with atrial fibrillation being the most common cause
Spontaneous coronary artery dissection	An increasingly recognized cause of MINOCA, accounting for 1.7–4% of all ACS. In female patients <50 years old, the prevalence can be up to 25%. It is associated with pregnancy, vascular Ehlers–Danlos syndrome and Marfan syndrome, and is the most common coronary artery manifestation of fibromuscular dysplasia
Imbalance of supply and demand	Can result from severe fixed atherosclerotic narrowing without acute thrombosis, or from supply and demand mismatch (type 2 MI), as in anaemia, hypotension or tachyarrhythmia

Table 1

Components of the GRACE 2.0, TIMI and HEART scores and their associated risk

	GRACE 2.0	TIMI	HEART
Purpose	Estimate hospital and 6-month mortality for patients with UA and NSTE-ACS	Predicts 14-day risk of MACE	Risk stratify chest pain in ED setting, predicts 30-day MACE
Variables			
Age	✓	✓ (>65)	✓
Heart rate	✓	✗	✘
Systolic BP	✓	✗	✘
HF (Killip class)	✓	✗	✘
Cardiac arrest	✓	✗	✘
ST segment deviation	✓	✓ (>0.5 mm)	✓
Elevated cardiac biomarkers	✓	✓	✓
Creatinine	✓	✗	✘
History (typical angina)	✗	✓ (>2 episodes)	✓
Known CAD	✗	✓	✓
CAD risk factors	✗	✓ (>3)	✓



BP, blood pressure; CAD, coronary artery disease; ED, emergency department; HF, heart failure; MACE, major adverse cardiovascular event; UA, unstable angina

Figure 3

hours is recommended; urgent PCI is required for those with refractory chest pain, cardiogenic shock or life-threatening arrhythmias. During PCI, drug-eluting stents are the predominant definitive treatment, with subsequent DAPT tailored to bleeding and ischaemic risk. In lower risk patients or those managed conservatively, PCI is reserved for recurrent symptoms, ischaemia on non-invasive testing or failure of medical therapy (Figure 5).

Drug-coated balloon (DCB) therapy is an emerging alternative to stent implantation for selected patients with NSTE-ACS, offering revascularization without leaving a permanent metal scaffold. In the setting of ACS, DCBs are primarily used after adequate lesion preparation (<30% residual stenosis achieved, no major dissection, TIMI 3 flow). Their potential advantages include avoiding stent-related complications such as late stent thrombosis, minimizing long-term DAPT and preserving vessel physiology.

In NSTE-ACS, DCB can be attractive when plaque morphology is suitable, angiographic results are optimal after preparation or prolonged DAPT is problematic. As trials continue, DCB-only strategies may expand but they currently require careful patient and lesion selection (Figure 5).

Considerations for coronary artery bypass grafting in NSTE-ACS: CABG is reserved for individuals with complex multivessel or left main disease unsuitable for PCI. CABG is often performed within 1–2 weeks after initial stabilization. In more urgent cases (e.g. continuing ischaemia, shock), it may be done earlier. Patients requiring CABG should be managed with antiplatelet therapy to reduce the risk of thrombotic events, but the timing of surgery is crucial because of the bleeding risk associated with DAPT. Generally, aspirin is continued alone before surgery.

Longer term management

Dual antiplatelet therapy

Patients managed medically or with PCI should remain on lifelong aspirin, with a $P2Y_{12}$ inhibitor for 12 months. The duration of the latter can be shortened to ≤6 months in individuals with a high bleeding risk.

Special circumstances – concurrent treatment with direct oral anticoagulants (DOACs): in individuals with atrial fibrillation, management requires balancing stroke prevention with coronary protection. Triple therapy (DOAC, aspirin and a $P2Y_{12}$ inhibitor) should be used for the shortest duration possible, typically up to 1 week. Therapy should then be de-escalated to dual therapy with a DOAC and clopidogrel for 6–12 months, depending on ischaemic risk (complex PCI, high thrombotic risk) versus bleeding risk (age, CKD, previous bleeding).

After the first year, most patients transition to DOAC alone for long-term management of atrial fibrillation. Aspirin is typically discontinued early unless the person has an extremely high ischaemic risk. Overall, therapy must be individualized using clinical judgement and validated bleeding/ischaemic risk tools.

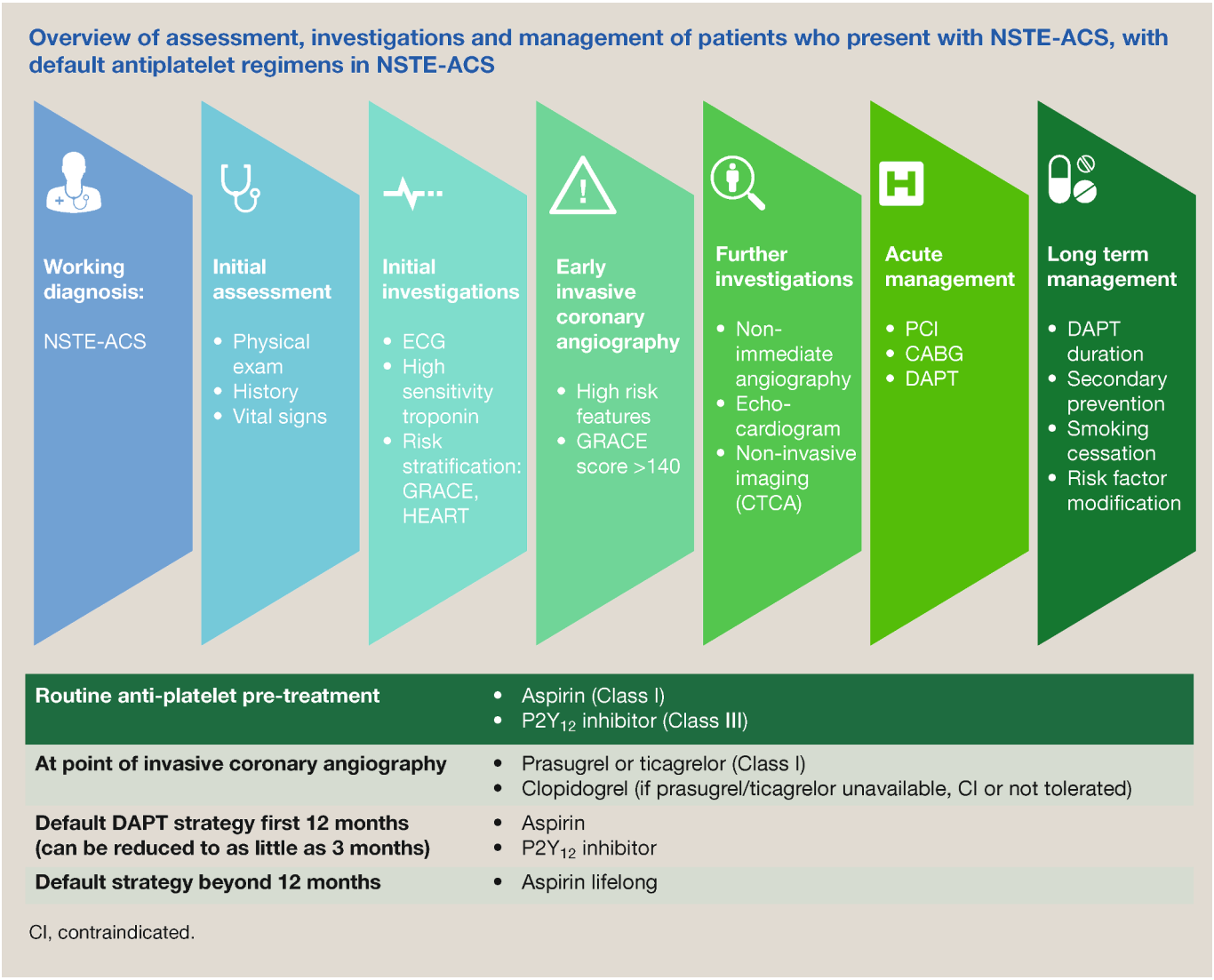


Figure 4

Special circumstances — pregnancy: NSTE-ACS in pregnancy is rare and often has non-atherosclerotic causes. It requires careful considerations because of the complexities of the altered physiology and the need to balance maternal and fetal risks.

Low-dose aspirin is generally considered safe in pregnancy. There are limited data for clopidogrel but it is thought to be safe for use in pregnancy. Ticagrelor and prasugrel are not recommended as there are limited safety data. PCI should not be delayed when clinically indicated.

Special circumstances — chronic kidney disease: CKD is linked to a higher risk of atherosclerosis and a high mortality rate after ACS. Most standard clinical trials for ACS exclude patients with significant kidney disease, and therefore their management must be tailored with careful consideration. Aspirin and clopidogrel are commonly used, with dose adjustment in severe renal impairment. Ticagrelor can be used cautiously, while prasugrel is generally avoided in moderate to severe CKD.

Secondary prevention

Secondary prevention aims to reduce recurrent cardiovascular events. A key component of secondary prevention is lipid modification. High-intensity statins (e.g. atorvastatin 40–80 mg, rosuvastatin 20–40 mg) should be initiated and continued indefinitely, with a low-density lipoprotein target of <1.8 mmol/litre (<1.4 mmol/litre in very high-risk patients). β-adrenoceptor blockers are recommended in individuals with left ventricular dysfunction or arrhythmias.

For patients with a reduced left ventricular ejection fraction (<40%), angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers or angiotensin receptor–neprilysin inhibitors should be prescribed. These improve survival by preventing ventricular remodelling and reducing the risk of heart failure.

Similarly, aldosterone antagonists (spironolactone, eplerenone) should be considered for individuals with heart failure or diabetes and low left ventricular ejection fraction, as they have been shown to reduce mortality and hospitalization rates.

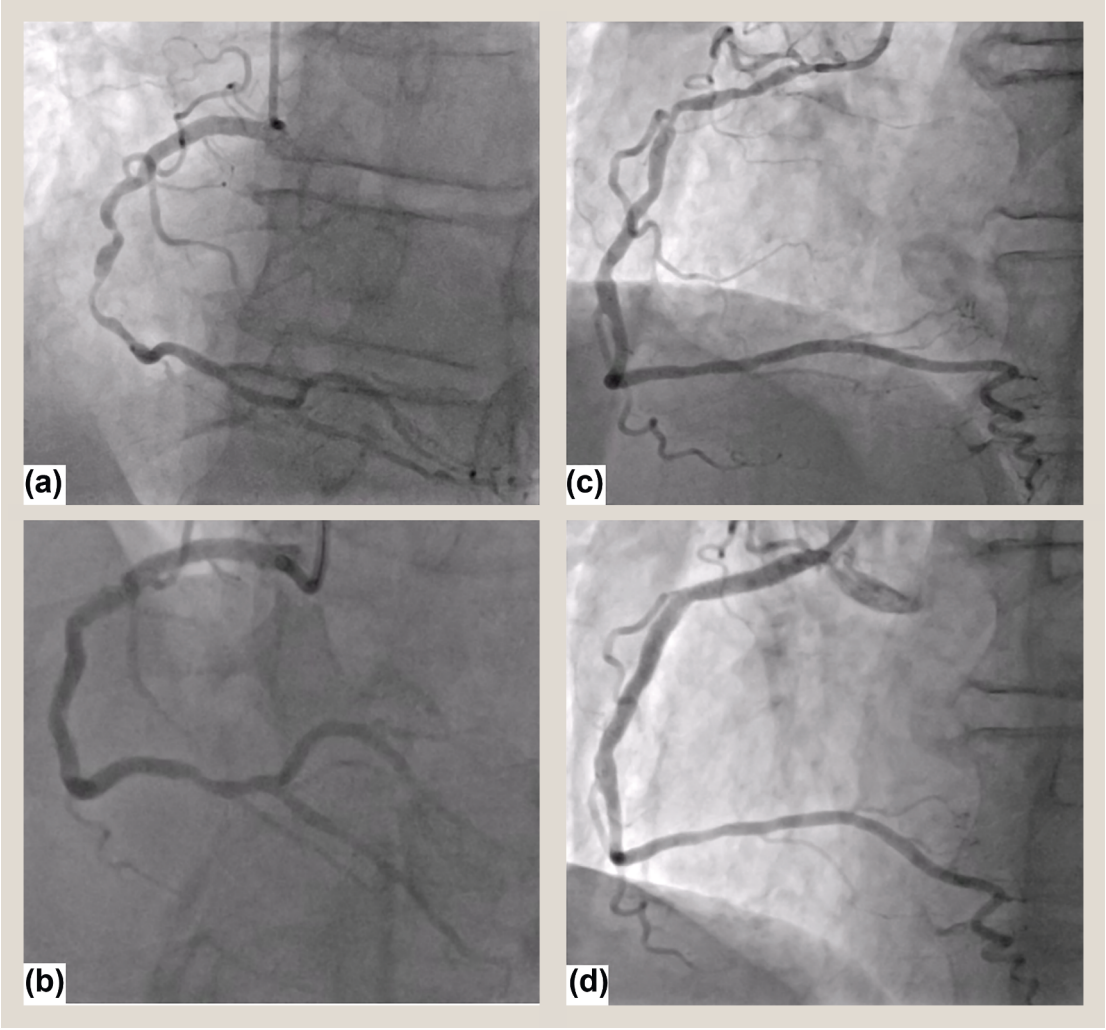


Figure 5 Coronary angiogram undertaken in two patients admitted with NSTE-ACS, demonstrating a severe lesion in the right coronary artery (**a**) before and (**b**) after treatment with a DCB. In comparison, a severe stenosis in the right coronary artery is demonstrated (**c**) before and (**d**) after treatment with a drug-eluting stent.

Sodium glucose co-transporter inhibitors should also be used for patients with reduced left ventricular function.

In addition to pharmacological treatment, lifestyle modification, including smoking cessation, weight control, blood pressure and glycaemic management, and participation in cardiac rehabilitation, is essential for long-term risk reduction. Participation in cardiac rehabilitation programmes can improve fitness, reduce anxiety and depression, and lower the risk of future cardiac events.

A comprehensive approach to secondary prevention incorporating pharmacological therapy, lifestyle changes and regular monitoring helps reduce the risk of recurrent cardiovascular events and improves long-term survival after NSTE-ACS.

Prognosis

Individuals with NSTE-ACS generally have lower short-term mortality than those with ST segment elevation MI, as complete coronary occlusion is less common. However, they often have more extensive coronary artery disease, advanced age and co-morbidities such as diabetes and renal dysfunction, resulting in a poorer long-term prognosis. Early mortality is approximately 3–5%, while 1-year mortality can reach 8–15%, largely because of recurrent ischaemia, heart failure and reinfarction. Adverse outcomes are strongly associated with elevated cardiac biomarkers, ST segment depression, haemodynamic instability and high GRACE 2.0 or TIMI scores.

Prognosis improves with early invasive management in high-risk patients and optimal medical therapy, including DAPT, statins, β-blockers and ACE inhibitors. Long-term outcomes depend on adherence to secondary prevention strategies and effective control of modifiable risk factors such as smoking, dyslipidaemia and hypertension. Despite advances in treatment, NSTE-ACS remains associated with a substantial risk of recurrent cardiovascular events, highlighting the importance of continuing follow-up and aggressive risk factor modification. ◆

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TEST YOURSELF

To test your knowledge based on the article you have just read, please complete the questions below. The answers can be found at the end of the issue or online here.

Question 1

A 62-year-old man presented to the emergency department with 45 minutes of substernal chest pressure radiating to the left arm. He received nitroglycerin and his chest pain was improving. He had a history of hypertension and hyperlipidaemia. His vital signs were stable.

Investigations

- ECG showed 1–2 mm horizontal ST depressions in leads V4–V6
- High-sensitivity troponin T was elevated above the 99th percentile upper reference limit and rose further on repeat testing 2 hours later
- Bedside echocardiogram demonstrated normal biventricular function with no regional wall motion abnormalities

Which of the following best confirms the diagnosis of non-ST elevation acute coronary syndrome in this patient?

A. Persistent chest pain relieved by nitroglycerin
B. ECG showing no ST segment elevation
C. Dynamic rise in troponin concentrations with ischaemic symptoms
D. Normal echocardiogram on admission
E. History of coronary artery disease

Question 2

A 58-year-old woman presented with 2 hours of exertional chest pressure. She had type 2 diabetes and hypertension. She was haemodynamically stable with no signs of heart failure. Her GRACE 2.0 score was 145 (<109; low, 109–140; intermediate, >140; high).

Investigations

- ECG showed 1 mm ST depressions in leads II, III and aVF
- High-sensitivity troponin was 237 ng/litre (0–13 ng/litre)

What is the most appropriate next step in management?

A. Discharge home with aspirin and arrange outpatient stress testing
B. Start aspirin and fibrinolytic therapy
C. Begin dual antiplatelet therapy and arrange invasive coronary angiography within 24 hours
D. Delay coronary angiography for 72–96 hours because she is haemodynamically stable
E. Manage medically with β-blockers only unless symptoms worsen

Question 3

A 72-year-old man underwent percutaneous coronary intervention (PCI) with drug-eluting stent placement after an episode of chest pressure, mild ST segment depression and raised high-sensitivity troponin concentrations. He had a history of hypertension and peripheral artery disease. He was taking aspirin. He had no history of stroke or major bleeding, and his renal function was normal.

What is the most appropriate $P2Y_{12}$ inhibitor to initiate after PCI?

A. Clopidogrel
B. Aspirin monotherapy
C. Ticagrelor
D. Cangrelor as long-term oral therapy
E. Dipyridamole