Heart Foundation

CSANZ
Cardiac Society of Australia and New Zealand

Australian clinical guideline for diagnosing and managing acute coronary syndromes 2025

Reader information

This version of the *National Heart Foundation of Australia & Cardiac Society of Australia and New Zealand: Australian clinical guideline for diagnosing and managing acute coronary syndromes 2025* presents the key information required for clinical practice. For detailed background, definitions and supporting evidence, please consult the Comprehensive Guideline. The full Comprehensive Guideline is available at https://www.heartfoundation.org.au/for-professionals/acs-guideline. References to the Comprehensive Guideline are included throughout this document.

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Acknowledgement of Country

We acknowledge the Traditional Owners and Custodians of country throughout Australia and their continuing connection to land, waters and community. We pay our respects to them and their cultures, and Elders past, present and future.

First Nations peoples is the term used throughout this guideline. However, the Heart Foundation recognises that the preferred term(s) when writing for, and about, First Nations peoples can differ between communities and individuals. No disrespect is intended to First Nations peoples who may identify with an alternative term.

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Updates and revisions

Medical knowledge is continually evolving, and guidelines may be updated as new information becomes available. Users are encouraged to consult the latest version of the guideline and to consider any new evidence that may have emerged.

Jurisdictional application

This guideline has been developed in accordance with Australian regulatory and clinical standards and may not be suitable for use in other jurisdictions without appropriate adaptation.

Disclosures

For a full list of disclosures/conflicts of interest, refer to Supplementary material A2.

Contents

Abbreviations

ACE	angiotensin-converting enzyme	**High-STEACS**	high-sensitivity troponin in the evaluation of patients with acute coronary syndrome
ACOMI	acute coronary occlusion myocardial infarction	**hs-cTn**	high-sensitivity cardiac troponin
ACS	acute coronary syndromes	**INR**	international normalised ratio
AMI	acute myocardial infarction	**IRA**	infarct-related artery
ARC-HBR	Academic Research Consortium high bleeding risk	**IRR**	incidence rate ratio
CABG	coronary artery bypass grafting	**IV**	intravenous
CAD	coronary artery disease	**IVI**	intravascular imaging
CDP	clinical decision pathway	**IVUS**	intravascular ultrasound
CI	confidence interval	**kg**	kilogram
CK-MB	creatine kinase MB-isoenzyme	**LBBB**	left bundle branch block
CTCA	computed tomography coronary angiography	**LDL-C**	low-density lipoprotein cholesterol
cTn	cardiac troponin	**LMWH**	low molecular weight heparin
cTnI	cardiac troponin I	**LV**	left ventricular
cTnT	cardiac troponin T	**LVEF**	left ventricular ejection fraction
DAPT	dual antiplatelet therapy	**LVH**	left ventricular hypertrophy
DOAC	direct oral anticoagulants	**MACE**	major adverse cardiovascular events
ECG	electrocardiogram	**mg**	milligram
ED	emergency department	**MI**	myocardial infarction
EDACS	Emergency Department Assessment of Chest Pain Score	**MINOCA**	myocardial infarction with non-obstructive coronary arteries
eGFR	estimated glomerular filtration rate	**mm**	millimetre
FFR	fractional flow reserve	**mmol/L**	millimoles per litre
GPI	glycoprotein IIb/IIIa inhibitor	**MVD**	multivessel disease
GRACE	Global Registry of Acute Coronary Events	**ng/L**	nanograms per litre
GRADE	Grading of Recommendations Assessment, Development and Evaluation	**NSTEACS**	non-ST-segment elevation acute coronary syndromes
HBR	high bleeding risk	**NSTEMI**	non-ST-segment elevation myocardial infarction
HEART	history, electrocardiogram, age, risk factors, troponin	**OAC**	oral anticoagulant
OR	odds ratio	**STE**	ST-segment elevation
p	probability	**STEACS**	ST-segment elevation acute coronary syndromes
PBS	Pharmaceutical Benefits Scheme	**STEMI**	ST-segment elevation myocardial infarction
PCI	percutaneous coronary intervention	**TIMI**	thrombolysis in myocardial infarction
PET	positron emission tomography	**UA**	unstable angina
POC	point-of-care	**UDMI**	Universal Definition of Myocardial Infarction
RCT	randomised controlled trial	**UFH**	unfractionated heparin
RR	risk ratio	**UK**	United Kingdom
SAPT	single antiplatelet therapy	**VA-ECMO**	venoarterial extracorporeal membrane oxygenation
SCAD	spontaneous coronary artery dissection	**µg**	microgram
SpO2	oxygen saturation	**µg/L**	microgram per litre

Introduction

This guideline is provided to assist clinicians in the diagnosis and management of people presenting with symptoms suggestive of acute coronary syndromes (ACS), or with confirmed ACS. ACS includes acute myocardial infarction (AMI) and unstable angina (UA), resulting from inadequate blood flow to heart muscle. ACS is a leading cause of morbidity and mortality and is a time-critical medical emergency.

The recommendations are based on contemporary evidence. They should complement, not replace, clinical judgement. Shared decision-making among clinicians, presenting individuals and their families is required and should be based on individual values, preferences and circumstances. Clear communication with individuals and those who support them is critical to these discussions.

This guideline was developed in consultation with a broad range of organisations, clinical experts and people with lived experience, representing different geographic regions, sex, genders, ethnicities, clinical settings and perspectives.

Purpose

This guideline replaces the *National Heart Foundation of Australia & Cardiac Society of Australia and New Zealand: Australian clinical guidelines for the management of acute coronary syndromes 2016.*

The guideline includes:

- recommendations for assessing and managing people with suspected or confirmed ACS
- a short summary and reference to the available evidence supporting the recommendations
- practical advice on how to apply the recommendations
- specific practice points for assessing and managing ACS in underserved populations.

Scope

This guideline addresses:

- assessment of adults (>18 years) with suspected ACS
- management of confirmed ACS
- recovery after ACS and secondary prevention of future vascular events.

The guideline primarily addresses the management of myocardial infarction (MI) caused by atherosclerotic plaque rupture, ulceration, fissure or erosion. Some recommendations may also apply to other MI types, such as MI due to oxygen supply/demand mismatch without acute coronary occlusion, particularly for acute treatment and post-hospital care (Comprehensive Guideline Figure 2). Specific guidance is included for MI due to non-atherosclerotic causes, such as spontaneous coronary artery dissection (SCAD).

Non-acute coronary syndrome presentations, non-cardiac chest pain and related cardiac conditions (e.g. heart failure, risk factors or comorbidities like cancer or diabetes) are outside the guideline's scope. Healthcare professionals should consult existing resources for comprehensive management of these conditions.

Terminology and definitions

Term	Definition
Acute coronary syndromes (ACS)	ACS encompass both acute myocardial infarction (AMI) and unstable angina (UA). ACS may also be classified as ST-segment elevation ACS (STEACS) and non-ST-segment elevation ACS (NSTEACS). This guideline adopts the term acute coronary occlusion myocardial infarction (ACOMI) which may present as ST-segment elevation myocardial infarction (STEMI) or STEMI equivalents. The term ACOMI is adopted to highlight STEMI equivalents, which are often under recognised or missed in emergency settings. See Comprehensive Guideline Figure 1 for classification of conditions associated with ACS.
Chest pain – cardiac	Chest pain due to an underlying cardiac aetiology. Includes classic chest discomfort based on quality, location, radiation, and provoking and relieving factors that make it more likely to be of cardiac ischaemic origin.
Chest pain – non-cardiac	Chest pain symptoms likely due to a non-cardiac cause.
Chest pain – possible cardiac	Chest pain symptoms that suggest a cardiac origin.
Clinical decision pathway	A structured framework used by healthcare professionals to guide the diagnosis, evaluation and management of cardiac conditions. It provides a systematic approach to clinical decision-making, ensuring that care is evidence-based, consistent and person-centred. These pathways outline step-by-step recommendations based on clinical guidelines and best practices, often incorporating decision points, diagnostic criteria and treatment options. Cardiac clinical decision pathways are designed to improve efficiency, reduce variability in care and enhance outcomes. They typically cover key aspects such as risk stratification, diagnostic testing, treatment initiation and follow-up care. For example, a pathway for acute coronary syndrome (ACS) may guide clinicians through initial risk assessment, diagnostic tests like troponin measurements or electrocardiograms (ECGs), and decisions on reperfusion strategies or medication. These pathways may also include decision aids to facilitate shared decision-making and ensure alignment with an individual's values and preferences.
Coronary artery disease (CAD)	CAD refers to the narrowing and/or blockage of the coronary arteries due to accumulation of plaque (atherosclerosis).
Coronary heart disease	Coronary heart disease refers to heart muscle damage that is caused by CAD, where there is reduced blood flow through the coronary arteries. Coronary heart disease is the major underlying cause of ACS.
Coronary microvascular dysfunction	Epicardial or microvascular endothelial or non-endothelial dysfunction that limits myocardial perfusion, most often detected as reduced coronary flow reserve.
Heart failure	Heart failure is a complex clinical syndrome resulting from any structural or functional impairment of ventricular filling or ejection of blood. The main manifestations of heart failure are dyspnoea and fatigue (which may limit exercise tolerance) and fluid retention. These may lead to pulmonary or splanchnic congestion and/or peripheral oedema. There is no single diagnostic test for heart failure; it is largely a clinical diagnosis based on a careful history and physical examination including 12-lead ECG, chest X-ray, transthoracic echocardiography and laboratory blood testing. When the diagnosis is unclear following initial clinical assessment and an echocardiogram cannot be arranged in a timely fashion, measurement of plasma natriuretic peptide levels is recommended.

Term	Definition
Myocardial infarction (MI)	This guideline uses the definition of MI that has been refined from the Fourth Universal Definition of Myocardial Infarction (UDMI) to more accurately capture the clinical syndromes associated with both occlusive and non-occlusive events. This guideline adopts the term ACOMI, which includes both atherosclerotic and non-atherosclerotic causes. MI is the irreversible necrosis of heart muscle. A common cause of infarction is deprivation in myocardial oxygen supply due to interruption of blood flow in at least one coronary artery caused by plaque rupture, erosion, fissure or coronary dissection. MI can also result from inflammatory, metabolic or toxic insults to the myocardium. Early and accurate detection of MI is important for initiating and maintaining appropriate therapy. In clinical trials, lack of a uniform MI definition can result in low concurrence between the initial clinical and later adjudicated assessments of MI, which will affect accuracy of primary end points and trial outcomes. Thus, uniform definitions are needed to ensure accurate reporting of MI events across clinical trials and registries.
MI – Type 1 as per Fourth UDMI	Type 1 MI is characterised by atherosclerotic plaque rupture, ulceration, fissure or erosion with resulting intraluminal thrombus in one or more coronary arteries leading to decreased myocardial blood flow and/or distal embolisation and subsequent myocardial necrosis. The person may have underlying CAD but non-obstructive coronary atherosclerosis or there may be no angiographic evidence of CAD.
MI – Type 2 as per Fourth UDMI	Type 2 MI is myocardial necrosis associated with an imbalance between myocardial oxygen supply and demand, and may be associated with hypotension, hypertension, tachy/bradyarrhythmias, anaemia, hypoxaemia, coronary artery spasm, spontaneous coronary artery dissection (SCAD), coronary embolism and coronary microvascular dysfunction.
MI – Type 3 as per Fourth UDMI	Type 3 MI is MI resulting in death when biomarkers are not available.
MI – Type 4 and 5 as per Fourth UDMI	Types 4 and 5 MI relate to percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG), respectively.

Term	Definition
Myocardial infarction with non-obstructive coronary arteries (MINOCA)	The diagnosis of MINOCA is made in people with acute myocardial infarction (AMI) that fulfils all following criteria: 1. AMI (modified from the Fourth UDMI criteria[1]): Detection of a rise or fall of cardiac troponin (cTn) with at least one value above the 99th percentile upper reference limit and corroborative clinical evidence of infarction based on at least one of the following: • symptoms of myocardial ischaemia • new ischaemic electrocardiographic changes • development of pathological Q waves • imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic cause • identification of a coronary thrombus by angiography or autopsy. 2. Non-obstructive coronary arteries on angiography, defined as the absence of obstructive disease on angiography (i.e. no coronary artery stenosis ≥50%) in any major epicardial vessel. (Note that additional review of the angiogram may be required to ensure the absence of obstructive disease). This includes people with normal coronary arteries (no angiographic stenosis), mild luminal irregularities (angiographic stenosis <30% stenoses), moderate coronary atherosclerotic lesions (stenoses >30% but <50%). 3. No specific alternate diagnosis for the clinical presentation. Alternate diagnoses include but are not limited to non-ischaemic causes such as sepsis, pulmonary embolism and myocarditis.[2]
Myocardial injury	Myocardial injury, acute versus chronic (or acute-on-chronic), is defined by the presence of an elevated cTn concentration above the 99th percentile of the upper reference limit. Myocardial injury is a frequently encountered clinical syndrome and is associated with an adverse prognosis. Myocardial injury is considered acute if there is a rise or fall of cTn concentrations over time, and chronic when cTn concentrations are persistently elevated. Clinicians must distinguish between one of the MI subtypes and non-ischaemic myocardial injury. Acute myocardial injury is related to the diagnosis of MI, particularly when accompanied by supportive evidence in the form of symptoms, electrocardiographic abnormalities, or imaging evidence of new regional wall motion abnormalities or new loss of viable myocardium. Non-ischaemic myocardial injury may arise secondary to cardiac or non-cardiac conditions.
Myocarditis	Myocarditis is an inflammatory disease of the myocardium caused by viral infections or a post-viral immune-mediated response. Clinical manifestations of myocarditis are varied and include chest pain that is often sharp and reflective of epicardial inflammation involving the pericardium. Myocardial dysfunction often causes fatigue and exercise intolerance. Predominance of heart failure distinguishes myocarditis from pericarditis; cTn is usually elevated.

Term	Definition
Non-ST-segment elevation acute coronary syndromes (NSTEACS)	NSTEACS encompasses non-ST-segment elevation myocardial infarction (NSTEMI) and unstable angina. NSTEMIs are characterised by the presence of both criteria: 1. Detection of a rise or fall of cardiac biomarker values (preferably cTn) with at least one value above the 99th percentile upper reference limit. Electrocardiographic changes or ischaemic symptoms may or may not be present. 2. Absence of electrocardiographic changes that are diagnostic of an ST-segment elevation myocardial infarction (STEMI) (see STEMI). Refer to definition of unstable angina.
Occlusion myocardial infarction (OMI)	OMI is acute coronary occlusion or near occlusion with insufficient collateral circulation, such that downstream myocardium will undergo imminent infarction without timely reperfusion. Occlusion MI may not always result in ECG findings of STEMI.
Older adults	Adults older than 75 years old.
Pericarditis	Pericarditis is inflammation of the pericardial layers characterised by chest pain, electrocardiographic changes and often pericardial effusion. It is often caused by an infectious or non-infectious process but can also be idiopathic. Pericarditis usually presents with sharp, pleuritic chest pain, which may be improved by sitting up or leaning forward, although in many instances such findings are not present. A pericardial friction rub may be audible. Widespread STE with PR depression is the electrocardiographic hallmark, although changes are non-specific and may be transient.
Pulmonary embolism	Intravascular migration of a venous thrombus to the pulmonary arterial circulation. It is diagnosed by a positive pulmonary angiogram, an unequivocally positive helical computed tomography (CT) scan, a high-probability ventilation-perfusion scan or autopsy.
Regional and remote	Based on the Australian Bureau of Statistics Australian Statistical Geography Standard (ASGS) Edition 3 Remoteness Structure that categorises areas based on relative access to services. Categories are 'major cities', 'inner regional', 'outer regional', 'remote' and 'very remote'.
Shared decision-making	Shared decision-making involves open communication between a person and their healthcare provider to deliver appropriate, person-centred care. Shared decision-making combines a person's values, goals and preferences with the best available evidence about benefits and potential risks of healthcare interventions.[3] A three-step approach to implement shared decision-making in clinical practice involves introducing choice, describing options (often with decision aids) and assisting people to explore their preferences.[4] Shared decision-making should be implemented across the care continuum for people with suspected or confirmed ACS, including risk assessment, choice of reperfusion strategy and discharge planning. Shared decision-making enables alignment between peoples' values and proposed treatment options.[5] Shared decision-making is well supported and encouraged in cardiovascular research and practice, although further study is needed on optimal implementation.[6–10]

Term	Definition
Spontaneous coronary artery dissection (SCAD)	Epicardial coronary artery dissection that is not associated with atherosclerosis or trauma and is not iatrogenic. Predominant mechanism of myocardial injury occurring due to SCAD is coronary artery obstruction caused by an intramural haematoma or intimal disruption rather than atherosclerotic plaque rupture or intraluminal thrombus.
ST-segment elevation myocardial infarction (STEMI)	STEMI is characterised by the presence of both criteria: 1. Electrocardiographic evidence of STEMI: new or presumed new ST-segment elevation at the J-point in two contiguous leads with the cut-off point: ≥1 mm in all leads other than leads V2–V3 where the following cut-off points apply: ≥2 mm in men ≥40 years; ≥2–5 mm in men <40 years; or ≥1–5 mm in women regardless of age. When the magnitudes of J-point elevation in leads V2 and V3 are registered from a prior ECG, new J-point elevation ≥1 mm (as compared with the earlier ECG) should be considered an ischaemic response. 2. Detection of a rise or fall of cardiac cTn with at least one value above the 99th percentile upper reference limit.
Unstable angina (UA)	Myocardial ischaemia at rest or on minimal exertion in the absence of acute cardiomyocyte injury/necrosis. UA is characterised by angina pectoris that occurs without stress or activity, or with decreasing stress or activity compared with stable angina and has been present for <2 weeks. ECG changes of ACOMI and elevated troponin values are not seen in UA.

Further information regarding definitions and terminology can be found in the Comprehensive Guideline.

Intended audience

This guideline is intended for all healthcare professionals involved in the care of people with ACS, including cardiologists, emergency physicians, general practitioners, nurses, nurse practitioners, First Nations health workers and practitioners, pharmacists and other allied healthcare professionals. Although the term 'general practitioner' is used throughout, the Heart Foundation recognises that in some communities, other primary healthcare professionals – such as primary care nurses, nurse practitioners, and First Nations health workers and practitioners – are the first point of contact with the health system.

Classifications of guidance

There are three classifications of guidance used throughout this guideline, in a hierarchy that reflects the strength of evidence and the context of their application: GRADE recommendations, consensus recommendations and practice points.

GRADE recommendations provide the most robust guidance, using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) methodology.[3] These recommendations balance benefits and harms, incorporate an individual's preferences and consider resource use, offering either strong or weak recommendations depending on the certainty of evidence and the intervention's impact. GRADE definitions can be found in Table 1 in the Comprehensive Guideline.

Consensus recommendations are used when the GRADE approach is not applicable, often due to indirect or limited evidence. These recommendations are informed by expert opinion, supported by available evidence, and consider values, preferences and resources. They provide critical guidance in areas where clinical decisions are necessary, even though the evidence base may not be direct or comprehensive.

Lastly, practice points offer actionable and practical advice to facilitate the implementation of recommendations. They detail the specifics of applying recommendations, such as who, what, how and when, and they often include supplementary information like medication dosing or tools to enhance implementation. While they do not stand alone, practice points are essential for ensuring that recommendations are effectively applied, particularly in settings with geographical or resource-related challenges.

Further information regarding the development of recommendations can be found in Supplementary material A.

What's new in this guideline?

New terminology and definitions

This guideline adopts the new term acute coronary occlusion myocardial infarction (ACOMI).

ACOMI includes atherosclerotic and non-atherosclerotic causes, referred to in the previous guideline as 'type 1 myocardial infarction' and 'type 2 myocardial infarction' respectively.

This change in terminology is to emphasise clinical conditions which are considered equivalents to ST-segment elevation myocardial infarction (STEMI), such as spontaneous coronary artery dissection (SCAD), coronary embolism and coronary vasospasm or microvascular dysfunction. These equivalents are often under recognised in emergency settings, as they are similar in terms of clinical presentation and investigation findings.

Assessment and diagnosis

New guidance on the assessment and diagnosis of people with suspected or confirmed acute coronary syndromes (ACS):

- Description of multiple ECG patterns of ACOMI, beyond the traditional ST-segment elevation criteria, which should prompt consideration of emergency reperfusion.
- New clinical decision pathways incorporating high-sensitivity cardiac troponin assays to enable more efficient risk assessment compared with traditional (contemporary/conventional) troponin-based algorithms.
- For people classed as intermediate risk, invasive cardiac testing is now an option to further stratify and assess risk beyond 30 days.

Hospital care and reperfusion

New guidance on the acute management of people with STEMI or non-ST-segment elevation acute coronary syndromes:

- Stronger emphasis on the optimal timing of primary percutaneous coronary intervention (PCI) in people with STEMI:
  - <60 minutes from first medical contact at PCI-capable centres
  - <90 minutes from first medical contact at non-PCI capable centres/emergency services.
- New evidence for use of intravascular imaging-guided PCI in people with non-ST-segment elevation acute coronary syndromes.
- New recommendations for managing ACS with cardiac arrest and/or cardiogenic shock, including considerations for use of haemodynamic support devices and left ventricular assist devices.
- New recommendations on the treatment of multivessel disease, including specific timing of PCI of non-infarct related arteries and considerations for invasive physiology assessment.
- New recommendations for the management of ACS due to SCAD, including considerations for selective revascularisation.

Recovery and secondary prevention

New recommendations and guidance on non-pharmacological and pharmacological secondary prevention measures:

- More detailed advice on post-discharge care, including medicines and adherence strategies, vaccinations and screening for mental health conditions.
- Treatment algorithms to enable more tailored prescribing of antiplatelet and anticoagulation therapies.
- A new recommended treatment target for low density lipoprotein cholesterol (LDL-C) of <1.4 mmol/L and a reduction of at least 50% from baseline.
- New recommendations on select medicines including beta blockers and PCSK9 inhibitors.

Considerations for priority populations

- New practice points address the unique needs of priority populations with suspected or confirmed ACS, including women, older adults, First Nations peoples and people living in regional and remote areas.

Preamble

Impact of ACS in Australia

Acute coronary syndromes (ACS) are a significant public health concern in Australia, with substantial impacts on individuals, healthcare systems and society. In 2021, there were an estimated 57,300 acute coronary events among Australians aged 25 and over, equating to approximately 157 events per day.[11] Hospitalisations related to ACS also remain high, with 92,400 admissions recorded in 2020–21, representing over half of all coronary heart disease hospitalisations that year.[11] Tragically, coronary heart disease, which includes ACS, was the underlying cause of 17,300 deaths in 2021, accounting for 10% of all deaths in Australia.[11]

The economic burden of ACS is considerable. In 2017–18, the financial cost of ACS events to Australian governments was estimated at $1.93 billion.[12] Disparities exist, with men experiencing rates of acute coronary events 2.3 times higher than women after adjusting for age.[11] Women, however, face unique challenges, including longer symptom duration, delays in receiving treatment and lower rates of secondary prevention medication use.[13–15] Furthermore, First Nations peoples experience coronary events at twice the rate of non-Indigenous Australians, highlighting ongoing inequities in healthcare access and outcomes.[11, 16]

The age-standardised rate of acute coronary events declined by 31% between 2011 and 2018, progress has slowed for younger populations.[17] Encouragingly, improvements in care have been observed, with significant reductions in in-hospital events and six-month readmissions for people with ACS between 2000 and 2007.[12] These trends underscore the need for continued investment in prevention, early detection and equitable access to high-quality care to further reduce the burden of ACS on Australians.

Specific recommendations to improve outcomes for these high-risk populations, including older people and/or those with frailty, are incorporated throughout the guideline wherever possible.

Shared decision-making

Shared decision-making ensures person-centred care by aligning individual values and preferences with evidence-based treatment options. It involves introducing choice, describing options with decision aids, and exploring preferences. This approach is vital across the care continuum for acute coronary syndrome and is well supported in cardiovascular practice, although optimal implementation requires further study.[3–10] Please refer to Terminology and definitions for the full description of shared decision-making.

Collaborative approaches to care for First Nations peoples

A holistic, collaborative approach is crucial to the delivery of culturally appropriate, best-practice care for First Nations peoples with suspected or confirmed ACS. Shared decision-making is central to this but also includes the way healthcare settings and services are designed and interact. A truly collaborative approach is one that ensures First Nations peoples with ACS feel culturally safe at every point of contact with the health system.

It is the responsibility of all healthcare professionals along the ACS care continuum to be culturally competent and to embed shared decision-making in their clinical practice. Cultural competence refers to a set of consistent values, behaviours and actions that enable effective delivery of healthcare across cultures.[18]

Aboriginal and Torres Strait Islander health practitioners, Aboriginal health workers and Aboriginal liaison officers, where available, should be involved in a First Nations person's care as early as possible. Aboriginal and Torres Strait Islander health practitioners and Aboriginal health workers (collectively referred to in this guideline as First Nations health practitioners) play a critical role in the ongoing management and follow-up of First Nations peoples with confirmed or suspected ACS. These practitioners work in tertiary, secondary and primary care settings, including Aboriginal Community Controlled Health Organisations. Particularly in remote communities, Aboriginal health workers are vital to the delivery of primary health care.

Aboriginal liaison officers support the First Nations person and their family by acting as an intermediary with healthcare professionals. They help to overcome cultural and communication barriers to care and provide support in navigating the hospital system. Crucially, Aboriginal liaison officers also help facilitate the transition to outpatient care, acting as the central liaison between the person's specialist team and general practitioner.

Specific practice points for First Nations peoples have been embedded throughout this guideline. These practice points recognise the different health outcomes for First Nations peoples that have resulted from dispossession, discrimination, disadvantage and disempowerment. Several resources are available on the Heart Foundation website to guide yarning and shared decision-making between First Nations peoples and their healthcare providers.

Challenges in regional and remote settings

Presentations in regional and remote settings pose unique challenges, including limited availability of staff and technical resources, as well as potentially prolonged transfer times.[19] Approximately one-quarter of individuals presenting with chest pain outside of metropolitan areas require transfer to at least one other hospital, which can delay median times to angiography and therefore diagnosis and lengthen overall hospital stays.[20] Geographic location also affects clinical outcomes, with greater mortality observed at 18 months post-event in people presenting to non-PCI-capable centres, most of which are in regional or remote areas.

Shared decision-making is particularly vital for people with suspected ACS living in regional or remote areas, especially when considering investigation and management options. Decisions should reflect the person's preferences regarding ongoing management, which may involve remaining in a resource-poor setting or being transferred away from their community.

Sections addressing these specific challenges are incorporated throughout the document, where relevant, to provide tailored guidance for healthcare providers delivering services in regional and remote settings.

1. Assessment and diagnosis

Assessment of people with suspected ACS

Acute chest pain is a relatively common emergency department (ED) presentation, yet only a minority of people will be diagnosed with ACS.[21, 22] Among those presenting with acute chest pain to the ED in whom ACS is suspected, <5% will have STEMI, 5–10% NSTEMI, 5–10% UA, 15–20% other cardiac conditions, and 50–60% non-cardiac conditions.[22–25] Most people will therefore require further follow-up to assess and diagnose their condition.[26]

Assessment for ACS includes:

- history and physical examination
- ECG
- troponin testing.

These are required to diagnose as well as inform risk assessment and help guide the location and timing of further investigations, management and follow-up. Rapid identification and diagnosis of ACS is crucial as treatments are often time sensitive and earlier intervention improves outcomes.

Assessing a person's relative risk for ACS is the key initial goal, rather than achieving a conclusive diagnosis of ACS, which may not always be possible at the time (Section Risk assessment and clinical decision pathways for suspected ACS).

Assessment in suspected ACS should:

1. identify people with acute coronary occlusion myocardial infarction (ACOMI) (STEMI and STEMI equivalents)
2. identify people with NSTEMI
3. identify people with UA at high risk for 30-day MACE
4. identify people with underlying CAD in whom ACS is not confirmed.

Assessment for people with suspected ACS within an ED setting is described below. Specific guidance for people presenting in regional/remote and primary care settings is given in Primary care and regional and remote presentations.

Initial assessment summary

In people presenting with symptoms suggestive of ACS, the following steps are recommended (see Practice points for setting considerations):

- Vital signs including blood pressure, heart rate, respiratory rate and peripheral oxygen saturation should be recorded.
- An ECG should be reviewed by a clinician experienced in ECG interpretation to examine for evidence of ACOMI within 10 minutes of presentation (Initial ECG assessment).
- If a diagnosis of ACS is considered likely, further investigations, including troponin testing, should be performed (Section Biomarkers and Section Risk assessment and clinical decision pathways for suspected ACS).
- Promptly identify people with ACOMI suitable for urgent reperfusion.
- People without symptoms of ACOMI but who have ECG evidence of cardiac ischaemia, and/or people who are otherwise stratified as high risk of index MI or 30-day MACE (Section Clinical decision pathways) should have continuous cardiac monitoring.
- In people who are symptomatic and/or haemodynamically compromised, ECGs should be performed at (a minimum of) 15-minute intervals until the symptoms have resolved.
    a. Additional ECGs should be performed if the person's symptoms reoccur, there are changes in character or a change in their clinical condition.
- If there is no evidence of ACOMI on ECG, a targeted history and physical examination should be performed and differential diagnoses considered, particularly time-critical emergencies, including aortic aneurysm, pulmonary embolism or pneumothorax.
    a. A chest X-ray may be useful in identifying some other causes, including pneumonia or pneumothorax, and to assess for cardiac size or evidence of cardiac failure. Acquiring a chest X-ray should not delay urgent reperfusion. This guideline does not apply to these other time-critical emergencies.[27, 28]

If the ECG is normal, and symptoms are clearly attributable to a non-cardiac cause, this guideline no longer applies. Clear communication is essential. Explaining that they do not have ACS may reduce a person's and their carers' anxiety (Section Discharge planning and advice).

The following sections describe the subsequent processes of assessment and diagnosis to be followed if ACS is suspected.

Table 2. Differential diagnoses of acute chest pain

Cardiac: ACS	AMI, unstable angina
Cardiac: Other	Stable angina, myopericarditis, tachyarrhythmia, hypertensive emergencies, severe aortic stenosis, Takotsubo cardiomyopathy, cardiac trauma
Pulmonary	Pulmonary embolism, pneumothorax (including tension), infection (pneumonia, bronchitis), pleuritis
Vascular	Aortic dissection, expanding aortic aneurysm, sickle cell crisis
Gastrointestinal	Oesophagitis, reflux, spasm, rupture, peptic ulcer disease, pancreatitis, cholecystitis and biliary disease
Other	Musculoskeletal disease (including costochondritis, trauma), anxiety disorder, infectious disease (including herpes zoster)

Abbreviations: ACS, acute coronary syndromes; AMI, acute myocardial infarction.

History of the presenting complaint

After assessing vital signs and ECG, obtain a focused medical history, including symptoms consistent with MI, onset and timing, associated symptoms and ACS risk factors. Use translator services and culturally competent healthcare workers as needed to address language, cultural or hearing barriers (Practice Points: First Nations peoples).[29]

Chest pain, angina equivalents and associated symptoms

Chest pain is the most common symptom of ACS, yet it is not always present. Chest pain due to myocardial ischaemia is often described as substernal discomfort or pressure, which may radiate to the neck, arms or jaw. The pain is often exacerbated by exertion and relieved following 15–20 minutes of rest.[30] Chest pain due to MI or UA generally occurs at rest. People may also refer to a discomfort, pressure or heaviness and deny pain. In this guideline, discomfort, pressure and heaviness are included under the umbrella term of chest pain.

Descriptions of myocardial ischaemic pain vary according to sex, ethnicity and culture. The description of the pain may help in determining whether the person's presentation is consistent with myocardial ischaemia (Figure 3).

A response or lack of response to treatment (such as glyceryl trinitrate, standard analgesia or antiacids) should not be used as a diagnostic criterion for ACS (Section Initial therapeutic management).[31]

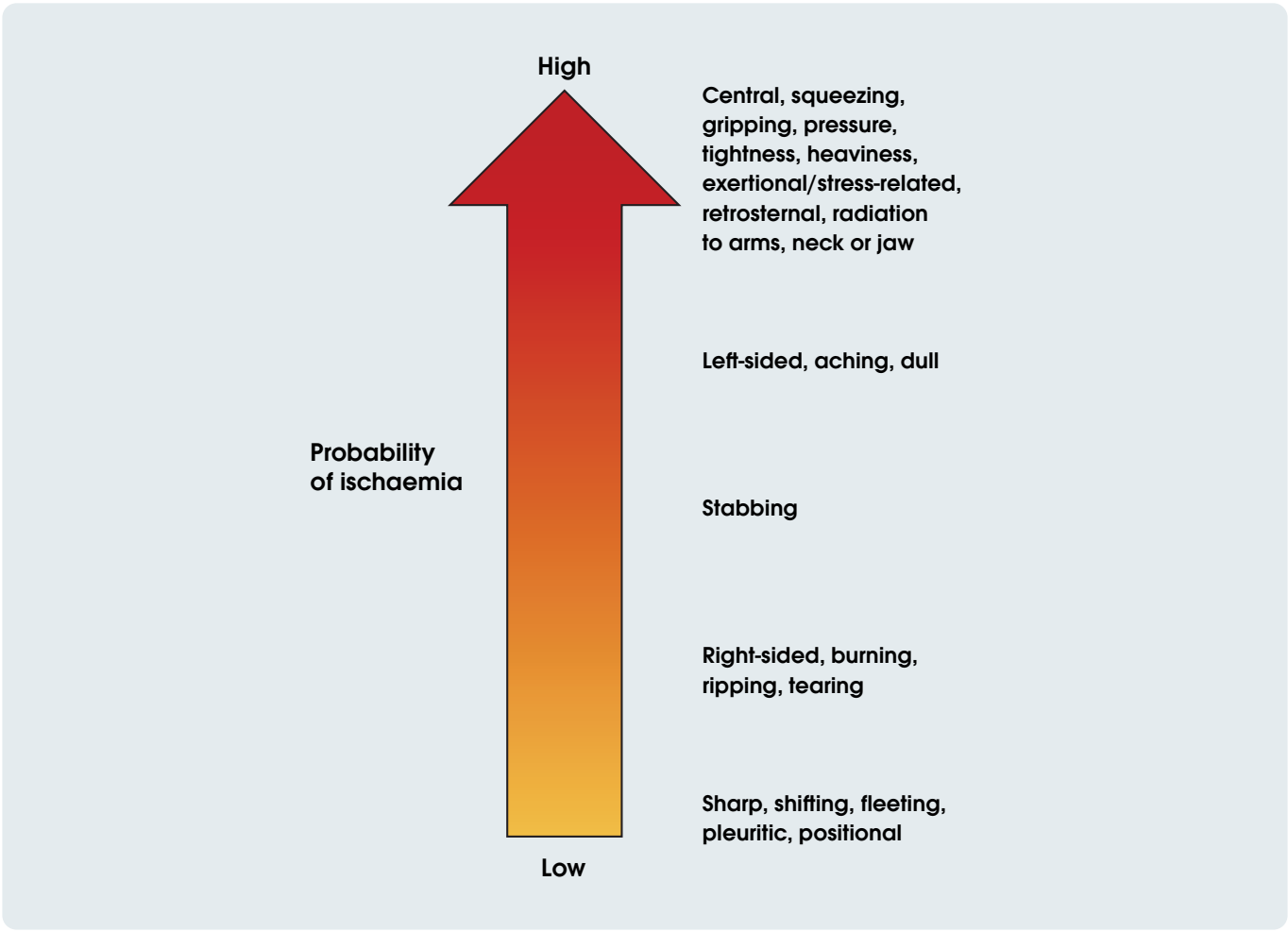


Figure 3 Probability of cardiac ischaemia based on commonly used descriptors of chest pain.

Shortness of breath, fatigue, nausea, diaphoresis or vomiting are relatively common associative symptoms of ACS. Women are more likely than men to report these symptoms (Practice Points: Women). Some people, particularly older adults and people with diabetes, may not describe any chest pain or discomfort and report only associated symptoms (sometimes referred to as angina or chest pain equivalents).

The terms typical and atypical have previously been used to describe cardiac ischaemic symptoms. However, given their wide variation, cardiac, possible cardiac or non-cardiac symptoms are now recommended terms (Practice Points: Women and Older adults).[26]

Factors associated with MI types

The risk factors for the different types of AMI are listed in Table 3. An absence of risk factors for CAD does not exclude ACS, which may present as either MI with acute coronary occlusion or MI due to oxygen supply/demand mismatch.

Table 3. Factors associated with spontaneous MI with coronary pathology and oxygen supply/demand imbalance.

Factors associated with atherosclerosis[32, 33]

- Older age (>75 years)
- Diabetes mellitus
- Hypertension
- Hypercholesterolaemia
- Obesity
- Smoking
- Socioeconomic disadvantage
- Family history of premature ASCVD (first-degree male relative aged <55 years; first-degree female relative aged <65 years)

Selected additional risk factors for atherosclerosis (for full details refer to www.cvdcheck.org.au):[33]

- Chronic inflammatory conditions, such as psoriasis, rheumatoid arthritis, lupus, or systemic sclerosis
- Familial hypercholesterolaemia confirmed by genetic testing
- Chronic kidney disease (eGFR 15–59 mL/min/1.73 m^2 with or without albuminuria; not treated with dialysis or kidney transplantation)
- Severe mental illness (defined as a current or recent mental health condition requiring specialist treatment, whether received or not, in the five years prior to risk assessment)
- Ethnicity (e.g. South Asian or First Nations ancestry)
- History of premature (before age 40 years) or early (before age 45 years) menopause
- History of pregnancy-associated conditions that increase later ASCVD risk, such as hypertensive disorders of pregnancy (e.g. pre-eclampsia) and gestational diabetes
- Polygenic risk score indicating higher risk of atherosclerosis
- COVID-19 (historical or current infection)

Factors associated with SCAD[34]

- Female sex
- Younger age (<50 years)
- Lack of cardiovascular risk factors
- Pregnancy or postpartum
- Fibromuscular dysplasia
- Inherited connective tissue disorders

Factors associated with coronary embolism[35]

- Aortic or mitral valve, left atrial appendage or left ventricle thrombus, vegetation or neoplasm
- Patent foramen ovale, atrial septal defect or pulmonary arteriovenous malformation with a venous source (e.g. deep vein thrombosis)
- Atrial fibrillation without adequate anticoagulation

Factors associated with coronary vasospasm[36–38]

- Smoking
- Older age (>75 years)
- Allergy
- Chemotherapy (5-fluorouracil)
- Some illicit drugs (cocaine, methamphetamine)

Factors associated with oxygen supply/demand imbalance (+/- atherosclerosis)[1]

- Severe anaemia
- Hypotension/shock
- Sustained tachycardia or tachyarrhythmia
- Sustained bradycardia or bradyarrhythmia
- Respiratory failure
- Sepsis
- Pulmonary embolism
- Critical illness

Factors associated with coronary microvascular dysfunction[39]

- Female sex (especially post-menopausal)
- Atherosclerotic disease
- Chronic inflammation (e.g. systemic lupus erythematosus, rheumatoid arthritis)
- Myocardial diseases
  - Hypertrophic cardiomyopathy
  - Dilated cardiomyopathy
  - Anderson-Fabry's disease
  - Amyloidosis
  - Myocarditis
  - Aortic stenosis

Note: Takotsubo cardiomyopathy is not classified as MI and is not discussed in this guideline.

Abbreviations: AIDS, acquired immunodeficiency syndrome; ASCVD, atherosclerotic cardiovascular disease; eGFR, estimated glomerular filtration rate; HIV, human immunodeficiency virus; MI, myocardial infarction; SCAD, spontaneous coronary artery dissection.

Practice points

Women

- Recognise chest pain as the most common symptom in women with ACS, occurring at similar rates to men. Be aware that associated symptoms in women may include jaw, neck, shoulder or back pain, fatigue, nausea, vomiting, dizziness, indigestion and shortness of breath.[40–42]
- Remain vigilant to the risk of misdiagnosis in women presenting with ACS.[43, 44] Women are more likely than men to be misdiagnosed with non-cardiac pain and are more likely to experience delays in receiving life-saving procedures in hospital, and healthcare professionals should consider potential sex bias when interpreting symptoms.[13, 45]
- Increase awareness of sex differences in ACS symptoms and presentation patterns. Clinician education and recognition of these differences may improve diagnosis and management of ACS in women.
- Consider SCAD as a potential cause of ACS, particularly in young to middle-aged women. Refer to (Section Treatment for SCAD) for management recommendations.[32, 34]

Older adults

- Recognise that older age (>75 years) is an independent risk factor for ACS and other conditions with similar presentations. Comorbidities are also more prevalent in this age group, which may complicate diagnosis and management.[46]
- Be aware that chest pain may not be the primary symptom of AMI in older adults. In this population, including those with STEMI and STEMI equivalents, angina equivalents (such as shortness of breath, fatigue, or dizziness) being commonly observed.[47–50]

First Nations peoples

- Recognise that First Nations peoples with ACS are typically younger, have higher rates of cardiac risk factors, experience lower intervention rates and face poorer outcomes compared to non-Indigenous Australians. These disparities highlight the importance of tailored interventions and equitable care.[16, 51, 52]
- Provide access to First Nations health practitioners, liaison officers and culturally appropriate interpreter services within hospitals. This facilitates accurate history-taking and improves the quality of care.[16, 51]
- Incorporate culture-specific attitudes and values into health promotion tools and offer culturally appropriate pastoral care. These steps can help bridge cultural gaps and enhance engagement with First Nations peoples.[16, 51]
- Prioritise education on cultural awareness, competency and safety for healthcare professionals. Such education has been shown to reduce unconscious bias and improve health outcomes for First Nations peoples.[16, 51]

Initial ECG assessment

Recommendations	Strength of recommendation	Certainty of evidence
In people presenting with chest pain or other symptoms suggestive of ACS, record an ECG for evidence of ACOMI within 10 minutes of first clinical contact.	Consensus	
In people with suspected ACS, record additional ECGs if there is diagnostic uncertainty or if symptoms persist, change or recur. For those with ongoing ischaemic symptoms and an inconclusive standard 12-lead ECG, record right-sided and/or posterior leads.	Consensus	
Continuous cardiac monitoring is recommended while assessment for ACOMI continues in people with ongoing ischaemic symptoms, haemodynamic compromise or have new ischaemic findings on ECG. Ensure a defibrillator is readily available.	Strong	Low

Evidence supporting the recommendations

The priority in screening for ACS is to identify ACOMI early to expedite reperfusion and improve outcomes. Urgent reperfusion can save viable myocardial tissue and reduce morbidity and mortality. ECGs should be performed within 10 minutes of clinical contact and be interpreted by experienced clinicians, with remote processes in place if needed. Continuous cardiac monitoring is recommended for people at high risk and should be regularly reviewed. For those with non-ischaemic ECGs, resolved symptoms and normal troponin levels, monitoring is not required. Repeated 12-lead ECGs should be done at intervals or if symptoms change.[53–55]

ECG findings of acute coronary occlusion myocardial infarction

ST-segment elevation (STE) is the key ECG criterion required to determine whether reperfusion is warranted (Comprehensive Guideline Figure 4A).[56] STE is not specific to ACOMI and may occur in other cardiac and non-cardiac disease states.[57] These include pericarditis, left ventricular hypertrophy (LVH), left ventricular aneurysm, left bundle branch block (LBBB), right ventricular pacing, Takotsubo or other cardiomyopathies and Brugada patterns. Non-cardiac STE conditions include normal variant STE (early repolarisation), pulmonary embolism, hyperkalaemia, hypothermia and raised intracranial pressure.

In the clinical context of myocardial ischaemia, STE should be assumed to represent ACOMI until proven otherwise.

In addition to STE criteria, there are other ECG patterns indicative of an ACOMI.[58–60] Recognising these improves accurate ECG detection rates for acute coronary occlusion and may prompt consideration for reperfusion (Comprehensive Guideline Figure 4).[61] Supplementary lead ECGs may be needed to interrogate areas of the heart such as the inferior, basal, posterior and right ventricular walls (Comprehensive Guideline Table 4). Additional ECG information and findings are included in the Comprehensive Guideline.

Detecting new STE with an abnormal baseline ECG

ECG evidence for ACOMI may be difficult to discern in people with LBBB, right ventricular pacing or LVH.

The validated Modified Sgarbossa criteria improves diagnosis of STE in people with LBBB or right ventricular pacing. The criteria can discern STE with a specificity of 99% and a sensitivity of 80% in these populations (Comprehensive Guideline Figure 4F).[62–64] The Modified Sgarbossa criteria has a straightforward threshold, STE exceeding >25% of the depth of the preceding S wave in any lead.

Currently, there are no validated methods to distinguish STE due to ACOMI, LVH or hypertrophic cardiomyopathy using an ECG alone. Clinical suspicion for ACOMI should be high in the presence of haemodynamic compromise and/or symptoms consistent with ACS.

In people with LVH, current and historical ECGs should be compared. If historical ECG data are unavailable, continuous cardiac monitoring, close clinical observation and repeated ECGs are required to monitor for development of acute coronary occlusion. Expert consultation should be sought for people with persisting ischaemic symptoms and equivocal ECG findings of ACOMI.

High-risk ECG findings

Acute coronary occlusion may not be evident on the initial ECG. Certain ECG patterns are associated with potential progression to ACOMI. They require prompt and continuous clinical ECG monitoring.

Wellens T waves: defined by characteristic T wave inversions in the precordial leads. Where symptoms have resolved, these inversions may indicate a reperfusion syndrome linked to severe stenosis of the left anterior descending artery, known as Wellens syndrome (Comprehensive Guideline Figure 5A).[65] In such cases, avoid provocative tests (for example, exercise stress testing) and consider invasive coronary angiography. If or when ischaemic symptoms recur, the ECG recorded during those symptoms will often appear pseudonormalised, with T waves becoming more upright.

Diffuse ST-segment depression across multiple leads with STE in aVR: may represent global ischaemia of various etiologies including a left main occlusion, triple vessel disease or oxygen supply/demand mismatch ischaemia seen in type 2 MI (Comprehensive Guideline Figure 5B).[66] People with persisting symptoms with no identifiable alternative causes of ischaemia or who do not respond to treatment of alternative causes (e.g. hypoxia, anaemia, hypotension) should be considered for coronary angiography.[67]

Hyperacute T waves: symmetrical, broad-based T waves disproportionately large to the preceding QRS complex can be the first ECG finding of an evolving MI, although its prognostic significance has been questioned (Comprehensive Guideline Figure 5C).[68, 69] These people should be subject to close clinical and continuous cardiac monitoring and serial 12-lead ECGs to examine for signs of ACOMI. An important differential diagnosis is hyperkalaemia (Comprehensive Guideline Figure 5C).

Other signs of myocardial ischaemia on ECG

Additional ECG findings in a person with suspected myocardial ischaemia which warrant continuous cardiac monitoring and consideration of treatment for NSTEACS include:

- **ST-segment depression: ≥0.5 mm at the J-point in ≥2 contiguous leads which is horizontal or downsloping:** (Comprehensive Guideline Figure 5D). The deeper and more widespread the depression, the more severe the ischaemia.[70, 71] ST-segment depression in contiguous leads should be first considered as reciprocal change of ACOMI and the ECG examined for corresponding STE, as ST-segment depression secondary to subendocardial ischaemia does not generally localise to a regional coronary territory (Comprehensive Guideline Figure 4, Figure 5 and Table 4).[72] Although ST-segment depression occurs in other conditions (e.g. LVH, hypokalaemia, digoxin use), a systematic review found it to be highly specific (97.2–99.3%) but poorly sensitive (16.6–20.0%) for ischaemia.[73]
- **T wave abnormalities:** including dynamic inversion or flattening (Comprehensive Guideline Figure 5E). New T wave inversion compared to a previous ECG or dynamic T wave changes during serial ECGs may represent ischaemia. Specificity of T wave inversion for ischaemia is higher in the context of other signs of ischaemia on the ECG.[74]

Computer-assisted ECG interpretation

There is currently no international standardised system for computer-based ECG interpretation. Manufacturers of ECG machines use distinct algorithms, leading to variability in sensitivity and specificity for diagnosing cardiac conditions. Common computer-assisted interpretation errors in diagnosing ACS and ACOMI include misattributing Q waves associated with LVH, LBBB and/or dilated or hypertrophic cardiomyopathy to ACOMI. Additionally, errors often fail to distinguish between STE caused by early repolarisation, pericarditis, or LBBB and ACOMI.[75] Accurate ECG interpretation by a clinician, with consideration of the clinical context, remains essential.

Continuous ECG monitoring

Continuous ECG monitoring is not required in people with no ongoing symptoms, normal or non-ischaemic ECG changes and initial normal troponin values. In people with suspected ACS, ongoing ECG monitoring is recommended for those at high-risk (see Table 5).

Table 5. High-risk clinical features for people with suspected ACS requiring ongoing ECG monitoring

Haemodynamic instability or cardiogenic shock	Recurrent or ongoing chest pain refractory to medical treatment	Cardiac arrest	Recurrent dynamic ST-T wave changes or other changes consistent with myocardial ischaemia or infarction on ECG
Arrhythmias (e.g. sustained ventricular tachycardia, high degree atrioventricular block)	Mechanical complications of MI (e.g. new systolic murmur)	Acute heart failure	

Abbreviations: ACS, acute coronary syndromes; ECG, electrocardiogram; MI, myocardial infarction.

Practice points

- Do not rely on an initial normal ECG to exclude ACS. If myocardial ischaemia is strongly suspected, record and interpret serial ECGs.
- Do not delay treatment decisions/treatment in people with symptoms of ischaemia and clear evidence of ACOMI on ECG while waiting for troponin test results.
- Do ensure consistent lead placement when performing serial ECGs to prevent artefactual errors in interpretation.

Future direction

Artificial intelligence (AI) and machine learning has been applied to ECG and clinical data with the aim of delivering a more accurate and timely assessment of ACS and ACOMI.[76] Its utility remains in research at this time.

Biomarkers

Recommendations	Strength of recommendation	Certainty of evidence
In people with suspected ACS, evaluation with high-sensitivity cardiac troponin (hs-cTn) assays is recommended.	Strong	High
Elevated hs-cTn values should be defined using sex-specific >99th percentiles.	Consensus	
Apply the assay-specific troponin values relevant to the cTn assay being used.	Consensus	
When evaluating changes (deltas) in troponin values, serial results from a single assay must be used.	Consensus	

Evidence supporting the recommendations

High-sensitivity cardiac troponin (hs-cTnI or hs-cTnT) is the preferred biomarker for diagnosing ACS due to its precision, early detection of myocardial injury and improved accuracy for MI. These assays enable faster decision-making, reduce unnecessary admissions and account for sex differences (see Section Risk assessment and clinical decision pathways for suspected ACS). If unavailable, contemporary troponin assays can be used with longer testing intervals and clinical risk assessment.[1, 77–89]

Analytic properties of cardiac troponin assays

The cut-off or threshold indicative of myocardial injury is a cTn value above the assay-specific 99th percentile derived from a healthy population.[1] Contemporary assays in Australia use µg/L, while high-sensitivity assays use ng/L, reflecting their greater sensitivity. Other performance metrics, such as the limit of detection and limit of blank, are also critical for evaluating assay reliability (see Table 6, Figure 6).

Women have lower circulating cTn concentrations, resulting in sex-specific 99th percentile values.[90] Using older, non-sex-specific cut-off values can lead to underdiagnosis of myocardial injury and MI in women.[91]

Table 6. Contemporary vs high-sensitivity cTn assay features.

Characteristic	Contemporary troponin assays*	High-sensitivity troponin assays
Precision	Variable	≤10% CV at 99th percentile
Detection	~20–50% of healthy reference population	≥50% of healthy reference population
Units	Micrograms per litre (µg/L)	Nanogram per litre (ng/L)
Sex-specific 99th percentiles	No. Overall 99th percentile values only	Yes. Female and male 99th percentiles
Timing of serial testing for MI using 99th percentile	0 and 6–8 hours	0 and 3 hours
Single low-risk troponin values for MI*	No	Yes
Ability to use in rapid, early assessment strategies	No	Yes
Platform	POC and laboratory-based	POC and laboratory-based

*Using a contemporary assay, if a person presents symptom-free for >6–8 hours, only one test needed. If ≤99th percentile at 6–8 hours, no second test is required. If >99th, a second test is needed. Abbreviations: cTn, cardiac troponin; CV, coefficient of variation; MI, myocardial infarction; POC, point-of-care.

In transgender individuals, sex hormone use may affect myocardial mass and hs-cTn reference ranges, potentially differing from those based on sex assigned at birth.[92] To ensure safety, the lower female-specific cut-off should be applied, although further research is needed to establish standards for transgender populations.[92]

While cTn 99th percentiles tend to increase in people over 60 years, age-adjusted cut-offs have not been adopted in clinical practice.[90, 93–95]

Point-of-care troponin assays

Contemporary POC troponin assays require serial measurements over 6–8 hours in people with suspected ACS. POC troponin assays may lead to more timely management of people with suspected ACS, with comparable safety to laboratory-based assays (Comprehensive Guideline Table 7).[87–89, 97]

Early data support rapid assessment using POC hs-cTn assays.[87–89, 98] Knowledge of POC hs-cTn assays is rapidly evolving. Their use in clinical decision pathways in EDs, outpatient clinics and primary care may become more common given the clinical safety and cost efficacy of such assays.[97]

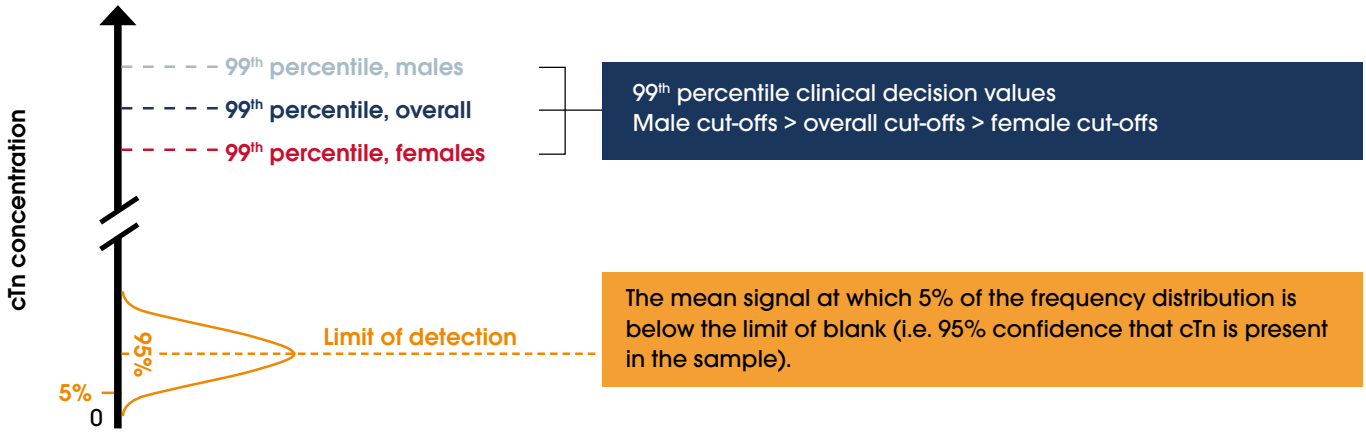


Figure 6 Various analytic definitions for troponin assays. Adapted with permission from Januzzi et al.[96]

Abbreviations: cTn, cardiac troponin.

Clinical interpretation of troponin values

cTn results must be interpreted alongside the clinical context and ECG findings (Figure 7).[26] Serial measurements are required to track whether cTn elevation is stable or changing. Stable elevation occurs with chronic myocardial injury as well as in the plateau phase of troponin release in MI (such as when presentation was delayed). People with changing values (increasing or decreasing) warrant evaluation for evidence of myocardial ischaemia. Acute myocardial injury due to other causes (e.g. acute heart failure, pulmonary embolism) needs to be considered. Differentiation between MI subtypes and other myocardial injury requires careful evaluation (Figure 7).[99]

The introduction of hs-cTn assays has led to a decrease in the proportion of people with UA, defined with cTn values ≤99th percentile, and many who would previously be classified as UA are now found to have MI.[100]

Time from onset of coronary occlusion vs symptom onset

In the setting of ACOMI, there may be a delay in elevation of cTn levels. This delay has shortened with more sensitive tests, including hs-cTn assays, able to detect elevations earlier (Figure 8). Repeat troponin testing is required for people with ongoing or recurrent symptoms or where there is a high suspicion of ACS.

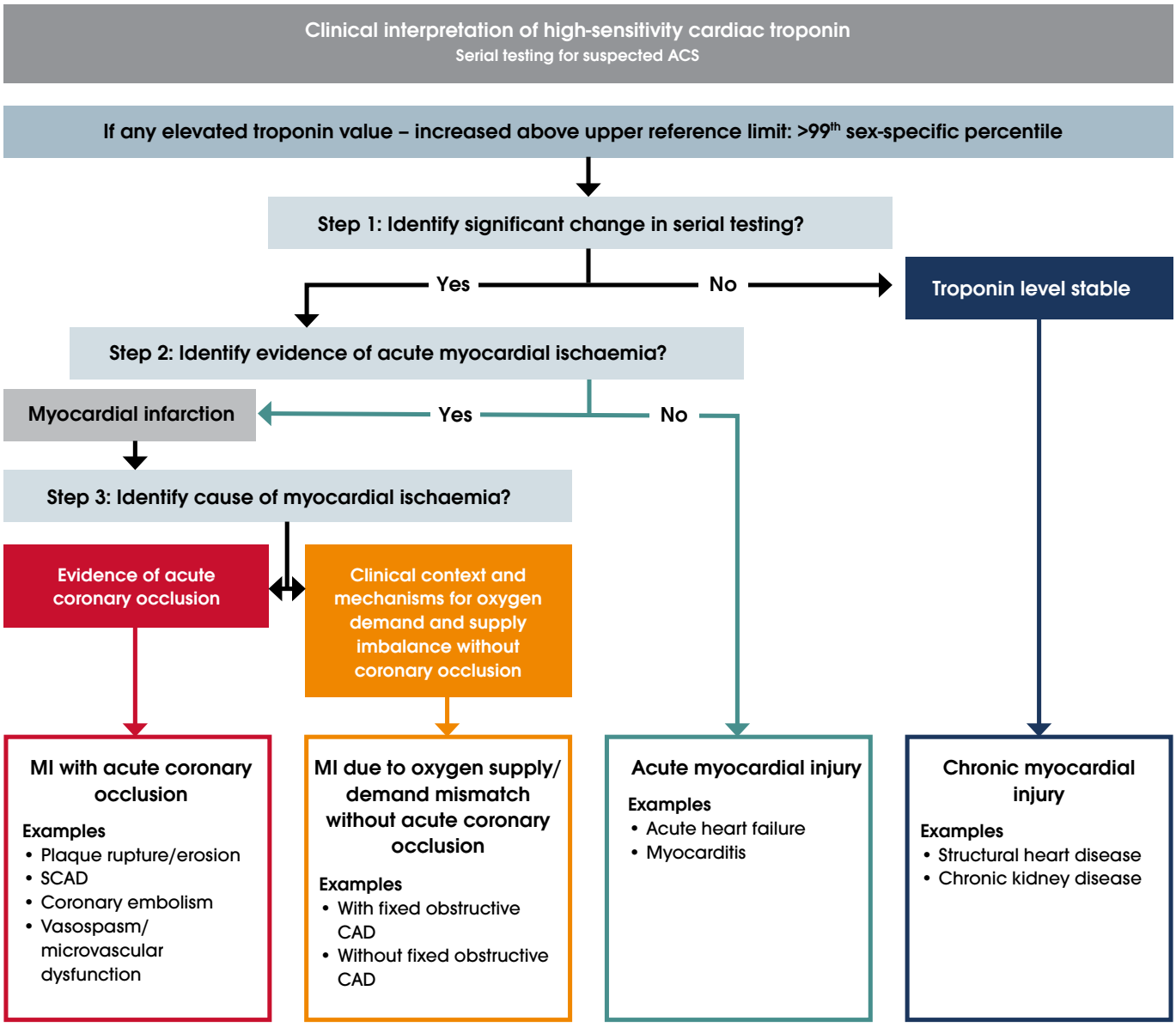


Figure 7 Clinical interpretation of high-sensitivity cardiac troponin (hs-cTn) results. Adapted with permission from the *Accelerated Chest Pain Risk Evaluation (ACRE) Project*, Clinical Excellence Queensland, Queensland Health.

For guidance on identifying evidence for acute myocardial ischaemia, refer to High-risk ECG findings and Other signs of myocardial ischaemia on ECG.

Abbreviations: ACS, acute coronary syndromes; CAD, coronary artery disease; MI, myocardial infarction; SCAD, spontaneous coronary artery dissection.

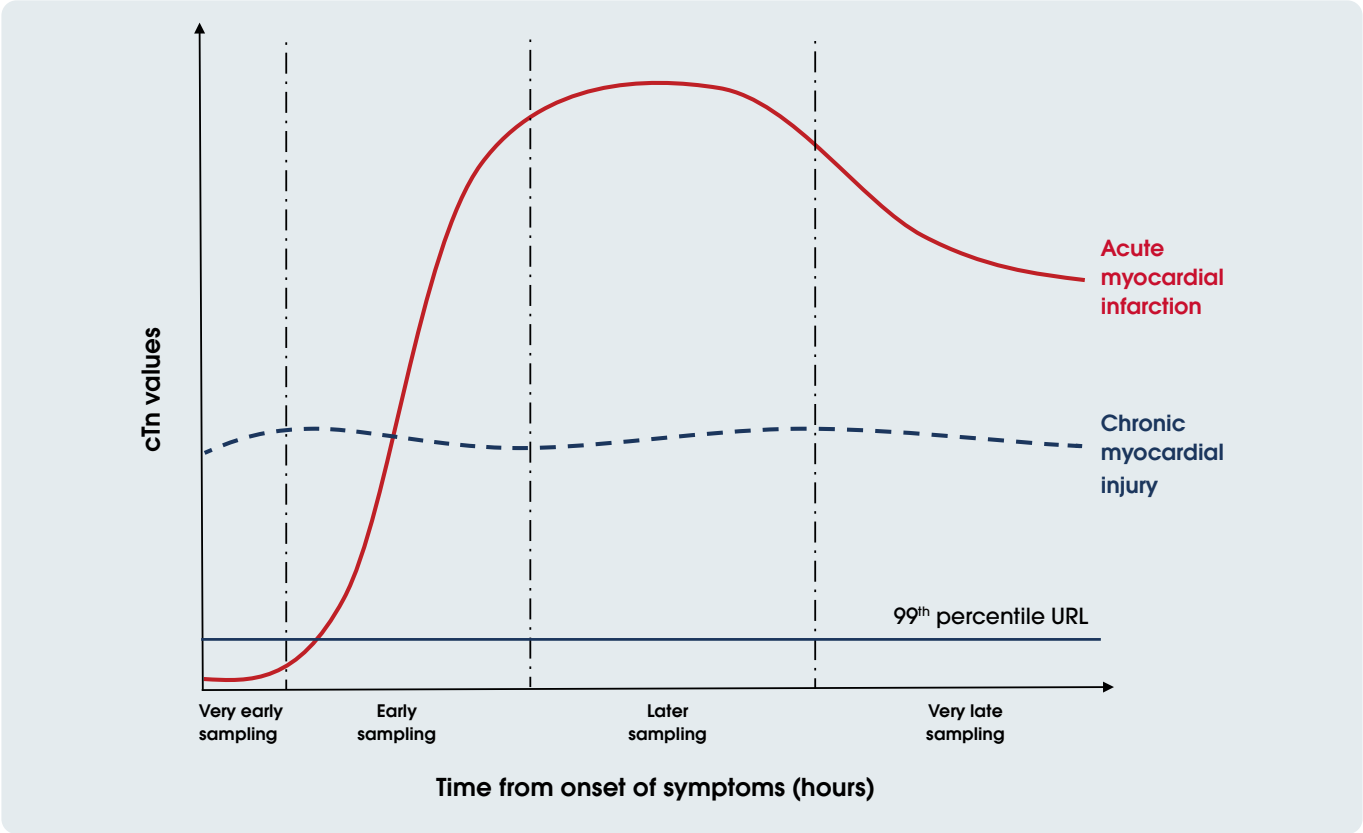


Figure 8 Early troponin kinetics in people with acute myocardial infarction.

Abbreviations: cTn, cardiac troponin; URL, upper reference limit.

Comparing results from different troponin assays

cTn assays developed by various diagnostic companies use different antibody combinations, resulting in different numerical results for the same amount of circulating troponin.[101] Results of one assay cannot be interpreted using the reference range of a different assay. Serial testing of cTn concentrations can only be interpreted when measured using the same assay.

Differences between troponin T and I assays

High-sensitivity cardiac troponin I (hs-cTnI) and high-sensitivity cardiac troponin T (hs-cTnT) have comparable accuracy for the early detection and diagnoses of MI.[102]

Troponin T is more likely to be elevated among people with poor renal function (see Renal disease) and chronic muscular diseases (e.g. chronic myopathy, myositis). This is possibly due to re-expression of cTnT in the diseased muscle or due to cross reactivity of the cTnT assay with skeletal muscle troponin T.[103, 104]

Non-MI causes of troponin elevation

Numerous ischaemic, non-coronary cardiac and non-cardiac causes of myocardial injury can result in elevated cTn concentrations (Figure 9).[1, 79, 105, 106] Life-threatening conditions including aortic dissection and pulmonary embolism may result in elevated cTn values. Cardiac troponin elevation indicates myocardial injury but is not specific to the underlying pathophysiology.[1]

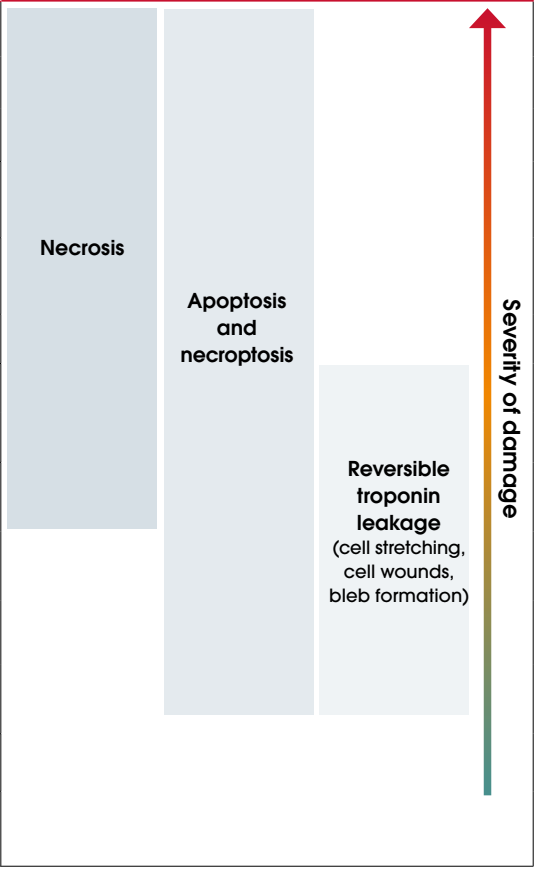
Clinical manifestation	Possible causes of elevation	Possible mechanism
AMI	Prolonged ischaemia	
Acute HF		
Pulmonary embolism		
Chest trauma or surgery	Mechanical cell destruction, local inflammation	
Stroke or brain trauma	Catecholamine-derived myocyte overload or ischaemia due to type 2 MI	
Cardiotoxicity	Cardiotoxic agents (drugs, CO, poisons)	
Myocarditis, endocarditis	Inflammation	
Sepsis		
Atrial fibrillation	Brief ischaemia Muscle overload	
Chronic HF		
Stable CAD		
Physical exercise		
Renal failure	Impaired clearance	
Skeletal muscle disorders	Expression of cTnT in regenerative skeletal muscles	

Figure 9 Conditions associated with troponin elevation. Adapted from Katrukha et al.[69]

Abbreviations: AMI, acute myocardial infarction; CAD, coronary artery disease; CO, carbon monoxide; cTnT, cardiac troponin T; HF, heart failure; MI, myocardial infarction.

Renal disease

Chronically elevated cTn concentrations are often reported with decreased renal function, more so cTnT than cTnI. MI diagnosis in people undergoing haemodialysis requires serial cTn measurements, rather than management according to an elevated baseline value.[107]

False positive and false negative cardiac troponin results

False positive or negative cTn results are rare but possible. False positives may occur due to antibody interference, such as macrotroponins – high molecular weight complexes of cTn fragments and immunoglobulins (cTn autoantibodies) – which delay troponin clearance and cause artificially elevated readings. Heterophilic antibodies cause another type of interference, as these can bind to test antibodies and yield a positive result without actual cTn elevation.

While the exact cause of heterophilic antibodies is unclear, they are sometimes associated with conditions like rheumatoid arthritis or viral infections, including Epstein–Barr virus and cytomegalovirus.[108] Conversely, severe haemolysis or plasma substances like biotin can lead to false negatives. If troponin levels do not align with the clinical presentation, consulting the hospital laboratory is essential to rule out these rare false positive cTn results.[109, 110]

Other biomarkers

Additional biomarkers exist but are not used to diagnose MI. There is no role for creatine kinase MB-isoenzyme (CK-MB) to identify reinfarction in people with AMI.[111]

Risk assessment and clinical decision pathways for suspected ACS

Recommendations	Strength of recommendation	Certainty of evidence
People with symptoms and ECG changes consistent with ACOMI require urgent reperfusion. Do not use further steps in a clinical decision pathway.	Strong	Very low
People presenting with acute chest pain or other symptoms suggestive of ACS without definite ACOMI should receive care guided by an evidence-based clinical decision pathway that includes assay-specific troponin results to categorise people as high, intermediate or low risk.	Consensus	
A high-sensitivity troponin-based clinical decision pathway is recommended, using the 0/1-hour or 0/2-hour strategy, or the *high-sensitivity troponin in the evaluation of patients with acute coronary syndrome* (High-STEACS) algorithm.	Consensus	
When contemporary troponin assays are used, a clinical decision pathway incorporating formal clinical score-based risk stratification vis recommended.	Consensus	

Evidence supporting the recommendations

Clinical decision pathways improve care and efficiency in suspected ACS by identifying MI and those at high-risk of MACE within 30 days (e.g. those requiring further investigation) while reducing unnecessary tests and admissions for low-risk individuals. Structured risk assessments incorporating clinical data, troponin and ECG findings achieve missed MI or 30-day MACE rates of <1%.[22, 112] People with suspected ACS or ECGs suggestive of ischaemia, or high-risk features, should undergo inpatient evaluation.[22, 113–115]

Clinical decision pathways for people without ACOMI or ischaemic ECG findings include those based on hs-cTn results alone or clinical risk scores like the *Emergency Department Assessment of Chest Pain Score* (EDACS) and *history, ECG, age, risk factors and troponin* (HEART).[116]

Clinical decision pathways

Risk stratification

A three-tiered stratification system groups people into high, intermediate or low risk of MACE, including MI (Figure 10). This system uses cTn levels, clinical history, physical examination and ECG findings (normal, non-ischaemic or unchanged from previously).

- **High risk:** Risk of a 30-day event, most commonly MI, exceeds 50–70%.[117] Admission and further evaluation are required. Not all high-risk individuals have MI, so clear communication about risk is essential. Refer to Section 2 Hospital care and reperfusion for further information on the evaluation and management of high-risk people.
- **Intermediate risk:** These people have a 30-day MACE risk of 2–22% using hs-cTn-based decision pathways (0/1 or 0/2-hour strategies) and require further evaluation.[118-120] Serial cTn values of ≤99th percentile allow outpatient testing, as the 30-day MACE rate is <2% (Section Further diagnostic testing for people with suspected ACS).[22] Elevated (>99th percentile) cTn values require evaluation in an inpatient setting. Elevated but stable cTn values consistent with chronic myocardial injury increases long-term cardiac risk without MI, and therefore is beyond this guideline's scope (Biomarkers).[1, 121]
- **Low risk:** The 30-day MACE risk is <1% using hs-cTn-based clinical decision pathway or clinical risk score (Supplementary material B2). hs-cTn strategies identify more low-risk individuals than contemporary cTn pathways. In low-risk people defined by a hs-cTn strategy, further testing to exclude AMI is not required.[26, 122, 123]

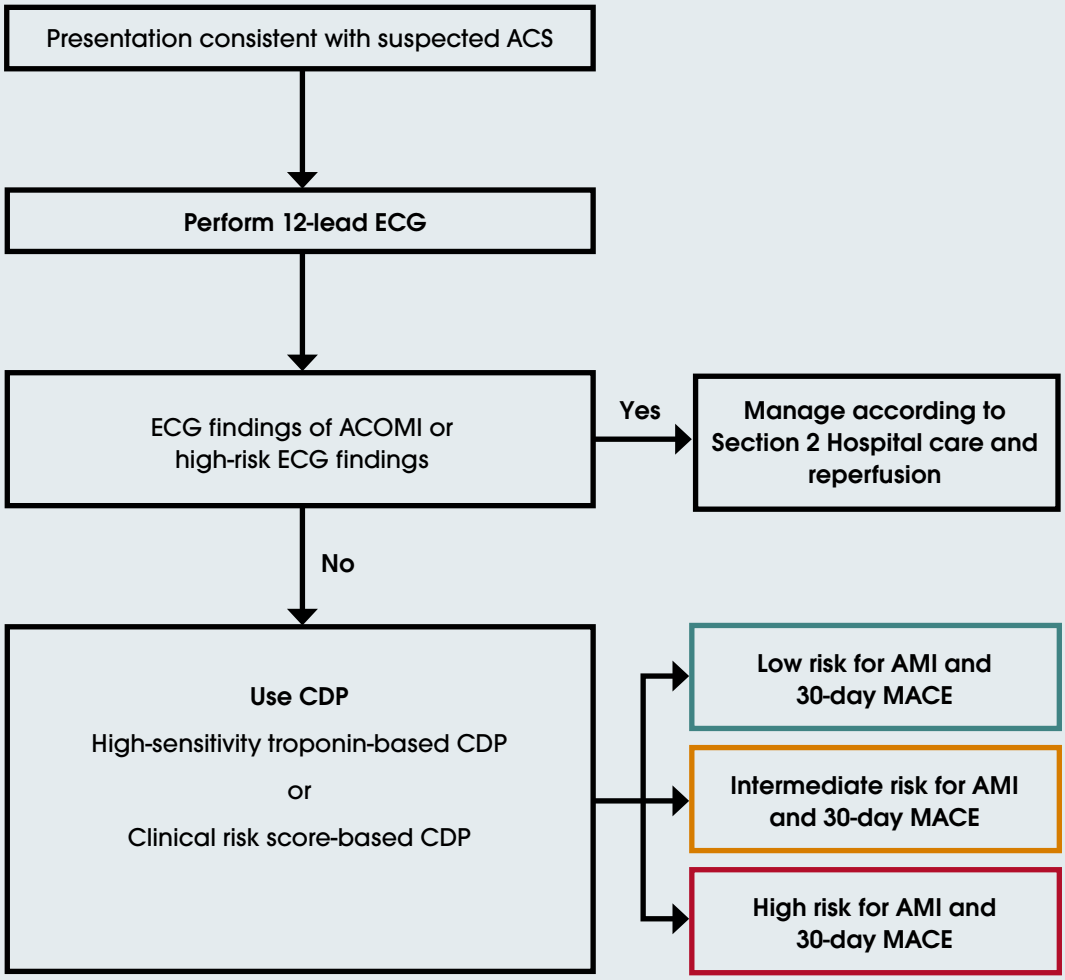


Figure 10 Assessment process for people with suspected ACS.

For further information, refer to Initial ECG assessment, High-sensitivity troponin-based clinical decision pathways and Clinical score-based clinical decision pathways in the guideline.

Abbreviations: ACOMI, acute coronary occlusion myocardial infarction; ACS, acute coronary syndromes; AMI, acute myocardial infarction; CDP, clinical decision pathway; ECG, electrocardiogram; MACE, major adverse cardiovascular events.

Risk stratification for people with suspected ACS: identifying MI and UA

For people without findings consistent with ACOMI on the initial ECG, further assessment aims to identify NSTEMI and UA through evaluation of clinical features, additional ECGs and troponin testing. NSTEMI is associated with elevated cTn values.

People with ongoing or recurrent ischaemic symptoms, or new ECG findings suggestive of ischaemia during initial or repeat testing, should be classified as high risk for ACS. If clinical suspicion remains high, serial cTn testing is recommended, as late cTn rises have been described in <1% of people with NSTEMI.[124]

High-sensitivity troponin-based clinical decision pathways

The use of hs-cTn assays is recommended over contemporary troponin assays for safe and rapid decision-making. In people presenting with chest pain, hs-cTn-based risk stratification typically identifies 50–65% as low risk, 20–30% as intermediate risk and 15–25% as high risk for MACE.[125, 126] When combined with non-ischaemic ECG findings in validated algorithms, stratification of an individual's risk of adverse cardiac event can safely and effectively be achieved without clinical risk scores.[26, 117]

The 0-hour, 0/1-hour and 0/2-hour protocols are time-critical pathways designed to reduce myocardial damage and improve outcomes, using hs-cTn levels and clinical history to guide decisions. These strategies have been developed for most hs-cTn assays and the values are assay-specific, with details provided in Figure 11, Comprehensive Guideline Table 7 and Supplementary material B1.[25, 82, 117, 119, 124, 127–134]

Single high-sensitivity cardiac troponin measurements

A single hs-cTn measurement is not suitable to guide treatment decisions for people with symptom onset <2 hours. These people require serial testing.[119, 82, 131, 135–138] In people with symptom onset ≥2 hours, combining a single hs-cTn result with non-ischaemic ECG findings can very safely classify 20–50% of people presenting with possible ACS as low risk.[25, 30, 77, 81, 119, 123, 125-128, 131, 138–145]

Single hs-cTnT and hs-cTnI assays have been extensively validated, demonstrating high negative predictive alue and sensitivity for excluding index MI and a <1% risk of MACE during short- and longer-term follow-up.[23, 80, 81, 123, 125, 138, 145–148] Unlike hs-cTn assays, single contemporary troponin measurements have not been validated to assess risk.[149]

0/1- and 0/2-hour strategies

Index or 30-day MACE rates range between 2–22% for people identified as intermediate risk using the 0/1- or 0/2-hour strategies. Those deemed intermediate risk require additional evaluation (refer to Section Further diagnostic testing for people with suspected ACS).[25, 119, 120] For those with normal serial cTn values, 30-day MACE rates are ≤2%.[22]

While primarily evaluated in large observational studies, randomised trials of the 0/1-hour strategy have demonstrated 30-day MI and death rates of <1% when implemented successfully.[25, 130, 150–152] The thresholds for changes (deltas) in the 0/1- and 0/2-hour algorithms are both assay and time dependent, making collection of blood specimens within the specified windows critical (See Comprehensive Guideline Table 7).

In most hospitals, delays in central laboratory assay turnaround times render the 0/1-hour strategy impractical. POC hs-cTn assays may overcome this limitation but are not yet widely available. A 0/2-hour strategy is therefore currently the most practical option in most settings.

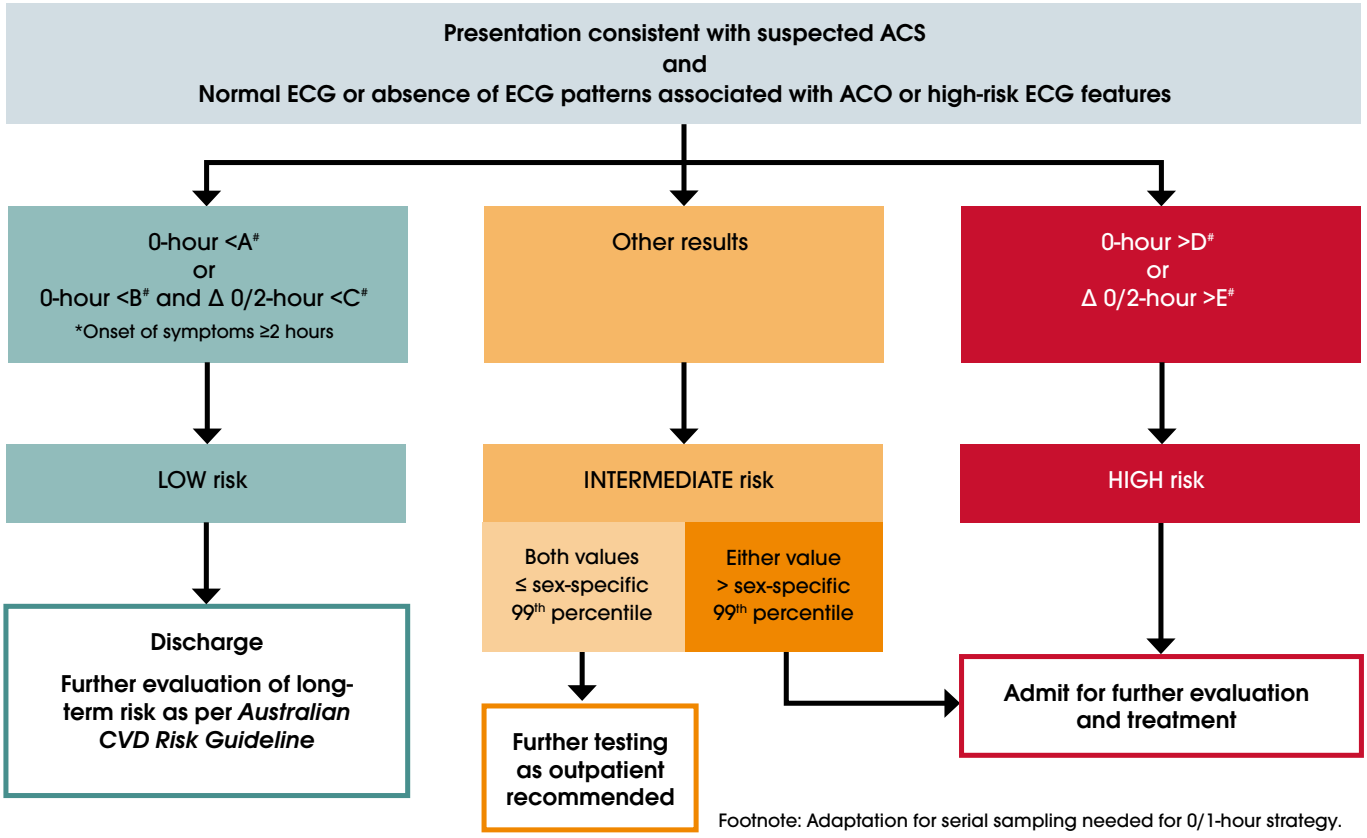


Figure 11 hs-cTn 0/2-hour testing recommendations.

Note: the 0/2-hour time points are shown in this figure. If using a 0/1-hour strategy, change timeframes accordingly.

#Refer to Table 7 in Comprehensive Guideline for interpretation of cTn assay-specific values and sex-specific 99th percentiles.

*All people with symptom onset <2 hours need serial testing. People with ongoing symptoms should be assessed according to high-risk criteria.

Abbreviations: ACO, acute coronary occlusion; ACS, acute coronary syndromes; ECG, electrocardiogram.

High-STEACS algorithm

The United Kingdom (UK) High-STEACS algorithm is a validated, safe and effective approach for the diagnosis and management of ACS using a variety of hs-cTn assays.[23, 123, 126, 130, 137] Further details are described in the Supplementary material B1.

Clinical score-based clinical decision pathways

Sites using contemporary cTn assays

Clinical score-based tools, such as EDACS and the HEART score, are recommended for assessing people with suspected ACS when using contemporary cTn assays. These tools are the most widely validated, demonstrating high sensitivity for index AMI and 30-day MACE (see Supplementary material B2).[141, 143, 153–161]

Compared to hs-cTn-based clinical decision pathways, clinical score-based tools identify fewer low- or intermediate-risk individuals and may require additional testing without significantly improving MACE outcomes.[30, 77, 118, 127, 128, 139, 144, 149, 162–164]

Furthermore, because sex-specific considerations are not included in all scoring systems, their effectiveness in men and women may not be equal.[165] Further information on these clinical score-based tools is provided in the Supplementary material B2.

Implementing a clinical decision pathway for suspected ACS

Multidisciplinary teams and clinical decision pathways for suspected ACS, especially those using hs-cTn assays, offer substantial benefits for people and healthcare systems.[22, 122, 134, 136, 152, 156, 157, 166–169] For example:

- Australian randomised control trial data showed that when using hs-cTnT, the 0/1-hour strategy resulted in more frequent ED discharge and a reduced ED length of stay. Similar clinical outcomes occurred at 30 days compared to usual care (0/3-hour cTn measurements with an hs-cTnT threshold of ≥30 ng/L).[150]
- A large, randomised trial showed failure to follow recommended management processes for low-risk people increased resource use with no differences in 30-day MACE.[122]

Centres choosing to implement an alternate strategy to the recommended CDPs should validate the chosen clinical decision pathways. Validation requires evaluating 30-day mortality and re-presentation with confirmed ACS in all people who presented with chest pain.

Practice points

Women

- Apply sex-specific 99th percentile upper reference limits when using hs-cTn assays.[1, 23, 91, 118, 132, 170]
  - Women are frequently misdiagnosed with non-ischaemic chest pain, and their ACS risk is often underestimated.[24, 29, 165, 171, 172] This is due in part to traditional risk tools lacking sex-specific considerations and clinician bias.

Older adults

- Use uniform hs-cTn cut-offs for clinical assessment, recognising that concentrations increase with age in healthy individuals. This may result in fewer older adults being classified as low risk for MI.[1]
  - In people aged 65 years and older with comorbidities such as renal impairment, the specificity of hs-cTn assays for MI is reduced.[25, 129, 170, 173]

First Nations peoples

- Use a single hs-cTn measurement in First Nations peoples to identify people as low risk of MI and 30-day MACE.[92]
- Be cautious when implementing the HEART score and IMPACT pathways, which have been evaluated in small studies in First Nations populations (see Supplementary material B2).[174]
- Investigate all First Nations adults (aged 18 years and over) with suspected ACS for underlying CAD, given their high risk of future cardiac events.[175, 176]

People with renal impairment

- Use hs-cTn-based strategies in people with renal dysfunction, noting that fewer individuals will be classified as low risk compared to other approaches.[25, 173]
  - Elevations in cTn are common in this population, leading to their exclusion from many assessment trials.[177] The safety of hs-cTn-based strategies appears to be similar in people with and without renal dysfunction.

Future direction

Newer strategies for individualised determination of likelihood of MI

Newer strategies to determine risk of MI have been developed based on large international datasets using machine learning techniques. These include the MI[3] algorithm study, the ARTEMIS study and the CoDE-ACS study.[178–180] Such strategies incorporate additional information (e.g. specific interval time of cTn testing, biometric measurements) to support decisions. Validation studies suggest large proportions of people can be defined as low risk, with improved specificity for MI in high-risk people.

Initial therapeutic management

Recommendations	Strength of recommendation	Certainty of evidence
In all people with suspected or confirmed ACS, give aspirin (300 mg orally, dissolved or chewed) unless contraindicated.	Strong	High
People with suspected or confirmed ACS with oxygen saturation (SpO2) ≥90% do not require oxygen therapy.	Strong	Moderate
In people with suspected or confirmed ACS receiving oxygen therapy, SpO2 should not exceed 96%.	Strong	Moderate
In the presence of ongoing chest pain, give glyceryl trinitrate sublingual tablet or spray every 5 minutes for up to three doses if no contraindications exist.	Consensus	
In people with chest pain and in the absence of contraindications, it is reasonable to administer intravenous (IV) fentanyl or morphine boluses.	Consensus	

Evidence supporting the recommendations

Aspirin

Aspirin reduces the risk of vascular events (vascular death, MI and stroke) in individuals with ASCVD, with benefits outweighing the small risk of major bleeding. A 300 mg loading dose is recommended to fully inhibit platelet activation, followed by a maintenance dose of 100 mg, which is as effective as higher doses.[181–183]

Oxygen therapy

Routine supplemental oxygen for suspected ACS without hypoxaemia does not improve mortality at 30 days or 12 months and is associated with increased risk of recurrent MI and revascularisation. Higher oxygen saturation levels are cautioned due to a dose–response link with increased mortality in acute and intensive care settings.[184–186]

Practice points

Oxygen therapy

- Provide oxygen therapy routinely if oxygen saturation falls below 90%, as hypoxaemia at this level is assumed to contribute to coronary ischaemia. The clinical outcome benefits remain uncertain.[184]
- Recognise that oxygen therapy is commonly administered when oxygen saturation is 90–92%, although its benefit is unknown.[187-189]
- Exercise caution in people with chronic obstructive pulmonary disease (COPD) when administering supplemental oxygen, aiming for a target arterial oxygen saturation (SaO2) of approximately 88–92%.

Nitrates

- Use IV glyceryl trinitrate for more effective symptom relief in acute ischaemia compared to the sublingual form but be aware that it does not improve prognosis.[190, 191]
- Do not administer glyceryl trinitrate in cases of hypotension, right ventricular infarction or recent use of phosphodiesterase 5 inhibitors (e.g. sildenafil, vardenafil, tadalafil). Consider alternative therapy if symptoms persist.

Opioid analgesia

- Titrate opioid doses to resolve chest pain, adjusting for individual needs based on age, comorbidities and concurrent medication use.[192–194]
- Consider fentanyl for its short time to peak effect, short duration of action and minimal impact on histamine release.
- Note that both morphine and fentanyl are associated with increased platelet reactivity, reduced antiplatelet effect of $P2Y_{12}$ inhibitors and slower absorption of oral medicines such as ticagrelor during the early hours of ACS.[193, 195–197]

Other medicines

- Do not administer non-steroidal anti-inflammatory drugs (NSAIDs) in confirmed ACS, especially during the early phase, due to an increased risk of MACE.[198, 199]
- Do not initiate additional antiplatelet, anticoagulation or beta blocker therapies without a confirmed or probable diagnosis of ACS. For further information refer to Section Antiplatelet therapy in the acute phase and Anticoagulant therapy in the acute phase.

Further diagnostic testing for people with suspected ACS

Recommendations	Strength of recommendation	Certainty of evidence
In people classified as intermediate risk (as defined by a validated CDP) with elevated troponin concentrations (>99th percentile), inpatient investigation is recommended.	Strong	Moderate
In people classified as intermediate risk without elevated troponin concentrations, consider outpatient investigation with non-invasive testing.	Consensus	
In people classified as low risk who remain symptom-free, further cardiac testing for CAD is not routinely required. Assess and manage cardiovascular risk factors.	Consensus	

Evidence supporting the recommendations

For people at intermediate risk, invasive angiography or non-invasive cardiac testing is recommended to refine risk stratification, identify alternative causes of chest pain and assess future risk of ACS beyond 30 days. Inpatients with elevated hs-cTn levels above the 99th percentile should undergo testing due to a 30-day cardiac event rate of 2–22%, while those with hs-cTn ≤99th percentile may consider outpatient testing within 30 days, as their event rate is <2%.[16] Non-invasive testing is not routinely recommended for low-risk individuals, as their likelihood of cardiac events over two years is minimal. General practitioner follow-up is advised for symptom resolution, treatment and assessment of long-term cardiovascular risk using Australian guidelines (cvdcheck.org.au).[22, 26, 88, 118, 122, 123, 125, 137, 146, 200–204]

Practice points

Non-invasive test selection – anatomical versus functional

Anatomical testing (CTCA)

- Use computed tomography coronary angiography (CTCA) as a first-line investigation for people without previously known coronary artery disease (CAD) presenting with intermediate-risk ACS, if no contraindications exist (see Comprehensive Guideline Table 8).
- Consider functional testing with close monitoring and a graduated exercise regime for people with contraindications to CTCA.
- Recognise that a normal CTCA (ruling out both obstructive and non-obstructive plaque) reliably excludes ACS and indicates an extremely low risk of ACS for at least 4–5 years.[205–210] Identifying non-obstructive plaque on CTCA can guide preventative therapies, such as lipid-lowering treatment.
- Do not rely on coronary artery calcium scoring alone in ACS evaluation unless combined with CTCA.

Functional testing

- Favour functional testing for people with known CAD, prior stents or extensive coronary calcification, where CTCA interpretation may be more challenging. Functional tests can help identify whether symptoms are caused by obstructive plaque and assess ischaemic burden and short-term prognosis.
- Select functional tests based on clinical needs, including stress echocardiography, stress cardiac magnetic resonance imaging (MRI), stress/rest single-photon emission computed tomography (SPECT), stress/rest positron emission tomography (PET), or exercise ECG (see Comprehensive Guideline Table 8).
- Recognise the additional diagnostic benefits of stress cardiac MRI and echocardiography, such as evaluating left ventricular function, regional wall motion abnormalities and valvular function, and excluding differential diagnoses like myopericarditis and Takotsubo cardiomyopathy.

Considerations for test selection

- Consider individual cardiovascular risk factors, local expertise and the availability of health services, particularly in regional and remote areas, when selecting non-invasive cardiac investigations as these all may influence the selection.[26, 211–213]
- Prioritise inpatient non-invasive testing for low-risk individuals with factors limiting access to timely follow-up or the ability to re-present to ED, such as First Nations peoples with suspected ACS or those facing sociodemographic challenges.[175]

Cost-effectiveness

Reducing unnecessary testing has benefits for the individual and health services. For example, an Australian study estimated a total cost saving of $13.5 million per annum after implementation of an accelerated diagnostic pathway (using cTnI, ECG and TIMI score) that reduced hospital admission rates and ED length of stay.[157]

Considerations for regional, remote and First Nations peoples

Regional, remote and First Nations peoples are disproportionately affected by reduced access to healthcare services, longer wait times and greater travel distances to diagnostic services. Definitive early identification of CAD using CTCA may be of significant benefit in this group because it is a relatively more accessible imaging technology.[214] To note, there is limited evidence on how long a negative CTCA ensures low risk before retesting.

An Australian telemedicine program has demonstrated the potential to reduce waiting times by supporting remote exercise stress testing with specialist cardiology support. This initiative has enabled a significant number of people to be managed within their local health facilities, improving access and reducing the need for travel.[215]

Role of rapid access chest pain clinics

Rapid access chest pain clinics provide screening, investigations and management for people presenting with chest pain, including those discharged after an ACS.[196] Studies from the UK report these clinics as safe, efficient and cost-effective alternatives to hospital admission.[216] Australian models of chest pain clinics have shown comparable outcomes, demonstrating similar benefits regardless of referral patterns or specific investigations undertaken.[217]

These services offer improved access to diagnostic tests, individual satisfaction, and cost savings. They have shown safety outcomes comparable to, or better than, traditional hospital-based care, with reduced rates of invasive investigations, fewer ED re-presentations, and streamlined follow-up of test results.[218–222] Access should be prioritised for selected people at intermediate risk with cTn levels below the 99th percentile.

Re-presentation with symptoms

People re-presenting to ED within 30 days with possible ACS symptoms without prior non-invasive testing for CAD and/or coronary ischaemia may warrant further functional or anatomical testing. A detailed reassessment for alternate diagnoses is also required. If prior exercise ECG testing was negative, more sensitive and specific investigations or anatomical tests should be considered.

Primary care and regional and remote presentations

Recommendations	Strength of recommendation	Certainty of evidence
For people with suspected ACS initially evaluated in the primary care setting, prompt transfer to a facility where definitive risk assessment can occur (e.g. ED) is recommended.	Consensus	
Metropolitan health services should establish centralised support systems for regional and remote health services to facilitate: • prompt assistance with ECG interpretation and access to troponin results when on-site access is not available • provision of clinical advice to healthcare professionals • access to cardiac investigations if required.	Strong	Low

Evidence supporting the recommendations

All individuals with suspected ACS should have access to best-practice care, regardless of location. Centralised and coordinated care systems, supported by telehealth, ensure prompt specialist input for services outside tertiary centres.[223]

An Australian model demonstrated reduced mortality with early cardiologist support for ECG interpretation, POC troponin testing and decision-making. Data also show fewer missed STEMIs when tertiary-level support was routinely available compared to usual care in hospitals without an emergency physician.[54]

Considerations for primary care presentation

Initial assessment

As outlined previously, assessment of ACS incorporates:

- ECG findings
- clinical findings from history and physical examination
- troponin testing.

The ability of a healthcare professional or clinic to reliably diagnose or exclude ACS is determined by their capacity to perform and interpret these components. If the clinician suspects ACS, transfer to the nearest medical facility where capacity for definitive assessment for ACS can occur is mandatory.

Initial ECG assessment

People presenting with suspected ACS require prompt access to an ECG (within 10 minutes) and interpretation by a suitably trained clinician. If ACS is suspected, recording an ECG should not delay transfer to a facility that can perform serial troponin testing and provide reperfusion therapy, as delays are associated with greater harm.[224–229]

If an ECG cannot be performed within 10 minutes, prompt transfer via ambulance to a location where an ECG can be performed is necessary. This may mean the first ECG is evaluated by trained paramedics.

If an ECG is non-ischaemic and the clinical presentation does not align with ACS as the likely diagnosis, it is reasonable to continue assessment in the primary care setting.

Troponin testing

If ACS remains a possible diagnosis after initial history, examination and ECG assessment, and cTn testing is required, the person should be transferred to the nearest facility (usually an ED) for definitive risk assessment.[21, 26, 230]

In Australia, there are no single test strategies using POC contemporary troponin assays to exclude AMI.[231] Serial testing is required and typically cannot be performed in the primary care setting. hs-cTn POC assays are available, but not widely distributed. Currently, there is limited evidence for single test strategies to exclude MI in primary care settings.[89]

Risk assessment and clinical decision pathways

Risk scores such as the Marburg Heart Score, Grijseels and Bruins Slot rules are not recommended for excluding ACS in a primary care setting.[232] A systematic review of older risk assessment rules without cTn results found no difference between the use of these scores and a general practitioner's clinical judgement in ruling out ACS.[233]

Considerations for regional and remote presentations

While the initial assessment remains unchanged, key factors influencing the decision to transfer people with suspected ACS include:[19, 20, 234, 235]

- local service capabilities and support availability in regional and remote settings
- availability of relevant investigations including chest X-rays, cTn testing and/or other cardiac tests (e.g. CTCA, exercise stress testing, echocardiography)
- the healthcare professional's clinical judgement.

Initial ECG assessment

If the ECG can be performed but not interpreted, it is reasonable to seek urgent remote evaluation (e.g. via telehealth).[54]

When the clinical and ECG assessment supports a diagnosis of ACOMI, consideration of urgent reperfusion therapy is required. Urgent transfer of the person to the nearest facility for fibrinolysis or primary PCI is needed (refer to Section 2 Hospital care and reperfusion).

Troponin testing

Many regional and remote settings are reliant on contemporary cTn assays, including POC platforms.[112] Clinicians must be aware of the type of troponin assay in use locally and ensure results are used in an evidence-based clinical decision pathway (refer to Section Biomarkers and Section Risk assessment and clinical decision pathways for suspected ACS).

Where contemporary cTn assays are in use, UA should be considered in the presence of normal cTn results if clinical suspicion for ACS is high based on ECG interpretation and/or clinical history. Further serial cTn testing over 6–8 hours should occur.[21] Management may include initial treatment for presumed ACS, a period of continuous cardiac monitoring and/or transfer to a PCI-capable centre.

Risk assessment and clinical decision pathways

Evidence supporting the use of clinical risk scores without incorporating troponin values is limited. In the absence of hs-cTn assays, incorporation of contemporary cTn results with clinical risk scores within a validated clinical decision pathway is crucial (refer to Section Clinical score-based clinical decision pathways and Supplementary material B2). In a rural New Zealand setting, use of the EDACS accelerated diagnostic pathway with serial POC contemporary cTn measurements safely stratified risk in people with suspected ACS (see Supplementary material B2).[236]

Further diagnostic testing

If a particular diagnostic test is required but unavailable regionally, transfer to another facility should be considered.

Tertiary centres have an obligation to support appropriate testing in people from regional and remote areas. Decisions on further diagnostic testing can be informed by consultation with metropolitan cardiac teams or, when available, rapid access chest pain clinics. Remote access to such clinics may help improve diagnostic pathways for people in regional and remote settings.

Discharge planning and advice

Following a comprehensive and structured assessment, people with suspected ACS who do not require admission for further assessment and/or management can be discharged.

The outcome of ED assessment will determine the guidance provided to the person (and their support people) prior to discharge.

While many people will not receive a definitive diagnosis for their symptoms, life-threatening conditions like AMI and UA will have been deemed to be of very low probability.[237] Specific discharge advice for non-ACS presentations is beyond the scope of this guideline

Discharge planning and advice supports reduced ED presentations and leads to better outcomes. Clinicians, including registered nurses and nurse practitioners, should be supported to undertake comprehensive pre-discharge assessment and discharge planning. This is important to help manage a person's anxiety; high levels of anxiety are associated with an increased likelihood of symptom recurrence and re-presentation to hospital.[238, 239]

Discharge communication for general practitioners

To support ongoing management of people after discharge, concise information in the form of a discharge summary must be promptly provided to a person's general practitioner.

Discharge advice for people at low risk for ACS

During discharge, people classified as low risk should be provided with written information and verbal advice that includes:

- evidence-based information on the condition(s) diagnosed and any further investigation/management required
- clear communication that the person has been comprehensively assessed to exclude AMI and to determine UA is unlikely
- a clear statement that CAD has not been excluded, and that follow-up with the person's general practitioner is recommended for assessment and management according to the *Australian guideline for assessing and managing cardiovascular disease risk* (cvdcheck.org.au)
- information about what steps the person should take if they experience recurrent symptoms
- education on cardiovascular health and cardiovascular causes of chest pain and other key symptoms
- guidance on where to find reliable sources of online health information and how to contact telephone-based triage services.

The use of a decision-support tool may assist in conveying risks of heart disease and of the lack of benefit, and possible harm, of further testing in low-risk people (see Supplementary material B2).[122]

Discharge advice for people at intermediate risk for ACS

During discharge, people classified as intermediate risk should be provided with written information and verbal advice that includes:

- clear reassurance for the person and their support people that they have been comprehensively assessed as safe to be discharged
- clear information on management of existing and/or new symptoms, including when to call an ambulance, re-present to the ED or contact their general practitioner
- information on referral for outpatient assessment and management; this should include clear guidance on the clinician/clinic referred to, and whether an appointment has already been arranged or whether the person or support people need to do this; if the latter, then a clear timeline and contact details to arrange the appointment should be provided in the written discharge advice
- clear guidance on new or continuing medicines, including when and how to take them
- education on cardiovascular disease risk and steps that the person can take to reduce their risk
- guidance on where to find reliable sources of online health information and how to contact telephone-based triage services.

Discharge advice for people with a prior history of coronary heart disease who have a chronic or stable coronary syndrome

A small number of people presenting to the ED with a prior diagnosis of CAD may be discharged with probable chronic or stable CAD. The advice given needs to balance:

1. reassurance that the person has been comprehensively assessed and is deemed safe for discharge at this time, notwithstanding a probable cardiac cause for their symptoms and presentation
2. a clear plan for further follow-up for assessment and management
3. a clear plan for managing existing or new symptoms
4. education on their cardiovascular health and reiteration of when to call an ambulance, re-present to ED or contact their general practitioner.

Shared decision-making is strongly encouraged. More research is needed on how to best implement shared decision-making to achieve the goals of people at risk of or living with cardiovascular disease.[6–10]

2. Hospital care and reperfusion

Acute management of STEMI – reperfusion for STEMI

Eligibility for reperfusion

Recommendations	Strength of recommendation	Certainty of evidence
In people with STEMI, perform emergency reperfusion with either primary PCI or fibrinolytic therapy within 12 hours of symptom onset.	Strong	Moderate
In people with STEMI whose symptoms lasted more than 12 hours before presentation and have evidence of continuing myocardial ischaemia (persistent ischaemic symptoms, haemodynamic compromise and/or life-threatening arrhythmias), perform emergency reperfusion with primary PCI.	Strong	Moderate

Evidence supporting the recommendations

Timely reperfusion reduces the extent of MI and mortality, with the greatest benefit within the first hour and diminishing after 12 hours.[240–244] Routine reperfusion beyond 12 hours is not recommended unless ongoing ischaemia is present, where studies have shown primary PCI in the presence of ongoing ischaemia may improve survival, reduce infarct size and lower four-year mortality.[245–248] For people with STE and multivessel disease, complete revascularisation should be the goal during PCI.

Practice points

- Assess cognitive function, comorbidities and frailty when determining eligibility for reperfusion, as these factors significantly influence overall survival outcomes.
- Consider percutaneous coronary intervention (PCI) as it shows advantages over fibrinolysis in older populations. However, note that evidence is limited by small sample sizes, a lack of data for individuals over 90 years, and the absence of assessments for frailty or comorbidities in supporting trials.

Choice of reperfusion strategy

Recommendations	Strength of recommendation	Certainty of evidence
PCI is the preferred repercussion strategy in people with STEMI whose symptoms have lasted less than 12 hours. PCI should be performed within 120 minutes of first medical contact.	Strong	High
Fibrinolysis should be performed in people with STEMI whose symptoms have lasted less than 12 hours if primary PCI cannot be delivered within 120 minutes of first medical contact.	Strong	Moderate

Evidence supporting the recommendations

Reperfusion for STEMI involves either primary PCI or fibrinolytic therapy, with PCI preferred if it can be performed within 120 minutes of first medical contact. At PCI-capable centres, wire crossing should occur within 60 minutes, or within 90 minutes for people who have been transferred.[249] Fibrinolysis reduces 35-day mortality compared with no treatment.[250] However, PCI is more effective than fibrinolysis in reducing short- and long-term risks of death, non-fatal reinfarction and stroke. Early fibrinolysis followed by angiography may be comparable to PCI, particularly if initiated within two hours of symptom onset.[241]

Administering fibrinolysis very early, including pre-hospital administration, may result in better outcomes than PCI for people presenting within two hours of symptom onset.[236] Fibrinolysis is not recommended after 12 hours post-symptom-onset; instead, PCI is preferred for people with ongoing myocardial ischaemia.[247, 248, 250–256] For a decision-making chart see Figure 12 in the Comprehensive Guideline.

Practice points

- Establish effective care pathways tailored to the specific healthcare services available in the region. These pathways should include ambulance, primary health, emergency, cardiology and regional or remote healthcare services to optimise reperfusion times.
- Implement specific measures to reduce time to reperfusion,[257] such as:
  - pre-hospital ECG and single-call catheter laboratory activation (i.e. streamlined communication between emergency services and the receiving hospital to bypass the ED and reduce reperfusion delay)
  - pre-hospital fibrinolysis by suitably trained clinicians (e.g. paramedics, nurses, First Nations health practitioners)
  - direct transfer to PCI-capable hospitals and direct transfer to the catheterisation laboratory on hospital arrival.

Administration of fibrinolytic therapy

Recommendations	Strength of recommendation	Certainty of evidence
In people with STEMI for whom fibrinolysis is the preferred reperfusion strategy, it should be delivered within 30 minutes of first medical contact. Consider pre-hospital administration.	Strong	Moderate
In people aged ≥70 years, half the standard dose of tenecteplase is recommended as part of a pharmaco-invasive strategy.	Strong	Moderate

Evidence supporting the recommendations

Timing of fibrinolytic therapy

Fibrinolysis should be considered when primary PCI is delayed by more than 120 minutes and there are no absolute contraindications. It should be administered as soon as possible, ideally within 30 minutes of first medical contact, and, if feasible, before hospital arrival. People with absolute contraindications should be transferred for PCI (Table 9). People with a relative contraindication need to have the risks and benefits of treatment considered.[249, 257–259]

Table 9. Contraindications for fibrinolysis. Adapted with permission from O'Gara et al.[229]

Absolute contraindications	Relative contraindications
• Any prior intracerebral haemorrhage • Known structural cerebral vascular lesion (e.g. arteriovenous malformation) • Known malignant intracranial neoplasm (primary or metastatic) • Ischaemic stroke within 3 months • EXCEPT acute ischaemic stroke within 4.5 hours • Suspected aortic dissection • Active bleeding or bleeding diathesis (excluding menses) • Significant closed-head or facial trauma within 3 months • Intracranial or intraspinal surgery within 2 months	• History of chronic, severe, poorly-controlled hypertension • Significant hypertension on presentation (SBP >180 mmHg or DBP >110 mmHg) • History of prior ischaemic stroke >3 months • Known intracranial pathology not covered in absolute contraindications • Dementia • Traumatic or prolonged (>10 min) CPR • Major surgery (<3 weeks) • Recent internal bleeding (within 2 to 4 weeks) • Non-compressible vascular punctures • Pregnancy • Active peptic ulcer • Oral anticoagulant therapy

Abbreviations: CPR, cardiopulmonary resuscitation; DBP, diastolic blood pressure; SBP, systolic blood pressure.

Dosing fibrinolytic therapy in older people

A comparison of pre-hospital fibrinolysis with angiography 6–24 hours later against primary PCI in people unable to receive the PCI within 60 minutes showed no significant difference in outcomes such as death, cardiogenic shock, heart failure or recurrent MI. In people aged 75 and older, full-dose tenecteplase was associated with higher rates of intracranial haemorrhage, a risk reduced by halving the dose without affecting efficacy.[258] Further research in older people (over 60 years, mean age 70 years) confirmed similar efficacy between half-dose tenecteplase and routine angiography 6–12 hours later against primary PCI, although intracranial haemorrhage was slightly higher in the fibrinolysis group (1.5% vs 0%), half of these events linked to dosing errors.[260]

Practice points

- An easily administrable fibrinolytic agent that can be given as a bolus dose, such as tenecteplase, is advisable, especially in the pre-hospital setting.
- Currently available fibrinolytics include:
  - tenecteplase (weight-adjusted 30–50 mg IV bolus and age-adjusted half-dose IV bolus)
  - reteplase (10 units IV followed by 10 units IV, 30 minutes later)
  - alteplase (weight-adjusted accelerated bolus and infusion regimen).

Procedural recommendations in primary percutaneous coronary intervention

Recommendations	Strength of recommendation	Certainty of evidence
For people with STEMI at a PCI-capable centre, deliver primary PCI within 60 minutes of arrival. For people with STEMI transferred from a non-PCI centre, deliver primary PCI within 90 minutes of first medical contact.	Consensus	
Use radial access over femoral access when performing primary PCI, unless contraindicated.	Strong	High
In people undergoing primary PCI, do not perform routine thrombus aspiration of the infarct-related artery (IRA).	Strong	Moderate
In people who are asymptomatic and stable for more than 48 hours following occlusion of an IRA, do not perform routine PCI to this artery.	Strong	Moderate

Evidence supporting the recommendations

Time targets for primary PCI

For people diagnosed with STEMI, reducing treatment delays from first medical contact to reperfusion is crucial for improving mortality outcomes.[261] Recommended targets include primary PCI within 60 minutes for those arriving at a PCI-capable centre, or within 90 minutes for those transferred from a non-PCI-capable centre.[249] Pre-hospital diagnosis and direct activation of the catheterisation laboratory, and bypassing ED on arrival, can minimise delays.[262, 263]

Radial versus femoral access

Radial access is preferred for primary PCI in STEMI due to its association with lower mortality (1.6% vs 2.1%) and major bleeding (1.5% vs 2.7%) compared to femoral access.[264–268] A radial-first approach is recommended unless contraindicated.[117, 258, 264, 269]

Treatment of the infarct-related artery

Thrombus aspiration of the infarct-related artery (IRA) carries a small increased risk of stroke without survival benefits and may be considered for individuals with a high thrombus burden. Technical strategies to minimise embolisation should be employed.[270–272]

When stenting is required, drug-eluting stents are preferred over bare metal stents due to lower rates of restenosis and stent thrombosis, including in individuals at high bleeding risk, those requiring triple antithrombotic therapy or short-duration dual antiplatelet therapy (DAPT).[249, 273–278]

Routine deferred stent implantation in people with STEMI does not improve outcomes compared with standard immediate stent implantation, and the need for unplanned target vessel revascularisation may be increased. However, it may be considered in cases of significant thrombus burden where immediate PCI is unlikely to succeed.[279–282]

Routine PCI of a completely occluded IRA beyond 48 hours in asymptomatic, stable individuals is not advised, as it may increase the risk of recurrent MI without improving survival or major cardiovascular outcomes.[253, 283]

Practice points

- Where stenting is required, drug-eluting stents are preferred over bare metal stents.
- Routine deferred stenting of the IRA is not recommended. In people with STEMI and risk factors for slow or no reflow, such as high thrombus burden, consider deferred stent implantation.
- In people with STEMI where primary PCI of the IRA is not feasible (e.g. severe left main artery disease or an uncrossable coronary lesion), CABG may be an appropriate primary reperfusion strategy. CABG may be particularly appropriate if there is a large area of myocardium at risk and surgery is available in a timely manner (refer to Section Coronary artery bypass graft surgery in ACS).[284, 285]

Women

- Understand that women with STEMI have documented delays to reperfusion, lower rates of invasive angiography and radial access as well as poorer outcomes compared with men.[13–15]
- Improve clinician awareness of sex-specific differences in:
  - presenting symptoms
  - ECG diagnostic criteria
  - underlying MI aetiologies, such as a higher prevalence of SCAD and MINOCA, as these may improve outcomes.
- Perform primary PCI as the preferred revascularisation strategy in pregnant women with STEMI, except when caused by SCAD. Use appropriate shielding to minimise radiation exposure to the foetus.[269]

Ongoing management of fibrinolytic-treated people

Recommendations	Strength of recommendation	Certainty of evidence
People successfully treated with fibrinolytic therapy should be transferred to a PCI-capable centre as soon as possible. Angiography should be performed within 2–24 hours upon arrival.	Strong	Moderate
Consider transferring people as soon as possible to a PCI-capable centre if fibrinolytic therapy is unsuccessful. If appropriate, consider subsequent PCI at the centre.	Weak	Moderate

Evidence supporting the recommendations

Primary PCI is associated with the lowest mortality compared to fibrinolysis alone.[286] Among individuals receiving initial fibrinolysis, a pharmaco-invasive approach (PCI ≥2 hours after fibrinolysis) reduces reinfarction and trends towards lower mortality compared to fibrinolysis alone or facilitated PCI (<2 hours).[286] A Bayesian analysis further suggested that the probability of adverse outcomes was lower with the pharmaco-invasive approach compared to facilitated PCI.[286]

Routine early PCI after fibrinolysis significantly reduces reinfarction and the composite of death and reinfarction at 30 days, with benefits sustained at 12 months and no significant increase in major bleeding.[287] The greatest benefit is achieved when PCI is performed as soon as possible after fibrinolysis, without shifting to facilitated approaches.[288]

Rescue PCI for failed fibrinolysis reduces reinfarction but does not impact mortality.[289] For people in hospitals without PCI capability, pathways should support early transfer for angiography when indicated.[290]

Practice points

- Perform regular ECG monitoring following fibrinolytic therapy according to local protocols, continuing until the person is pain-free and for at least 60–90 minutes post-fibrinolysis.
- Recognise failed reperfusion in people treated with fibrinolytic therapy if they exhibit any of the following:
  - ongoing ischaemic chest pain
  - ≤50% ST recovery on an ECG performed 60–90 minutes after fibrinolysis
  - ongoing haemodynamic instability.

First Nations peoples

- Ensure that education about ongoing management is culturally appropriate and includes the importance of transferring to a PCI-capable centre when necessary.
- Provide cultural and language support before, during and after transfer, ensuring family and cultural considerations are addressed by appropriately trained staff.

People living in regional and remote areas

- Establish formal care pathways to facilitate timely and efficient transfer between non-PCI-capable centres and PCI-capable centres, often located in metropolitan areas. These pathways should address logistical challenges and ensure seamless continuity of care.
- Consider an additional half-dose of fibrinolytics with caution if fibrinolysis fails and timely transfer for rescue PCI is not feasible. However, this practice point is based on limited evidence from a single prospective trial with limited applicability to contemporary remote Australian settings.[291]

Acute management of NSTEACS

Risk stratification for people with confirmed NSTEACS

Recommendations	Strength of recommendation	Certainty of evidence
In people with NSTEACS, consider using the GRACE risk score to determine short- and long-term cardiovascular prognosis.	Weak	High
In people with ACS undergoing coronary angiography, consider using bleeding risk scores to determine short-term bleeding risk.	Weak	Moderate

Evidence supporting the recommendations

Assessment of the short- and longer-term risk of death and recurrent ischaemic and bleeding events in people admitted with ACS can guide the need for, and timing of, invasive management. Risk assessment can also guide selection and duration of antithrombotic therapy. Clinical assessment and objective tools may both contribute to risk stratification in people with confirmed NSTEACS.

Clinical risk assessment

A subset of people present with factors that are associated with a high risk of short-term mortality, including haemodynamic instability/cardiogenic shock, life-threatening arrhythmias, mechanical complications of MI, acute heart failure clearly related to NSTEACS, and/or ongoing symptoms in the presence of high-risk ECG changes. These changes may include ST-segment depression >1 mm in more than six leads, STE in aVR and/or V1, Wellens criteria or recurrent intermittent STE (see Initial ECG assessment). An early invasive management strategy is recommended for these individuals.

In the absence of these very high-risk criteria, clinical risk assessment performs poorly compared with objective risk tools in determining prognosis.

Objective risk prediction for ischaemic outcomes

The Global Registry of Acute Coronary Events (GRACE) risk score is a more accurate predictor of prognosis in NSTEACS compared to the thrombolysis in myocardial infarction (TIMI) risk score or subjective clinical assessment.[20, 276, 292–299] A stronger predictor of 30-day death or MI is baseline hs-cTn levels.[300]

Risk prediction for bleeding outcomes

Major bleeding in hospital is associated with increased mortality, and a range of scores have been developed to predict this outcome among people presenting with ACS. The parameters of bleeding risk score is presented in Table 10 of the Comprehensive Guideline. A comparison of scoring systems reported that *acute coronary treatment and intervention outcomes network (ACTION)* was the most accurate at predicting outcomes, followed by *can rapid risk stratification of unstable angina patients suppress adverse outcomes with early implementation of the American College of Cardiology/American Heart Association guidelines (CRUSADE)* and *acute catheterization and urgent intervention triage strategy* (ACUITY).[301] The Academic Research Consortium high bleeding risk (ARC-HBR) score is an alternative pragmatic approach recommended by European guidelines.[302]

These scores were developed in populations with a high prevalence of coronary angiography and DAPT use. While they may be considered when choosing procedural and antiplatelet strategies, their impact on outcomes has not been established.

Practice points

- Recognise that GRACE risk scores were developed before the introduction of hs-cTn assays. However, most people identified as high risk by the GRACE score are similarly identified using hs-cTn testing alone.
- Prioritise bleeding risk over ischaemic risk when making decisions about the duration of DAPT in people at risk of both bleeding and ischaemic events, as suggested by observational data. Refer to sections Antiplatelet therapy in the acute phase and Anticoagulant therapy in the acute phase.[303]

Women

- Use the GRACE 3.0 score for risk assessment in women with NSTEACS, as it provides a more accurate estimation of mortality risk than the GRACE 2.0 (which underestimates mortality) and helps address sex inequalities in risk stratification.[304]

Older adults

- Consider that the GRACE risk score heavily weights age and does not account for characteristics common in older adults, such as frailty, multimorbidity, polypharmacy and cognitive dysfunction, which can contribute to higher risk scores.[305]
- Assess frailty in older adults, as it is independently associated with adverse outcomes and increased bleeding risk.[306] Use validated frailty assessment tools to guide management decisions.[307–310]
- Consider a conservative management approach in older adults, even if they are deemed high risk for ischaemic events based on objective scoring, particularly when frailty and bleeding risk are significant concerns.

Routine versus selective invasive management for NSTEACS

Recommendations	Strength of recommendation	Certainty of evidence
In people with NSTEACS at high or very high risk of adverse cardiovascular events, perform routine invasive coronary angiography, with coronary revascularisation (PCI or CABG) where appropriate.	Strong	High
In people with NSTEACS at low or intermediate risk of adverse cardiovascular events, testing for inducible ischaemia (e.g. stress testing) may guide the need for invasive coronary angiography.	Weak	Moderate

Evidence supporting the recommendations

The following table outlines the criteria used to identify people with confirmed NSTEACS at high and very high risk of adverse cardiovascular events or death.

High risk	Very high risk
Criteria: • Confirmed diagnosis of NSTEMI according to the Fourth UDMI • High risk according to hs-cTn algorithms (High-sensitivity troponin-based clinical decision pathways) • Dynamic ST-segment or T wave changes • Transient STE • GRACE risk score >140	**Criteria:** • Haemodynamic instability or cardiogenic shock • Life-threatening arrhythmias • Mechanical complications of MI • Ongoing symptoms in the presence of ECG criteria such as ST-segment depression >1 mm in >6 leads additional to STE in aVR and/or V1, or Wellens criteria on ECG (Initial ECG assessment) • Recurrent intermittent STE

In people with NSTEACS at high or very high risk of adverse cardiovascular events, a routine invasive strategy can reduce the composite endpoints of death, recurrent MI and rehospitalisation for ischaemia; with most benefit from preventing non-fatal events.[311–316] A meta-analysis showed reductions in MI and death, with absolute reductions in MI and cardiovascular death of 2% for low-, 4% for intermediate- and 11% for high-risk groups (classified by the GRACE score).[313, 315] Findings align with current practice despite pre-dating hs-cTn use.

For lower-risk individuals, non-invasive anatomical or functional testing can guide the need for invasive angiography, reducing unnecessary procedures with good short- and mid-term prognosis (Section Further diagnostic testing for people with suspected ACS).[317–320]

Practice points

- Consider the goals of therapy, individual preferences and the impact of major comorbidities when deciding on the appropriateness of a routine invasive approach for people with NSTEACS.
- Use anatomical imaging with CTCA instead of functional testing to exclude or define CAD in people with NSTEACS who:
  - are not at high or very high risk of adverse cardiovascular events
  - do not have known CAD
  - present with an unclear NSTEACS diagnosis, as detailed in Further diagnostic testing for people with suspected ACS.[321, 322]

  This approach can enhance diagnostic clarity and tailor management strategies effectively.

Timing of invasive management for NSTEACS

Recommendations	Strength of recommendation	Certainty of evidence
In people with NSTEACS with a very high-risk of adverse cardiovascular events, immediate invasive procedure within 2 hours of diagnosis is recommended.	Consensus	
In people with NSTEACS with high-risk of adverse cardiovascular events, consider early invasive procedure within 24 hours of diagnosis.	Weak	High

Evidence supporting the recommendations

Studies on the timing of invasive coronary angiography in NSTEACS, comparing early intervention (e.g. within 24 hours) with delayed intervention (e.g. 2–3 days), found no overall benefit in mortality, MI or stroke when applied to all participants without considering individual risk. Risk stratification should guide timing decisions NSTEACS.[323–325]

For unstable or very high-risk individuals, immediate angiography (within two hours) is recommended based on poor outcomes without intervention, although this is supported by expert opinion rather than robust evidence (Figure 13).

In people at high risk (e.g. GRACE score >140), early intervention reduced death, MI and stroke at 6 months compared to delayed strategies (14% vs 21%), without increasing major bleeding. Mortality benefits were also observed in those with elevated biomarkers, diabetes, GRACE score >140, or aged ≥75 years, but evidence for specific risk-treatment interactions is limited. Data using hs-cTn-based GRACE scores remain unavailable.[323, 326]

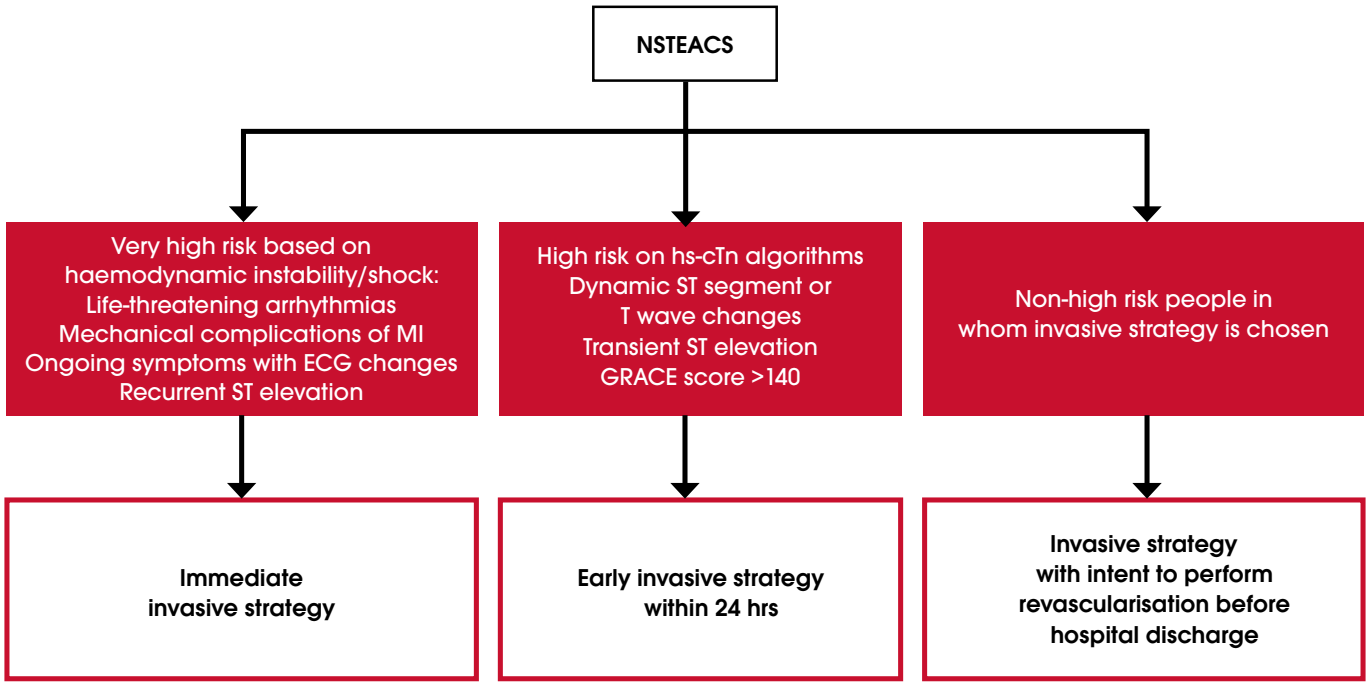


Figure 13 Timing of invasive management for NSTEACS.

Abbreviations: ECG, electrocardiogram; GRACE, Global Registry of Acute Coronary Events; hs-cTn, high-sensitivity cardiac troponin; MI, myocardial infarction; NSTEACS, non-ST-segment elevation acute coronary syndromes.

Procedural considerations in NSTEACS

Recommendations	Strength of recommendation	Certainty of evidence
In people with NSTEACS undergoing an invasive approach, radial access is preferred to femoral access, unless contraindicated.	Strong	High
In people with NSTEACS undergoing an invasive approach, consider intravascular imaging to guide PCI.	Weak	High

Evidence supporting the recommendations

Studies consistently show that radial access reduces mortality (1.6% vs 2.1%) and major bleeding (1.5% vs 2.7%) compared to femoral access in people with NSTEACS.[264–268] A radial-first approach is recommended unless there is a lack of operator experience or there are contraindications.

Intravascular imaging (IVI)-guided PCI, using optical coherence tomography or intravascular ultrasound (IVUS), reduced target lesion failure by lowering the risks of cardiac death, target vessel MI and target lesion revascularisation, in addition to reducing all MI and all-cause death compared to angiography-guided PCI.

Outcomes were similar for optical coherence tomography- and IVUS-guided procedures.[327] The benefit of IVI-guided PCI was of similar or greater magnitude in people with ACS, particularly for complex lesions and higher-risk individuals (e.g. bifurcations, calcifications, long lesions or diabetes).[328] However, recommendations for IVI-guided PCI should be tailored and not applied universally to all PCI procedures.

Practice points

Women

- Use a radial-first approach where possible for coronary interventions in both women and men, as it is associated with reduced complications and improved outcomes.
    - Consider a routine invasive approach for women with NSTEACS, as it has demonstrated benefits. Do not overlook the disparity in care, as observational data show women are less likely than men to receive an invasive strategy or radial access.[315, 329]

Older adults

- Consider an invasive strategy over an initial conservative approach in older adults with NSTEACS if they do not have frailty, multimorbidity or cognitive dysfunction based on objective assessment. The evidence to consider is:
    - Of five trials on invasive management in older adults with NSTEACS (mostly ≥75 years), four found no benefit in primary endpoints, but one showed reduced MI and urgent repeat revascularisation with a routine invasive strategy.[330–335]
    - For older individuals with MI and multivessel disease, physiology-guided complete revascularisation showed benefits, although frailty data were not provided.[336, 337]
    - Meta-analyses suggest that an invasive strategy likely reduces MI and recurrent revascularisation compared to conservative management, with observational studies suggesting a survival benefit and randomised trials showing a trend towards improved survival. However, this must be balanced against an increased risk of bleeding.[337–339]
    - A small trial of frail individuals aged over 70 years (mean age 86) with NSTEACS found no benefit from an initial invasive approach.[340]
- Individualise treatment decisions for older adults, balancing the potential for improved outcomes with the risks of complications, especially bleeding.

First Nations peoples

- Ensure information about transfers or invasive management is provided with the assistance of First Nations health practitioners or Aboriginal liaison officers.
- Communicate in the person's preferred language when required to enhance understanding and informed decision-making.
- Recognise that First Nations peoples from regional areas are less likely to receive angiography compared to non-Indigenous counterparts.[341]
- Be vigilant of barriers to equitable care, including:
    - inadequate cultural competency among healthcare providers
    - perceptions about medicine compliance
    - delayed transfers to PCI-capable hospitals
    - insufficient family and community engagement by clinicians.[51]

Antiplatelet therapy in the acute phase

Recommendations	Strength of recommendation	Certainty of evidence
In people with STEMI treated with fibrinolytic therapy, give dual antiplatelet therapy with aspirin and clopidogrel.	Strong	Moderate
In people with STEMI undergoing primary PCI and people with NSTEACS undergoing a routine invasive strategy, give dual antiplatelet therapy with aspirin and a potent $P2Y_{12}$ inhibitor (ticagrelor or prasugrel).	Strong	High
In people with STEMI undergoing primary PCI and people with NSTEACS undergoing a routine invasive strategy for whom ticagrelor or prasugrel are contraindicated, and those receiving oral anticoagulation, give clopidogrel.	Strong	High
In people with NSTEACS for whom a selective invasive strategy is planned, give ticagrelor or clopidogrel.	Strong	High
In people with NSTEACS, consider routine genotypic or platelet function guidance of $P2Y_{12}$ therapy.	Weak	Moderate
In people with NSTEACS, consider de-escalation from potent $P2Y_{12}$ inhibitor to clopidogrel 30 days following an ACS event.	Weak	Moderate
In people with ACS with concomitant non-valvular atrial fibrillation and CHA2DS2VA score >1, give aspirin and clopidogrel together as well as a non-vitamin K oral anticoagulant.	Strong	High
In people with STEMI undergoing primary PCI or those with NSTEACS undergoing an invasive strategy, routine glycoprotein IIa/IIIb inhibitor (GPI) is not recommended.	Consensus	

Evidence supporting the recommendations

Robust evidence supports the early use of antiplatelet therapy in ACS. Aspirin has proven benefits in reducing serious vascular events (vascular death, MI and stroke) in STEMI when used alone or in combination with fibrinolysis.[182, 342, 343] In people with STEMI treated with fibrinolysis, DAPT with aspirin and clopidogrel has been shown to reduce death, reinfarction and stroke when compared with aspirin alone.[344, 345] In those undergoing primary PCI, potent $P2Y_{12}$ inhibitors (ticagrelor or prasugrel) are preferred over clopidogrel, due to their more rapid onset and superior efficacy.[346, 347] People initially thrombolysed and given clopidogrel then transferred to another centre for PCI may safely be switched to ticagrelor following PCI. For NSTEACS, ticagrelor or prasugrel is recommended when a routine invasive strategy is planned, although clopidogrel remains effective in those for whom ticagrelor or prasugrel are contraindicated or who are receiving oral anticoagulation (OAC).[348, 349]

People with NSTEACS can defer $P2Y_{12}$ inhibitor loading until after coronary angiography, provided that angiography is performed within recommended timelines (Section Further diagnostic testing for people with suspected ACS).[350, 351] In STEMI undergoing primary PCI, pretreatment with a $P2Y_{12}$ inhibitor may be considered if the working diagnosis is certain, but if pretreatment is not given, all people should receive a $P2Y_{12}$ inhibitor loading dose at the time of PCI (see Supplementary material B3).[352] Genetic or platelet function guidance to tailor $P2Y_{12}$ therapy has not consistently demonstrated net clinical benefit, but ongoing studies may clarify its role.[353–357]

Evidence supports de-escalation from potent $P2Y_{12}$ inhibitors to clopidogrel one month post-ACS to reduce bleeding risk, without clear evidence of increased ischaemic events.[358–361] For people requiring concomitant OAC, particularly with non-valvular atrial fibrillation, initial short-term triple therapy (aspirin, clopidogrel and an OAC) followed by dual therapy (OAC plus clopidogrel) effectively reduces bleeding risk.[362–364]

The recommended discontinuation intervals prior to non-emergency cardiac surgery for ACS are five days for clopidogrel, three days for ticagrelor and seven days for prasugrel.[365]

Finally, routine IV GPI inhibitor use is not recommended in primary PCI or in routine invasive strategies for NSTEACS, although bailout use may be considered in select high-thrombus-burden circumstances.[366–372]

Further evidence to support the recommendations is provided in the Comprehensive Guideline, and further details on switching strategies, loading protocols and timing of administration are provided in the and the Supplementary material B3.

Practice points

- **Aspirin sensitivity:** In the event of aspirin sensitivity, risk assessment and consideration of desensitisation should be made using a standardised protocol to achieve adequate antithrombotic therapy.[373]
- **Selection of platelet $P2Y_{12}$ inhibitor therapy:** Prasugrel has Therapeutic Goods Administration (TGA) approval but is not currently available in Australia. Exercise care regarding timing and dosing of $P2Y_{12}$ inhibitors when switching between these agents to ensure their effectiveness is maintained and the bleeding risk minimised. For guidance on switching strategies, (see Supplementary material B3).
- **Timing of platelet $P2Y_{12}$ inhibitor administration in STEMI:** Deferring the administration of the $P2Y_{12}$ inhibitor until after the coronary anatomy is known is reasonable when the diagnosis of STEMI is uncertain or if there is a clinical suspicion of need for urgent cardiothoracic surgery (e.g. left main ischaemia pattern on ECG).
- **Timing of platelet $P2Y_{12}$ inhibitor initiation in NSTEACS:** Decisions regarding timing of initiation of $P2Y_{12}$ inhibitor in relation to invasive angiography may be institution-dependent and need to be clearly defined and communicated effectively between emergency and inpatient services.
- **Combining $P2Y_{12}$ inhibition with anticoagulation:** In people with ACS with an indication for vitamin K antagonist (e.g. mechanical heart valve), use aspirin with clopidogrel rather than ticagrelor or prasugrel to reduce the risk of bleeding. Target international normalised ratios (INRs) should be at the lower therapeutic range (e.g. 2.5–3 for mechanical mitral valves).
- **IV GPI administration:** Bailout GPI may be considered in people at high ischaemic risk such as high thrombus burden, no-flow or slow-flow.
- **Discontinuing platelet $P2Y_{12}$ inhibitor prior to CABG:** In people with NSTEACS with planned CABG, do not administer $P2Y_{12}$ inhibitor within three days of surgery for ticagrelor, five days for clopidogrel or seven days for prasugrel.
- **Selection of GPI therapy:** Tirofiban is the only GPI marketed in Australia, while eptifibatide and abciximab can be obtained through the TGA's Special Access Scheme.
- **Discontinuing IV GPI in thrombocytopaenia:** Glycoprotein IIb/IIIa inhibition is not recommended in people with thrombocytopaenia (platelet count <150,000/mL) and should be suspended immediately if platelet count falls below this level or drops by 50% or more from baseline.
- **Discontinuing IV GPI prior to CABG:** In people undergoing CABG, discontinuation of short-acting GPI (eptifibatide and tirofiban) for four hours and abciximab for 12 hours before surgery is recommended to reduce the risk of bleeding and transfusion.[374–376]

Anticoagulant therapy in the acute phase

Recommendations	Strength of recommendation	Certainty of evidence
People treated with fibrinolytic therapy should receive anticoagulation (unfractionated heparin or enoxaparin).	Strong	Moderate
People undergoing primary PCI should receive anticoagulation (unfractionated heparin or bivalirudin).	Strong	Moderate
People with NSTEACS should receive anticoagulation (unfractionated heparin, enoxaparin or fondaparinux).	Strong	Low

Evidence supporting the recommendations

Anticoagulation is recommended for people with ACS, whether managed with fibrinolytic therapy, primary PCI or a NSTEACS strategy. In fibrinolysis, the GUSTO trial showed the lowest mortality among people with STEMI who received tPA and IV heparin.[377] ASSENT-3 demonstrated fewer ischaemic events with tenecteplase plus enoxaparin (30 mg IV bolus followed by 1 mg/kg subcutaneously twice daily) compared with IV heparin.[378]

For primary PCI, early studies found bivalirudin had similar efficacy but lower bleeding risk than unfractionated heparin (UFH) when GPI inhibitors were used routinely.[273, 379, 380] More recently, the BRIGHT-4 trial (93% radial access; bailout, not routine, GPI) reported that bivalirudin significantly reduced mortality and major bleeding compared with UFH (0.7 units/kg).[381] Bivalirudin can therefore be considered instead of UFH in people undergoing primary PCI for STEMI, factoring in differences in cost and experience with administration. Bivalirudin should be used instead of UFH in people with heparin-induced thrombocytopaenia.

In NSTEACS, early trials showed that UFH reduces MACE without increasing bleeding,[349–351, 382–384] forming the basis for anticoagulation in higher-risk ACS. Although low molecular weight heparin (LMWH) on background therapy with aspirin can also reduce MACE,[385] a larger trial showed there was no difference in the ischaemic endpoint of death and MI but significantly increased bleeding (commonly related to femoral access) with LMWH in settings of peoples on DAPT, early angiography and frequent GPI use.[386] Contemporary meta-analyses suggest no ischaemic advantage of bivalirudin over UFH, especially with radial access.[387] Finally, fondaparinux halves major bleeding compared with LMWH in people on DAPT without compromising efficacy (there were high rates of angiography in these trials).[385, 388]

Practice points

Anticoagulant treatment with fibrinolytic therapy

- Omit IV bolus of enoxaparin in people >75 years receiving fibrinolysis and enoxaparin.
- Enoxaparin is recommended over UFH unless there is severe kidney impairment (eGFR <30 ml/min).[389, 390]

Anticoagulant therapy with primary PCI

- In people requiring PCI with a history of heparin-induced thrombocytopaenia, consider bivalirudin as an alternative to UFH. Outcomes with bivalirudin are optimised when followed by a high dose post-PCI infusion (1.75 mg/kg/hr) for 2–4 hours.[391]

Anticoagulant therapy in NSTEACS

- In people treated with fondaparinux undergoing coronary angiography and/or PCI, standard dose UFH is recommended at the time of the procedure to reduce the risk of guiding-catheter thrombosis.[392, 393]
- In people receiving LMWH in whom femoral access for coronary angiography is planned, it is common practice to omit the morning dose of enoxaparin to minimise access-related bleeding complications.[394]
- Parenteral anticoagulants can be ceased following PCI. In people who do not undergo PCI, UFH may be ceased at 48 hours and fondaparinux or LMWH at six days following presentation in the absence of other indications.[386, 392]

Anticoagulant use in people already receiving warfarin or direct oral anticoagulants

In people with continued indications for oral anticoagulants (atrial fibrillation and CHA2DS2VA score >1, mechanical heart valves or recurrent venous thromboembolism), do not cease this treatment.

In people with NSTEACS undergoing invasive management, wherever possible a brief washout period from the effects of oral anticoagulants (OACs) is desirable. This is to reduce the risk of potential bleeding complications among those who may require femoral access or resulting from additional anticoagulation during the procedure. The suggest washout period is 24 hours for people on direct oral anticoagulants (DOACs) with normal renal function and 48 hours for those with impaired renal function. For people on warfarin, an INR of <2.0 is recommended when using the radial approach and <1.5 when using the femoral approach.

- There are no randomised studies evaluating strategies for early anticoagulation in people with ACS who are already taking warfarin or DOACs. Guidance for these people is derived from expert opinion.[395]

Acute management of ACS with cardiac arrest and/or cardiogenic shock

ACS with cardiac arrest

Recommendations	Strength of recommendation	Certainty of evidence
In people with spontaneous return of circulation after resuscitated cardiac arrest and persistent STE on ECG, perform emergency reperfusion.	Strong	Low
In haemodynamically stable people with resuscitated cardiac arrest and no STE on ECG, do not perform routine emergency coronary angiography.	Strong	Moderate

Evidence supporting the recommendations

Cardiac arrest is a common early cause of death in the context of STEMI, often occurring out of hospital.[396] For people with resuscitated cardiac arrest and ECG-confirmed STEMI, primary PCI significantly improves survival.[397–399] In people without STE on ECG, data show no survival or neurological advantage of early or immediate angiography compared to delayed strategies.[400] However, as these trials excluded individuals with cardiogenic shock, emergency angiography may be appropriate in cases of haemodynamic instability.

Practice points

- In people with STEMI and resuscitated cardiac arrest, primary PCI is the preferred reperfusion strategy. Fibrinolysis may be considered if primary PCI is unavailable. However, evidence is lacking with potential for harm in cardiac arrest that is refractory, prolonged and/or traumatic.[249, 257, 401]
- In people with STEMI and resuscitated cardiac arrest, the decision for primary PCI should factor in treatment futility. For instance, advanced age, presence of severe metabolic acidosis and/or no return of spontaneous circulation for an extended period are associated with a low likelihood of meaningful long-term survival.[402]

ACS with cardiogenic shock

Recommendations	Strength of recommendation	Certainty of evidence
In people with ACS and cardiogenic shock, perform PCI of the IRA only.	Strong	Moderate
In people with ACS and cardiogenic shock, routine insertion of an intra-aortic balloon pump is not recommended.	Strong	High
In people with ACS and cardiogenic shock, routine venoarterial extracorporeal membrane oxygenation is not recommended.	Strong	Moderate
In select people with STEMI and cardiogenic shock, consider left ventricular assist devices.	Weak	Moderate

Evidence supporting the recommendations

Treatment of coronary microvascular disease in ACS with cardiogenic shock

In NSTEACS with coronary microvascular disease (MVD) and cardiogenic shock, culprit-lesion-only PCI reduced the composite of death or renal replacement therapy compared to multivessel PCI, driven by lower mortality.[403] For STEMI with cardiogenic shock, non-culprit lesion PCI during the initial procedure increased death and renal failure risk. Therefore, in the presence of cardiogenic shock, PCI of non-IRAs should not be performed at the time of the index procedure; staged PCI is recommended for complete revascularisation.[403–405]

Haemodynamic support devices in MI and cardiogenic shock

Routine intra-aortic balloon pump use in MI with cardiogenic shock increases bleeding without survival benefit.[406–408] Early venoarterial extracorporeal membrane oxygenation (VA-ECMO) showed no mortality benefit but increased major bleeding and peripheral vascular complications.[409, 410] Percutaneous left ventricular assist devices reduced mortality but increased bleeding, vascular complications and haemodynamic shock in people with severe left ventricular impairment.[411]

Practice points

- Consider intra-aortic balloon pump in select cases – for example, where there are mechanical complications (ventricular septal rupture, mitral regurgitation or free ventricular wall rupture) – and/or as bridging to heart transplant or left ventricular assist devices.
- Consider mechanical support including VA-ECMO on a case-by-case basis as rescue or bridging therapy. Such support can also be considered for treatment of intractable ventricular tachyarrhythmias, in consultation with a multidisciplinary team.
- Consider left ventricular assist devices in people with STEMI and cardiogenic shock on a case-by-case basis, given the selected population enrolled and the complication rate in the DanGer Shock trial.
- In people with ACOMI and cardiogenic shock, where PCI is unavailable, consider fibrinolysis with a plan for subsequent angiography (see recommendations in Section Ongoing management of fibrinolytic-treated people).[412]

Treatment for ACS with multivessel disease without cardiogenic shock

Recommendations	Strength of recommendation	Certainty of evidence
In haemodynamically stable people with STEMI and MVD, perform PCI of suitable non-IRA(s).	Strong	High
Consider performing PCI of the non-IRA at the time of primary PCI or within 19 days of the index procedure.	Weak	Moderate
In people with STEMI and MVD, routine invasive physiology assessment (e.g. fractional flow reserve (FFR)) to evaluate non-IRA severity is not recommended.	Consensus	
In people with NSTEACS and non-complex MVD, consider routine PCI of non-IRA in the same setting.	Weak	Low
In people with NSTEACS and MVD, consider invasive physiology assessment (e.g. FFR) to evaluate non-IRA severity.	Weak	Low

Evidence supporting the recommendations

Treatment of MVD in STEMI

Complete revascularisation in STEMI, MVD and without cardiogenic shock reduces cardiac death, MI and repeat revascularisation compared to IRA-only PCI. Immediate revascularisation at the index procedure is superior to outpatient-staged PCI but its advantage over inpatient-staged PCI remains unclear. CABG may be preferred for complex MVD cases (Section Coronary artery bypass graft surgery in ACS).[282, 413–426]

Treatment of MVD in NSTEACS

No trials specifically compare complete versus IRA-only PCI in NSTEACS. A meta-analysis of observational studies suggests higher short-term risk but improved long-term outcomes with complete revascularisation.[427]

Invasive physiology to evaluate the non-IRA in STEMI or NSTEACS and MVD

In STEMI with MVD, angiography-guided PCI is effective and may outperform physiology-guided approaches for non-IRA lesions.[336, 428, 429] In NSTEACS, physiology-guided PCI may reduce unnecessary revascularisation but outcomes are inconsistent.[430, 431] Among older adults, physiology-guided PCI improves outcomes with no difference in safety outcomes.[432]

For guidance on management of MVD in people with ACS, refer to Figure 14.

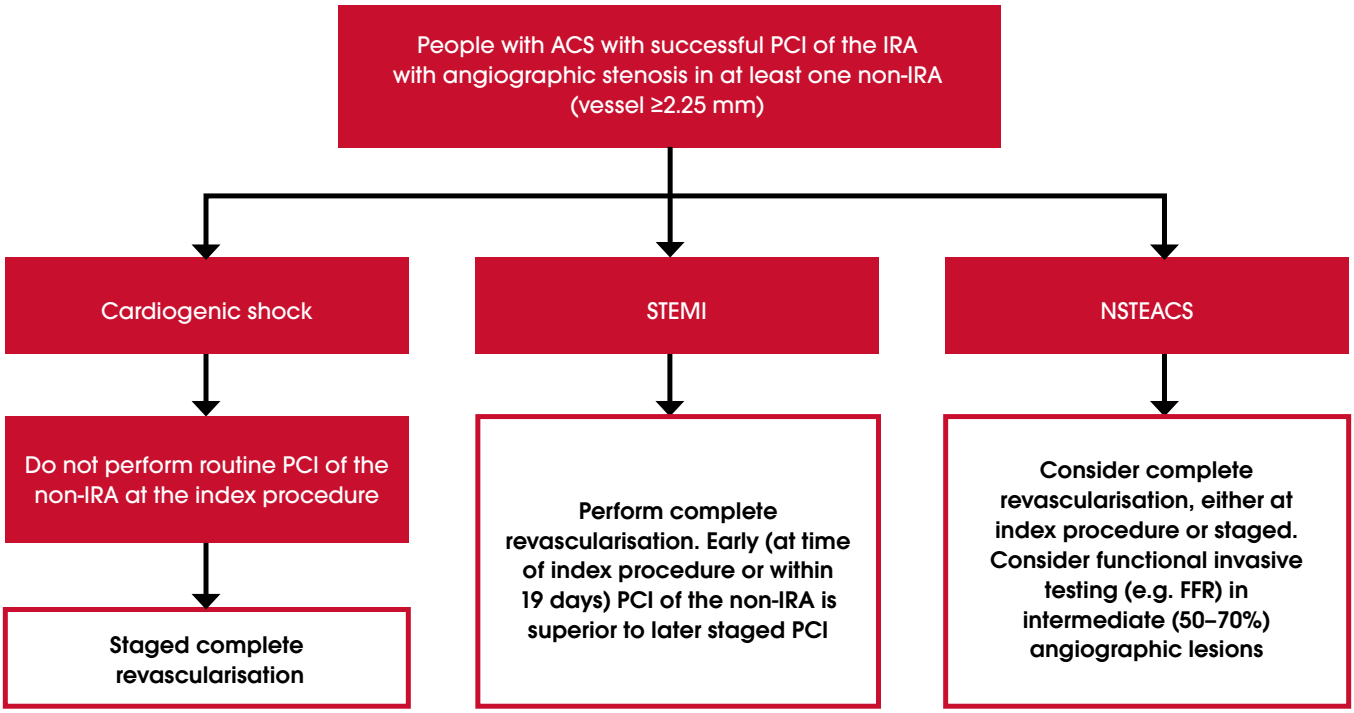


Figure 14 Management of multivessel disease in people with ACS.

Abbreviations: ACS, acute coronary syndromes; FFR, fractional flow reserve; IRA, infarct-related artery; NSTEACS, non-ST-segment elevation acute coronary syndromes; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction.

Practice points

Treatment of non-IRAs

- In people with STEMI and MVD, with unknown renal function, inpatient PCI as a staged rather than immediate procedure may be preferable if complex MVD is present or operator fatigue precludes same setting multivessel PCI.
- In people with NSTEMI and MVD, timing for complete revascularisation should consider factors such as the presence of cardiogenic shock, lesion complexity and risk of contrast nephropathy.
- While a benefit of FFR-guided over angiography-guided complete revascularisation has not been conclusively shown, it is reasonable to use FFR in intermediate (50–69%) non-infarct-related stenoses.[428]
- In people with ACS and complex MVD, a multidisciplinary heart team approach to the revascularisation strategy is recommended. Management of people with ACS and complex MVD should be guided by multidisciplinary heart team discussions incorporating person-based (e.g. age, frailty, infarct size, personal preference) and lesion-based (e.g. location, severity and complexity) factors.

Coronary artery bypass graft surgery in ACS

Recommendations	Strength of recommendation	Certainty of evidence
In people with STEMI, mechanical complications and mitral valve disease (e.g. ventricular septal rupture, mitral valve insufficiency because of papillary muscle infarction or rupture, or free wall rupture), perform CABG at the time of surgery.	Strong	Low

Evidence supporting the recommendations

Perioperative mortality after mechanical complications of STEMI remains high.[433] Few percutaneous or medical treatments are available, and urgent surgery is often the best option. A haemodynamically unstable person may require interim mechanical circulatory support. Performing CABG at the time of surgery for a mechanical complication of STEMI is based on small retrospective series with no randomised trial data.[434]

Practice points

- In people with STEMI where PCI cannot be performed, consider emergency CABG if there is ongoing ischaemia and a large area of jeopardised myocardium.
- Overall, 4–10% of people with NSTEACS will require CABG.[435] When deciding between PCI and CABG, consider comorbidities, fitness for major surgery and coronary anatomy. A multidisciplinary team should be involved in decision-making.
- In people with ACS and MVD where CABG has been chosen as the complete revascularisation strategy, performing CABG at day 1 to day 7 (compared to day 0 or >7 days) after diagnosis has lowest risk of mortality.[436]
- In people with ongoing ischaemia or haemodynamic instability with an indication for CABG, do not delay urgent surgery due to antiplatelet exposure.

Treatment for SCAD

Recommendations	Strength of recommendation	Certainty of evidence
In people with ACOMI due to SCAD but who are otherwise stable, routine revascularisation is not recommended.	Consensus	
In people with SCAD and haemodynamic instability and/or ongoing ischaemia, consider selective revascularisation.	Weak	Very low

Evidence supporting the recommendations

As there are no randomised controlled trials (RCTs) to guide therapy, recommendations in SCAD are based on observational studies or expert opinion.[437] Intervention is challenging, and routine revascularisation is not recommended as it has been associated with several complications. These include iatrogenic dissection, wiring of the false lumen, propagation of the intramural haematoma, acute vessel closure and stent or graft failure.[438–440] However, in a subgroup of people with SCAD who have significant ongoing ischaemia and haemodynamic compromise, urgent revascularisation with PCI or CABG may be required.[168, 441–443]

Myocardial infarction with non-obstructive coronary arteries

In people with myocardial infarction with non-obstructive coronary arteries (MINOCA), it is important to exclude alternative diagnoses.[444] Consider cardiac MRI in all people with MINOCA where the underlying cause is not obvious. Once the underlying cause has been established, manage people with MINOCA according to relevant disease-specific guidelines.[2] In all people with evidence of coronary atherosclerotic disease and/or risk factors, consider initiating secondary prevention measures (even if the underlying cause of MINOCA cannot be determined).[445]

MI due to oxygen supply/demand mismatch without acute coronary occlusion

No trials have examined the benefits of a routine invasive strategy in people with MI due to oxygen supply/demand mismatch without acute coronary occlusion.[446] Whether competing risks from non-cardiac conditions obscure the benefits of invasive management – and at what level of competing risk this occurs – remains uncertain. All available evidence demonstrates that people with MI due to oxygen supply/demand mismatch without acute coronary occlusion experience higher all-cause mortality than people with MI with acute coronary occlusion. This is, in part, related to associated non-coronary competing risks.[446]

In the absence of any trial evidence, angiography with a view to revascularisation may be considered if there is ongoing ischaemia or haemodynamic compromise despite adequate treatment of the underlying acute stressors that provoked the MI due to oxygen supply/demand mismatch without acute coronary occlusion (Section Administration of fibrinolytic therapy and Table 9).

Echocardiography

Left ventricular (LV) dysfunction is an important determinant of prognosis following ACS, and its detection should guide further evidence-based therapies.[447] Echocardiography to evaluate regional and global LV function, and to identify other cardiac pathology, should be performed during hospitalisation. If echocardiography is not possible, consider other aspects suggestive of LV dysfunction, including clinical signs/symptoms, and ECG, chest X-ray and biomarker features.[447]

Duration of cardiac monitoring

Cardiac monitoring plays a pivotal role as an adjunct therapy in the management of ACS. Continuous cardiac monitoring has become a firmly embedded standard of practice despite the absence of evidence from RCTs.[448] Clinical assessment for the risk of life-threatening arrhythmias should be individualised based on known associated risk factors: arrhythmias, ongoing symptoms, reduced LV function (LV ejection fraction LVEF <40%), failed coronary reperfusion, haemodynamic instability and complications of PCI (side branch occlusion, unsealed dissection, embolisation).

Practice points

- In people with ACS, initiate cardiac monitoring immediately, with ST-segment ischaemia monitoring where available. Continue uninterrupted for a minimum of 24 hours.
- People with ACS post-PCI should be monitored, with ST-segment ischaemia monitoring where available, continuously and uninterrupted for 24 hours.
- Re-evaluate the need for continuous ECG monitoring every 24 hours.
- Educate staff regarding proper skin preparation, assessment of skin turgor and ECG electrode replacement every 24 hours, as this reduces inappropriate alarms.[449, 450]
- Further guidance regarding cardiac monitoring can be found on the Agency for Clinical Innovation website (https://aci.health.nsw.gov.au/networks/cardiac/resources/cardiac-monitoring).[448]

3. Recovery and secondary prevention

Following ACS, participation in exercise-based cardiac rehabilitation and person-centred, secondary prevention programs (collectively termed cardiovascular risk management programs) is essential to helping reduce future vascular events and improve quality of life and prognosis.[451] These programs support earlier return to usual activities, including work.

All people with ACS benefit from these programs, including women, older adults, regional and remote residents, First Nations peoples, and people from culturally and linguistically diverse backgrounds.[50, 451] These programs complement the care of general practitioners and other allied health professionals by:

- supporting post-ACS recovery and adopting healthy behaviours (e.g. quitting smoking and/or drug and alcohol use, being physically active, eating healthily and maintaining good mental health)
- providing intensive clinical risk factor education and modification (e.g. managing blood pressure, lowering blood lipids, optimising diabetes management)
- ensuring medication adherence and prescription refills, and facilitating review for actual and potential medicine-related harm, when suspected
- educating people on appropriate management of new or ongoing symptoms post-discharge, including use of anti-anginal medicines and when to seek urgent medical attention
- taking actions to protect against influenza and other pathogens, exposure to climate extremes, severe air pollution and cardiac toxins where applicable
- empowering people and their carers/support people towards greater self-care and management of their underlying cardiac status and comorbidities.

A system-generated referral to a flexible, tailored risk management program should be made before hospital discharge. It is also vital to schedule a post-discharge review with a member of the treating team (e.g. cardiologist, specialist nurse) to address immediate needs such as medicines adherence, wound care and mental wellbeing.

Person-centred non-pharmacological secondary prevention

Recommendations	Strength of recommendation	Certainty of evidence
For all people with ACS, refer to a multidisciplinary exercise-based cardiac rehabilitation program prior to discharge.	Strong	Moderate
For all people with ACS, provide advice on lifestyle* changes such as healthy eating, regular physical activity, not smoking, limiting alcohol intake and caring for mental health.	Consensus	
For all people with ACS who smoke, advise to stop and refer for behavioural intervention (such as cognitive behaviour therapy or cessation counselling program), combined with pharmacotherapy where appropriate (nicotine replacement therapies, varenicline and bupropion individually or in combination).	Strong	Moderate
For all people with ACS, implement strategies to optimise adherence to preventative medicines.	Consensus	

*Use of the word lifestyle here refers to a collective group of modifiable risk factors. The authors wish to acknowledge that these risk factors are not solely dependent on individual choice, and instead reflect the cultural, social and environmental factors that influence behaviour. This term does not in any way attribute blame to individuals.

Evidence supporting the recommendations

Exercise-based cardiac rehabilitation can reduce the risk of further MI and all-cause hospital admissions in people post-MI or revascularisation.[451] Exercise-induced cardiac events are negligible in comparison to the risk associated with being habitually sedentary. Similarly, smoking cessation is strongly associated with a lower risk of future MI and death.[452–455]

Suboptimal adherence to prescribed medicines following ACS is linked to increased rehospitalisation and mortality.[456] One study found 45% of people with ACS were not taking lipid-lowering medicines 12 months after discharge.[457] Another audit found only 65% of people were discharged on recommended medicines (antiplatelets, lipid-lowering agents, beta blockers and angiotensin-converting enzyme ACE inhibitors).[458] Starting these treatments in hospital and providing clear medicines education are critical to improve adherence.[459]

Practice points

Cardiac rehabilitation and secondary prevention programs

- Cardiac rehabilitation and secondary prevention programs should offer evidence-based aerobic and resistance training in accordance with the current Cardiac Society of Australia and New Zealand Position Statement.[460] Where an exercise-based cardiac rehabilitation program is not available, refer to a flexible, cardiovascular risk management program.
- Evidence-based cardiac rehabilitation programs should be tailored, where possible, to meet the unique needs of groups with low attendance rates, including women and people from culturally and linguistically diverse communities.[461, 462]
  - For First Nations peoples, enable access to cardiac rehabilitation programs facilitated by Aboriginal and Torres Strait Islander health practitioners wherever possible.
  - For people whose first language is not English, enable access to bilingual educators wherever possible.
- For people with ACS living in regional and remote communities, cardiac rehabilitation via telehealth is an acceptable alternative to in-person programs.[463] The Cardiac Services Directory (cardiacserviceslist.heartfoundation.org.au) on the Heart Foundation website lists cardiac rehabilitation programs available throughout Australia, including those delivered via telehealth.
- Embed system-generated referral to a cardiac rehabilitation/risk management program based on a person's preference, values and the available resources.[464–469]
- Consider use of digital health interventions in the delivery of cardiovascular risk management programs post-ACS such as reminders, text messaging, mobile health (mHealth) apps, telehealth consultations, wearable devices and electronic decision-support tools.[467, 470, 471]

Lifestyle management

- Provide relevant disease and lifestyle education; the latter covering healthy eating, regular physical activity, not smoking, limiting alcohol intake and caring for mental health. Refer to the Heart Foundation website for guidance and resources on these topics.[472]
- Screen people with ACS for depression and other mental health conditions using validated tools and refer for appropriate mental health support as required. People with ACS commonly experience feelings of low mood, sadness, guilt, worry and anger.[473, 474]

Medicines adherence

- Provide effective medicines education at discharge. This should include discussion of:
  - what each medicine is for, the strength and dose, and when and how to take it
  - the importance of continuing to take medicines as prescribed, and not stopping or changing the dose unless advised by their general practitioner
  - what to do if they miss a dose
  - potential side effects and what to do if they believe they are experiencing a side effect.
- Implement practical strategies to promote adherence, such as daily alerts/reminders, combining medicines where possible (fixed combination medicines) and pharmacy-provided medicine packs.

- Consider post-discharge comprehensive medicine review, particularly in those with significant medicine changes, polypharmacy and/or multimorbidity, those on high-risk medicines such as anticoagulants, and those at risk of medicine non-adherence.[475]

Follow-up care

- Provide a verbal and written discharge summary that includes:
  - the diagnosis and treatment, as well as investigation findings
  - scheduled follow-up appointments (post-discharge review with the treating team, general practitioner, cardiac rehabilitation)
  - medicines commenced, altered or ceased while in hospital, and the importance of taking medicines as prescribed
  - healthy lifestyle changes and practical strategies to implement these
  - a chest pain/angina management plan.
- Ensure chest pain/angina management plans include guidance on management of new or ongoing symptoms post-discharge, including use of anti-anginal medicines, and when and how to seek urgent medical attention (i.e. calling Triple Zero (000) for an ambulance rather than driving to hospital).
- Two-way communication between the discharging hospital and the person's general practitioner is critical to support their ongoing care. Similarly, encourage people with ACS to establish/maintain regular contact with their general practitioner for ongoing follow-up.
- Initiate a general practitioner management plan or team care arrangement to assist in the management of comorbidities. This is particularly important for older adults with geriatric syndromes including frailty, impaired cognitive function and polypharmacy.[464–469]

Vaccination against influenza and other respiratory pathogens

Recommendations	Strength of recommendation	Certainty of evidence
In people with ACS, vaccinations for influenza and other respiratory pathogens are recommended.	Consensus	

Evidence supporting the recommendations

Vaccination against influenza can reduce the risk of further cardiac complications in people with ACS or cardiovascular disease.[476, 477] The *Australian immunisation handbook* outlines recommendations for people with CAD and other chronic cardiac conditions regarding respiratory syncytial virus (RSV), influenza, pneumococcal disease and COVID-19 vaccination.[478]

Practice points

- People with CAD should receive influenza and pneumococcal vaccinations as per recommended schedules.[478] The influenza vaccine can be safely administered within 72 hours of hospitalisation for AMI, including for an invasive coronary procedure.[476]
- People with CAD aged ≥60 years should receive RSV vaccination due to their increased risk of severe RSV disease.[478]
- People with chronic cardiac conditions, including coronary heart disease, are at increased risk of severe COVID-19 and may benefit from additional doses of COVID-19 vaccine.

Post-ACS pharmacotherapy

Antiplatelet therapy

Recommendations	Strength of recommendation	Certainty of evidence
In people discharged following an ACS who are at high ischaemic and/or low bleeding risk, prescribe DAPT with aspirin and a $P2Y_{12}$ inhibitor for 6–12 months.	Strong	High
In people discharged following an ACS who are at low ischaemic and/or high bleeding risk, cease DAPT at 1–3 months post-ACS and continue single antiplatelet therapy (SAPT).	Strong	High
In people discharged following an ACS who have completed a course of DAPT (i.e. 1–12 months), prescribe long-term $P2Y_{12}$ inhibitor over aspirin.	Strong	Moderate
In people discharged following an ACS who remain at high ischaemic and low bleeding risk, consider long-term DAPT (>12 months).	Weak	Moderate
In people discharged following an ACS with an indication for long-term OAC therapy, continue OAC and DAPT (preferentially aspirin and clopidogrel) for 1–4 weeks, then cease aspirin.	Strong	High
In people discharged following an ACS with an indication for long-term OAC therapy, cease antiplatelet therapy at 6–12 months and continue anticoagulation alone.	Strong	Moderate

Evidence supporting the recommendations

Landmark trials of $P2Y_{12}$ inhibitors in people with ACS undergoing PCI established 9–12 months of DAPT as the standard duration.[346, 347] However, recent advancements in stent technology and secondary prevention strategies have resulted in better ischaemic outcomes. More recent evidence suggests that so-called long-duration DAPT might be shortened to around 6 months, particularly for those who have had stents.[479]

It is important to note that most DAPT trials have focused on people undergoing PCI. Those managed with CABG or medical therapy alone are often only included in subgroup analyses, making it less clear how these recommendations apply to non-PCI groups.

Duration of DAPT in people with ACS

In people with ACS undergoing PCI, shorter DAPT (1–3 months vs 6–12 months) may result in significantly fewer bleeding events, with no major differences in MACE.[365] Prolonged DAPT has also been shown to reduce ischaemic events in people with ACS undergoing PCI, although not in people at high bleeding risk.[303]

However, some studies have shown that although prolonged DAPT can reduce the incidence of cardiovascular death, MI, stroke and stent thrombosis, it can increase the risk of bleeding.[480, 481] Latest evidence demonstrates that in people with ACS and those with stable disease undergoing stenting, continuing a $P2Y_{12}$ inhibitor (clopidogrel or ticagrelor) rather than aspirin may offer the best balance by reducing both major bleeding and the risk of MI.[482, 483]

People at high bleeding risk

Several scoring systems, such as the PRECISE-DAPT score and ARC-HBR, help identify people at HBR who are receiving DAPT after PCI.[117, 484–486] In people with HBR, shortening DAPT to 1–3 months may reduce major or clinically relevant bleeding without increasing MACE.[303, 487]

Long-term SAPT

In people undergoing PCI, SAPT ($P2Y_{12}$ inhibitor monotherapy) can reduce bleeding and provide similar protection against MI compared with DAPT (of 1–18 months duration).[488] $P2Y_{12}$ inhibitor monotherapy provides superior protection over aspirin alone, which has been linked to a higher MI risk.[488–490] It should be noted ticagrelor is not currently subsidised on the Pharmaceutical Benefits Scheme (PBS) as monotherapy. Current PBS criteria state ticagrelor must be prescribed in combination with aspirin.

Refer to the Comprehensive Guideline (Figure 15, Table 11a and Table 11b) for guidance on DAPT duration following an ACS.

People with atrial fibrillation requiring long-term anticoagulation

A meta-analysis of four DOAC-based RCTs in people with atrial fibrillation undergoing PCI (>50% ACS) found dual therapy (clopidogrel + DOAC) reduced bleeding compared to triple therapy but increased stent thrombosis rates.[491, 492] A network meta-analysis of five studies supported non-vitamin K OAC + $P2Y_{12}$ inhibitor therapy without aspirin as the safest option compared to regimens including aspirin or vitamin K antagonists.[493]

Higher stent thrombosis rates were observed within the first 30 days with dual therapy, suggesting aspirin may be continued for up to one month for people not at high bleeding risk.[492, 494, 495] Continuing a DOAC beyond one year without SAPT showed no effect on ischaemic or bleeding events in people with HBR, while combining aspirin with a DOAC increased mortality compared to DOAC alone at one year.[395]

It is recommended to stop antiplatelet therapy after 12 months and continue a DOAC for people with ACS requiring long-term OAC. Refer to Comprehensive Guideline Figure 16 for guidance on recommended antiplatelet treatment strategies for people with ACS requiring long-term DOAC for atrial fibrillation.[496, 497]

Practice points

Duration of DAPT in people with ACS

- In older people (≥70 years) with ACS, particularly if HBR, consider clopidogrel as the $P2Y_{12}$ receptor inhibitor.[445]

People at high bleeding risk

- A mobile phone-based application has been developed to assist with decision-making for people at HBR (see http://www.cerc-europe.org/arc-hbr-high-bleeding-risk-evaluator/). This is based on an algorithm that predicts risk of major ischaemic and bleeding events.[498]
- In people receiving DAPT with high risk of gastrointestinal bleeding, a proton pump inhibitor is recommended.

People requiring long-term anticoagulation

- In people with ACS undergoing PCI with other conditions that require long-term anticoagulation (e.g. mechanical heart valve), a lack of evidence prevents recommendations being made.
- The recommendations in this section can be applied to people with ACS and MI undergoing medical management.[364]
- In people who have undergone PCI and are at HBR, de-escalating therapy to anticoagulation alone after 6 months may be reasonable.[395]
- In people receiving triple therapy, a proton pump inhibitor is recommended.

Lipid-modifying therapy

Recommendations	Strength of recommendation	Certainty of evidence
In people with ACS, prior to hospital discharge, initiate and continue indefinitely the highest tolerated dose of HMG-CoA reductase inhibitors (statins), unless contraindicated or completely statin intolerant.	Strong	High
In people with ACS with initial or partial intolerance to statin, consider using a different statin, dose or dosing frequency to achieve person-specific therapeutic objectives.	Weak	Low
In people with ACS, an initial target low-density lipoprotein cholesterol (LDL-C) level of <1.4 mmol/L and a reduction of at least 50% from baseline is recommended, with further benefit gained from treating to the lowest achievable level.	Consensus	
In people with ACS with a suboptimal LDL-C level despite statin therapy or who are statin intolerant, consider adding ezetimibe.	Weak	Moderate
In people with ACS with a suboptimal LDL-C level despite maximally tolerated statin therapy and ezetimibe, give PCSK9 inhibitors.	Strong	High

Evidence supporting the recommendations

Statin therapy substantially reduces adverse cardiovascular outcomes in people with ACS and other vascular diseases. Specifically, lowering LDL-C by 1.0 mmol/L can reduce the risk of MI, stroke, coronary revascularisation and vascular death.[493, 499] This effect may be enhanced by adding ezetimibe to statins in people with ACS.[500] Additionally, monoclonal antibodies to PCSK9 (e.g. alirocumab, evolocumab, inclisiran) can further lower LDL-C levels in people already on intensive statin therapy and improve cardiovascular outcomes.[501, 502]

Practice points

- Initiate or continue high-potency statin therapy (e.g. atorvastatin or rosuvastatin) as early as possible during the ACS admission, irrespective of baseline LDL-C level.[499]
- For people on lipid-lowering therapy prior to index ACS admission, consider intensifying existing lipid-lowering therapy.
- Re-assess total cholesterol and LDL-C levels 4–6 weeks after initiating or intensifying treatment. Adjust statin therapy or add non-statin therapy accordingly. Note that additional non-statin therapies are frequently required to achieve target LDL-C levels and to prevent recurrent coronary events.
- In people with ACS (men <55 years and women <60 years), the Dutch Lipid Clinic Network score can guide the need for diagnostic genetic testing. If genetic predisposition is confirmed, consider cascade testing, genetic counselling and initiating statins in family members.[503]
- In people with ACS with triglyceride levels of 1.5–5.6 mmol/L and LDL-C 1.0–2.6 mmol/L despite statin therapy, consider adding icosapent ethyl.[504] Note that the current PBS eligibility criteria for icosapent ethyl is a triglyceride level of ≥1.7 mmol/L.

Women

- In women at risk of a major vascular event, commence statin therapy.[505]
- Note women are less likely than men to be prescribed statin therapy post-ACS.[506]

Older adults

- In older adults with evidence of occlusive vascular disease (such as prior MI), consider statin therapy to reduce the risk of major vascular events.[507]

Beta blocker therapy

Recommendations	Strength of recommendation	Certainty of evidence
In people with ACS and LV impairment, beta blockers are recommended.	Consensus	
In people with ACS and preserved LV systolic function who have undergone coronary revascularisation and are receiving optimal medical therapy, consider withholding beta blockers.	Weak	Moderate

Evidence supporting the recommendations

Beta blockers can reduce the risk of recurrent MI in people with LV dysfunction.[508–510] However, they produce no reduction in all-cause death or MI in people with preserved ejection fraction undergoing early angiography.[511] Current studies are attempting to address the lack of evidence for people with preserved ejection fraction.[509, 510]

Practice points

- In people with MI and risk factors for cardiogenic shock, exercise caution when initiating beta blockers as these people may be at increased risk of early mortality.[512]
- IV beta blockade in STEMI prior to PCI has not been shown to reduce death or MI at one year.[513, 514]
- In people with confirmed LV dysfunction, consider using a beta blocker of proven benefit in heart failure with reduced ejection fraction (bisoprolol, carvedilol, metoprolol controlled or extended release or nebivolol). See the *Guidelines for the prevention, detection, and management of heart failure in Australia* for further details including other recommended therapies.[447]
- In people with preserved ejection fraction, no benefit in continuing beta blockers beyond 12 months has been seen but more research is being done.[515–518]
- In asymptomatic people discharged following an episode of UA (i.e. without MI) and with normal LVEF, there is little evidence for protection against MACE from beta blocker therapy in the absence of other indications.

Renin-angiotensin antagonist therapies

Recommendations	Strength of recommendation	Certainty of evidence
In people with ACS and heart failure symptoms, LVEF ≤40%, diabetes, hypertension and/or chronic kidney disease, initiate and continue angiotensin-converting enzyme (ACE) inhibitors, or angiotensin receptor blockers if ACE inhibitors are not tolerated.	Strong	High
In people with ACS and LVEF ≤40% and heart failure with or without diabetes, initiate and continue mineralocorticoid receptor antagonists.	Strong	High
In people with ACS use of an angiotensin receptor–neprilysin inhibitor is not recommended.	Strong	High

Evidence supporting the recommendations

Following MI, ACE inhibitors can reduce early mortality, cardiovascular events, non-fatal MI and stroke.[388, 519, 520] Angiotensin receptor blockers exert comparable effects to ACE inhibitors in people with reduced LVEF after MI, but no benefit is seen with the use of angiotensin receptor–neprilysin inhibitors.[521–524]

Practice points

- In people with ACS and LVEF ≥40% or without clinical heart failure, consider use of ACE inhibitors (or angiotensin receptor blockers if ACE inhibitors not tolerated) to improve survival.[520]
- For people with ACS and concurrent hypertension, ACE inhibitors and angiotensin receptor blockers are indicated as first-line agents for hypertension management. Current blood pressure management and targets are provided in the Heart Foundation's *Guideline for the diagnosis and management of hypertension in adults.*[523]
- In people with ACS and heart failure, ongoing management should align with the *Guidelines for the prevention, detection and management of heart failure in Australia*, with consideration of referral to specialised heart failure services to optimise care and improve outcomes.[447]

Colchicine therapy

Recommendations	Strength of recommendation	Certainty of evidence
In people with ACS, consider initiating colchicine (0.5 mg daily) and continuing long-term unless contraindicated or colchicine intolerant.	Weak	Moderate

Evidence supporting the recommendation

Residual inflammation after ACS may increase the risk of subsequent reinfarction. Some evidence indicates that colchicine can lower the risk of coronary revascularisation and stroke in people with ACS, without significantly affecting all-cause or cardiovascular mortality, or recurrent MI.[525]

However, a subsequent trial of colchicine treatment post-MI showed no benefit to cardiovascular death, recurrent infarction, stroke or unplanned revascularisation over three years. Further meta-analyses are needed to clarify colchicine's role post-ACS.

Semaglutide

Semaglutide, a glucagon-like peptide-1 (GLP-1) receptor agonist, has demonstrated significant cardiovascular benefits in people with obesity. In the SELECT trial, which enrolled adults with established CVD, obesity (or overweight) and without diabetes, once-weekly subcutaneous semaglutide (2.4 mg) reduced the combined incidence of cardiovascular death, nonfatal myocardial infarction, or nonfatal stroke (hazard ratio 0.80; 95% CI 0.72–0.90).[526] These findings highlight the potential of semaglutide as an adjunct treatment to facilitate weight loss and reduce cardiovascular risk, including secondary prevention in people with ACS who meet criteria for overweight or obesity.

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