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GUIDELINE SUMMARY OPEN ACCESS

National Heart Foundation of Australia and Cardiac Society of Australia and New Zealand: Australian Clinical Guideline for Diagnosing and Managing Acute Coronary Syndromes 2025

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ABSTRACT

Introduction: The *Australian clinical guideline for diagnosing and managing acute coronary syndromes 2025* establishes a new clinical standard for the diagnosis and management of acute coronary syndromes (ACS) in Australia. The new guideline replaces the 2016 guideline, representing the first major update in nearly a decade.

Main Recommendations: The new guideline features critical new information, including: (1) new terminology and revised definition of myocardial infarction; (2) electrocardiogram (ECG) patterns of acute coronary occlusion myocardial infarction (ACOMI), beyond ST-segment elevation; (3) use of clinical decision pathways incorporating high-sensitivity cardiac troponin (hs-cTn) assays for more efficient risk assessment; (4) stronger emphasis on the optimal timing of primary percutaneous coronary intervention in people with ST-segment elevation myocardial infarction (STEMI); (5) use of intravascular imaging-guided percutaneous coronary intervention in people with non-ST-segment elevation acute coronary syndromes (NSTEACS); (6) treatment guidance for specific groups, including those with cardiogenic shock, multivessel disease or spontaneous coronary artery dissection; (7) timing of platelet $P2Y_{12}$ inhibitor administration in STEMI and NSTEACS; (8) more detailed advice on post-discharge care, including cardiac rehabilitation and secondary prevention programs, medicine adherence strategies, vaccinations and screening for mental health conditions; (9) treatment algorithms to enable more tailored prescribing of antiplatelet and anticoagulation therapies; (10) new recommended treatment target for low-density lipoprotein cholesterol (LDL-C); and (11) new recommendations on select medicines including PCSK9 inhibitors, β-blockers and angiotensin receptor-neprilysin inhibitors.

Changes in Management as a Result of the Guideline: The new guideline introduces key practice changes including broader recognition of ECG patterns of ACOMI, integration of hs-cTn testing into clinical decisions pathways and selective use of intravascular imaging in NSTEACS. Updated $P2Y_{12}$ inhibitor timing, stricter LDL-C targets and PCSK9 inhibitor use support more tailored and evidence-based care in the secondary prevention of ACS. The full guideline is available at www.heartfoundation.org.au/for-professionals/acs-guideline.

JEL Classification: Cardiovascular diseases, Rehabilitation

1 | Introduction

Acute coronary syndromes (ACS) account for more than half of the coronary heart disease-related hospital admissions annually in Australia [1]. In 2021, 10% of all deaths in Australia were attributed to coronary heart disease, which includes ACS [1]. The economic burden of ACS is significant, costing Australian governments about $1.93 billion in 2017–18 [2].

Marked inequities in both the management and outcomes of ACS persist in Australia. These include women experiencing longer symptom duration, under or delayed diagnosis and treatment, including lower rates of secondary prevention medication use [3–5]; First Nations peoples experiencing coronary events at twice the rate of non-Indigenous Australians, exacerbated by ongoing inequities in healthcare access and outcomes [1, 6, 7]; older adults, having a higher rate of ACS presentations, often complicated by atypical symptoms and age-related comorbidities [8]; and people living in regional and remote areas with higher rates of missed diagnosis and poorer outcomes [9].

The *Australian clinical guideline for diagnosing and managing acute coronary syndromes 2025* (the guideline) [10] replaces the 2016 guideline [11], representing the first major update in nearly a decade. The guideline includes:

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

ACS includes acute myocardial infarction (AMI) and unstable angina. The guideline primarily addresses the management of myocardial infarction caused by atherosclerotic plaque rupture, ulceration, fissure or erosion. Some recommendations may also apply to other myocardial infarction types, including myocardial infarction due to non-atherosclerotic causes such as spontaneous coronary artery dissection (SCAD).

This article provides a summary of recommendations and changes that have been made since the 2016 guideline. A complete version of the guideline is available at www.heartfoundation.org.au/for-professionals/acs-guideline.

2 | Methods

The guideline was developed based on the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) methodology [12]. It was also informed by the 2016 National Health and Medical Research Council (NHMRC) Standards for Guidelines, with minor adaptations to meet the specific requirements of the guideline; for example, engaging one instead of two independent reviewers of the recommendations, and not specifying a future update date, as this will depend on the availability of resources and funding [13].

Guideline development was led by the National Heart Foundation of Australia in collaboration with leading experts and reference group organisations. The governance structure included an expert steering group, three expert subgroups, reference group organisations and a consumer advisory panel. Expertise was sourced across the disciplines of cardiology, emergency medicine, general medicine, general practice, nursing, pharmacy, epidemiology, cardiac rehabilitation and public health.

The expert steering group and expert subgroups determined the guideline scope and prioritised the clinical questions it sought to address, based on gaps identified in international guidelines, literature review, clinical experiences, and preferences and values of people with lived experience. The clinical questions were expressed in the PICOTS (patient/population, intervention, comparison, outcome, time, setting) format. ASERNIP-S (Australian Safety and Efficacy Register of New Interventional Procedures-Surgical), the evidence synthesis division of the Royal Australasian College of Surgeons, reviewed these PICOTS questions and conducted an independent systematic literature review for studies published between January 2015 to December 2022. This article uses these definitions: sex refers to biological attributes; gender to socially constructed roles, behaviours and identities. Evidence and recommendations are reported by sex/gender where such data were available.

The expert groups reviewed the evidence summaries, developed the recommendations and drafted the guideline content. Evidence summaries were supplemented with additional studies identified from conference attendances, database alerts and relevant international guidelines. If pertinent to the recommendations, studies published after the literature search dates up till November 2024 were included. An independent reviewer assessed the comprehensiveness and balance of the scientific evidence, certainty of evidence and rationale to inform the wording and strength of the recommendations.

Based on the GRADE methodology, three classifications of guidance were developed in a hierarchy that reflects the certainty of evidence, importance of the recommendation and the context of its application [12]. GRADE recommendations provide the most robust guidance based on the available evidence; consensus recommendations are informed by expert opinion when there is indirect supporting evidence and the GRADE approach is not applicable; and practice points offer actionable and practical advice in implementing the recommendations and addressing the unique needs of under-served populations.

A public consultation process was conducted over a period of 30 days from 27 September to 28 October 2024. A combination of both open and targeted consultation methods was used to capture feedback on the guideline from key stakeholders and individuals, which was critical in improving its quality, legitimacy and acceptability to end users and the public.

More information on the process for developing the guideline can be found in the supplementary materials available at www.heartfoundation.org.au/for-professionals/acs-guideline.

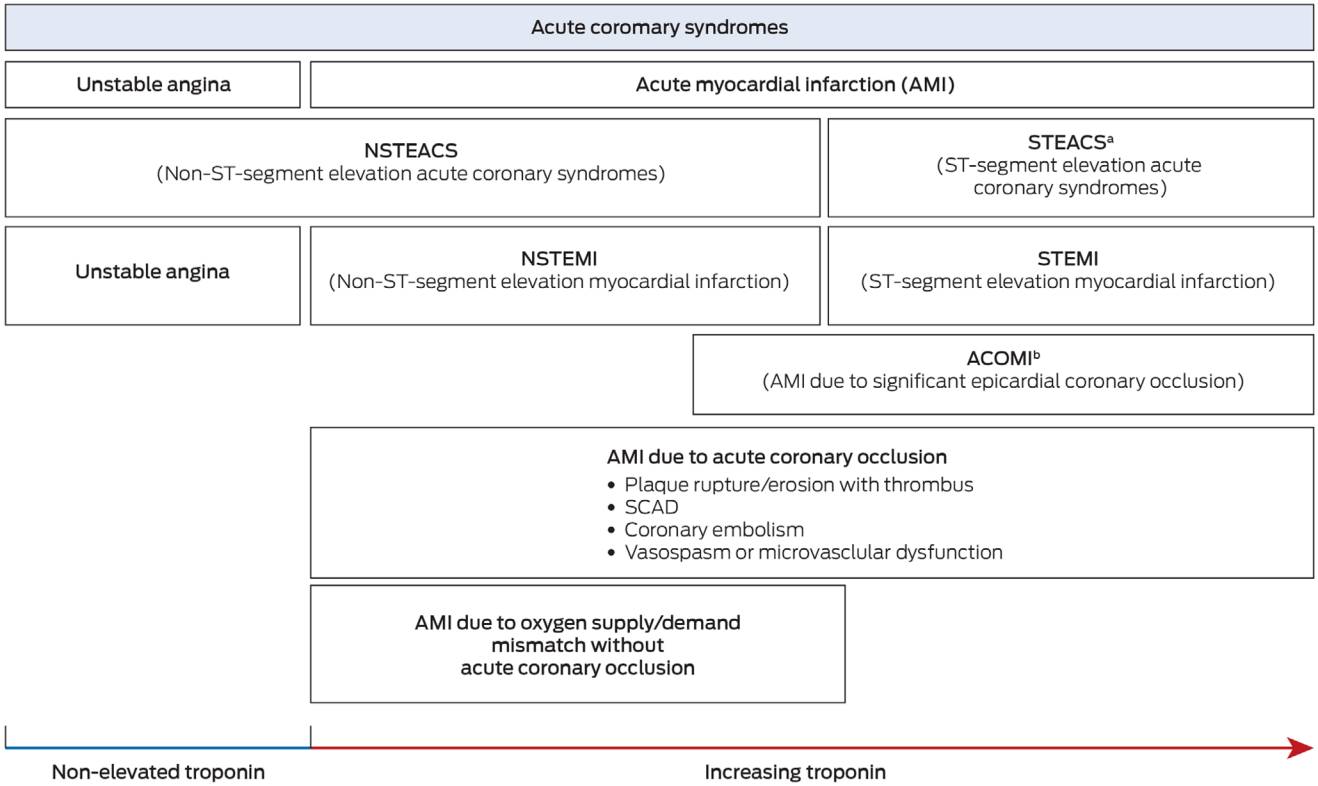


FIGURE 1 | Classifications of conditions associated with acute coronary syndromes. Coronary occlusion describes any local pathological process that reduces flow in coronary vessels (including the microvasculature) sufficient to cause myocardial necrosis (acute troponin elevation). [a]Not all people with ST-segment elevation will have elevated cardiac troponin values, resulting in the diagnosis of unstable angina. [b]The term ACOMI incorporates both STEMI and STEMI equivalents, which should prompt consideration for emergency reperfusion. ACOMI, acute coronary occlusion myocardial infarction; SCAD, spontaneous coronary artery dissection.

3 | New Terminology and Revised Definition of Myocardial Infarction

The guideline adopted the new term 'acute coronary occlusion myocardial infarction' (ACOMI) and revised definitions and classifications of myocardial infarction that align more closely with clinical syndromes that characterise occlusive and non-occlusive forms of myocardial infarction. ACOMI may present as STEMI or STEMI equivalents (Figure 1).

The term ACOMI has been adopted to acknowledge electrocardiogram (ECG) patterns beyond traditional ST-segment elevation (STE) criteria that have been found to reflect acute coronary occlusion without STE, such as posterior myocardial infarction and De Winters T waves (Figure 2) [14–17]. It also includes STE patterns often under-recognised in acute settings such as right ventricular or high lateral infarction (Figure 2) [18].

Coronary occlusion can result from both atherosclerotic and non-atherosclerotic causes, referred to in the 2016 guideline as 'type 1 myocardial infarction' and 'type 2 myocardial infarction', respectively. Conditions such as SCAD, coronary embolism and coronary spasm or microvascular dysfunction can present identically to atherosclerotic causes of AMI and may require urgent angiography to diagnose and treat appropriately. For this reason, these conditions have been classified as myocardial infarction with acute coronary occlusion (Figure 1) [19–21].

4 | Recommendations

The guideline recommendations are presented in three sections: assessment and diagnosis (Tables 1 and 2, Figures 3 and 4), hospital care and reperfusion (Table 3, Figure 5), and recovery and secondary prevention (Table 4, Figures 6 and 7). A summary of key updates is also included for each section.

4.1 | Section 1: Assessment and Diagnosis

Assessment of ACS involves ECG evaluation, clinical history and physical examination, and troponin testing. These results are combined to confirm diagnosis or guide risk assessment, which directs further investigations, treatment and follow-up. Clear communication is essential to risk assessment—language or cultural barriers should be addressed where needed. A summary of the recommendations for assessing and diagnosing people with suspected ACS is presented in Table 1.

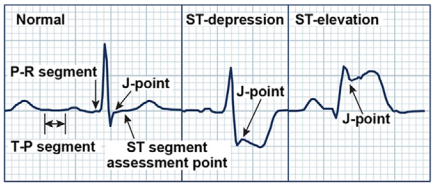
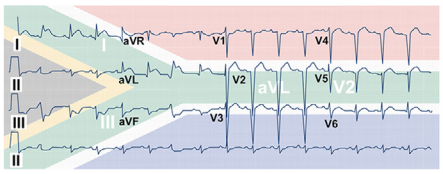
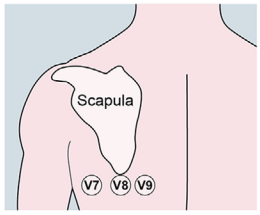
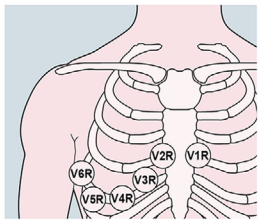
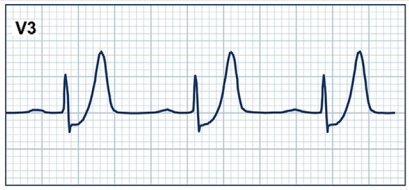
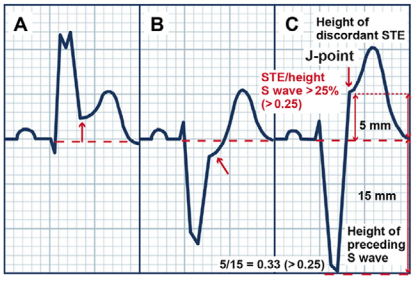
	Criteria	Supporting information and illustration	Recommendation for clinical action
A. Regional STE with reciprocal STD	STE ≥1 mm at the J-point in two contiguous leads in all leads other than V2-4. V2-4 STE criteria: ≥1.5 mm in women ≥2 mm in men ≥40 years ≥2.5 mm in men <40 years		Activate reperfusion pathway
B. High lateral MI	STE I, aVL, V2 STD III (+/- II, aVF) Subtle STE V5, V6 and reciprocal changes in aVF may be seen.		Activate reperfusion pathway
C. Posterior MI	Precordial STD ≥0.5 mm V1-3 Confirm with posterior leads (V7,8,9) with findings of STE: • ≥0.5 mm in women and men ≥40 years • ≥1 mm in men <40 years	V7, 8, 9 supplementary lead placement 	Activate reperfusion pathway
D. Right ventricular MI	STE ≥0.5 mm in any right-sided chest lead (V3R-V6R), but particularly V4R. STE ≥1 mm in men <30 years	Right precordial supplementary lead placement 	Activate reperfusion pathway
E. De Winter T waves	J-point depression with up-sloping ST segments and tall, prominent, symmetric T waves in precordial leads, with STE (≥0.5 mm) in aVR and an absence of STE in precordial leads		Activate reperfusion pathway
F. Modified Sgarbossa criteria **(LBBB or paced rhythm)**	Any of the following: A) Concordant STE >1 mm in leads with positive QRS complex B) Concordant STD ≥1 mm V1-3 C) STE ≥1 mm in one or more leads at the J-point which is proportionally discordant to the preceding S wave by >25%		Activate reperfusion pathway

FIGURE 2 | Electrocardiogram (ECG) findings consistent with acute coronary occlusion myocardial infarction (ACOMI). LBBB, left bundle branch block; MI, myocardial infarction; STD, ST-segment depression; STE, ST-segment elevation.

4.1.1 | What's New in the 2025 Guideline?

4.1.1.1 | Recognising ECG Patterns of ACOMI Beyond Traditional STE Criteria. STE is the key ECG criterion required to institute a reperfusion strategy for people with signs or symptoms of myocardial ischaemia; however, STE is not specific to ACOMI, which may occur in other disease states, both cardiac and non-cardiac [14, 15]. Comparison of ECGs with coronary angiogram results have revealed multiple ECG patterns of ACOMI beyond the traditional STE criteria, including high lateral myocardial infarction, posterior myocardial infarction, right ventricular myocardial infarction, De Winter T waves, modified Sgarbossa criteria and transient STE (Figure 2) [16–18]. Recognition of these patterns should prompt urgent consultation with cardiology services for consideration of reperfusion strategy [23].

TABLE 1 | Summary of recommendations for assessing and diagnosing people with suspected acute coronary syndromes (ACS).

Recommendation	Strength of recommendation	Certainty of evidence
Initial ECG assessment		
In people presenting with chest pain or other symptoms suggestive of ACS, record and assess an ECG for evidence of ACOMI within 10 min of first clinical contact.	Consensus	
In people with suspected ACS, record and assess additional ECGs if symptoms persist, change or recur, or there is diagnostic uncertainty. For those with ongoing ischaemic symptoms and an inconclusive standard 12-lead ECG, record and assess further ECGs with right-sided and/or posterior leads.	Consensus	
In people with ongoing ischaemic symptoms or haemodynamic compromise or new ischaemic findings on ECG, continuous cardiac monitoring and defibrillator availability is recommended while assessment for ACOMI continues.	Strong	Low
Biomarkers		
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	
Risk assessment and clinical decision pathways for suspected ACS		
People with symptoms and ECG changes consistent with ACOMI require urgent reperfusion. Do not use clinical decision pathway (CDP).	Strong	Very low
People presenting with acute chest pain or other symptoms suggestive of ACS should receive care guided by an evidence-based CDP 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- or 0/2-h 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 CDP incorporating formal clinical score-based risk stratification is recommended.	Consensus	
Initial therapeutic management		
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 (SpO_2) ≥ 90% do not require oxygen therapy.	Strong	Moderate
In people with suspected or confirmed ACS receiving oxygen therapy, SpO_2 should not exceed 96%.	Strong	Moderate
In the presence of ongoing chest pain, give glyceryl trinitrate sublingual tablet or spray every 5 min 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	

(Continues)

TABLE 1 | (Continued)

Recommendation	Strength of recommendation	Certainty of evidence
Further diagnostic testing for people with suspected ACS		
In people at intermediate risk (as defined by a validated CDP) with elevated troponin concentrations (> 99th percentile), inpatient investigation is recommended.	Strong	Moderate
In people at intermediate risk without elevated troponin concentrations, consider outpatient investigation with non-invasive testing.	Consensus	
In people at low risk who remain symptom-free, further cardiac testing for CAD is not routinely required.	Consensus	
Primary care and regional and remote presentations		
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

Abbreviations: ACOMI, acute coronary occlusion myocardial infarction; ACS, acute coronary syndromes; CAD, coronary artery disease; cTn, cardiac troponin; ECG, electrocardiogram; ED, emergency department.

TABLE 2 | Troponin assay and metrics for use in 0/1- and 0/2-h sampling strategies.

Assay	Sampling time points	A[a]	B[a]	C[a]	D[a]	E[a]	F Female 99th percentile	G Male 99th percentile
Hs-cTnI (Architect; Abbott)	0/1-h	< 4	< 5	< 2	≥ 64	≥ 6	16	34
	0/2-h	< 4	< 6	< 2	≥ 64	≥ 15	16	34
Hs-cTnI (Access; Beckman Coulter)	0/1-h	< 4	< 5	< 4	≥ 50	≥ 15	11	20
	0/2-h	< 4	< 5	< 5	≥ 50	≥ 20	11	20
Hs-cTnI (Centaur; Siemens)	0/1-h	< 3	< 6	< 3	≥ 120	≥ 12	40	58
	0/2-h	< 3	< 8	< 7	≥ 120	≥ 20	40	58
Hs-cTnI (Atellica; Siemens)	0/1-h	< 4	< 6	< 3	≥ 120	≥ 12	39	54
	0/2-h	NA	NA	NA	NA	NA	NA	NA
Hs-cTnI (Vitros; Clinical Diagnostics)	0/1-h	< 1	< 2	< 1	≥ 40	≥ 4	9	12
	0/2-h	< 1	< 2	< 3	≥ 40	≥ 5	9	12
Hs-cTnT (Elecsys; Roche)	0/1-h	< 5	< 12	< 3	≥ 52	≥ 5	9	17
	0/2-h	< 5	< 14	< 4	≥ 52	≥ 10	9	17
Hs-cTnI (Pathfast; LSI Medience)[b]	0/1-h	< 3	< 4	< 3	≥ 90	≥ 20	20	30
	0/2-h	< 3	TBD	TBD	≥ 90	TBD	20	30
Hs-cTnI (Triage True; Quidel)[b]	0/1-h	< 4	< 5	< 3	≥ 60	≥ 8	14	26
	0/2-h	< 4	TBD	TBD	≥ 60	TBD	14	26
Hs-cTnI (VTLi, Siemens)[b]	0/1-h	< 4	TBD	TBD	TBD	TBD	18	27
	0/2-h	< 4	< 6	< 5	≥ 60	≥ 15	18	27

Abbreviations: hs-cTnI, high-sensitivity cardiac troponin I; hs-cTnT, high-sensitivity cardiac troponin T; NA, not available; TBD, to be determined.
[a]A, B, C, D and E refer to cardiac troponin values in Figure 4.
[b]Point of care assay—99th percentiles presented in column F and G are as per the International Federation of Clinical Chemistry tables rounded to the nearest whole number [22].

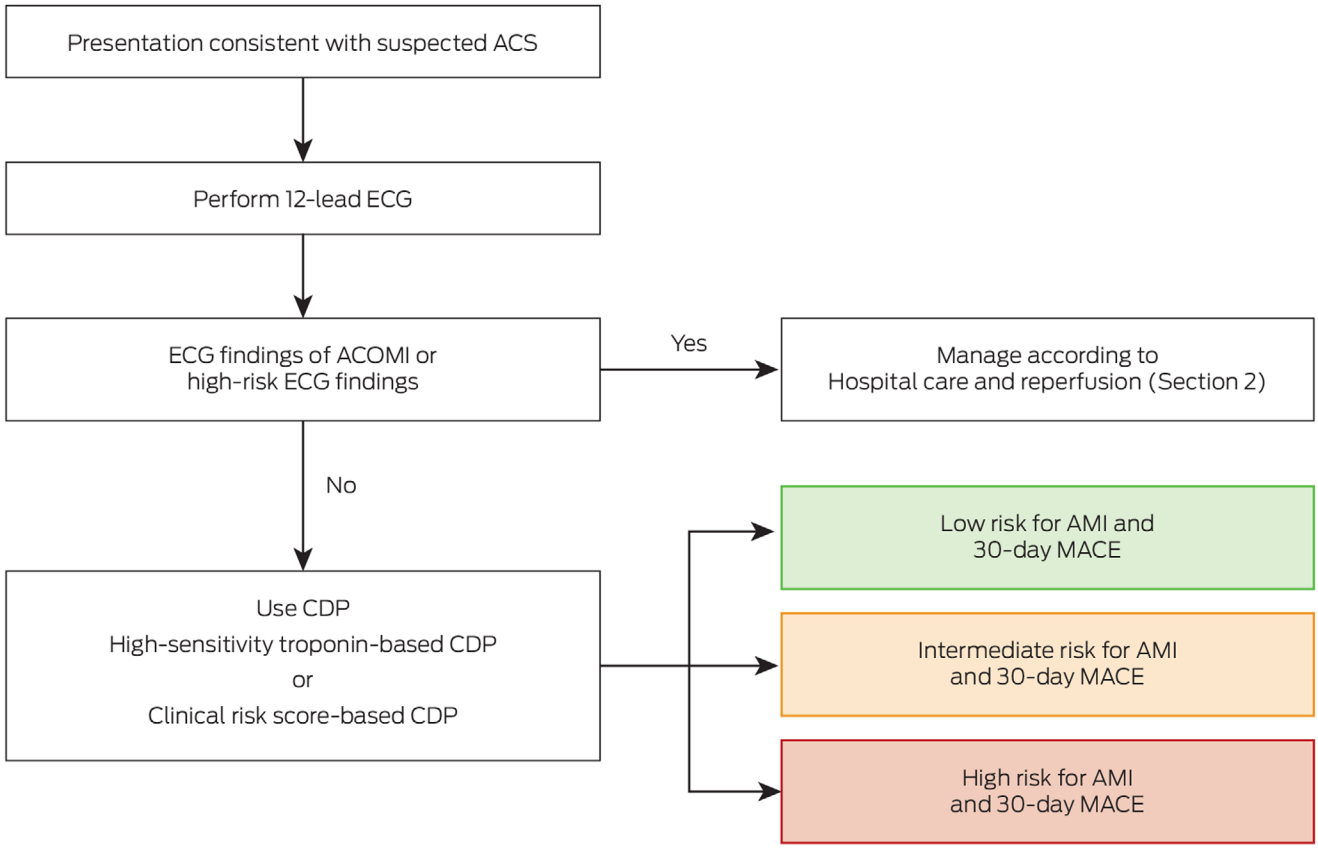


FIGURE 3 | Assessment process for people with suspected acute coronary syndromes (ACS). ACOMI, acute coronary occlusion myocardial infarction; AMI, acute myocardial infarction; CDP, clinical decision pathway; ECG, electrocardiogram; MACE, major adverse cardiovascular events.

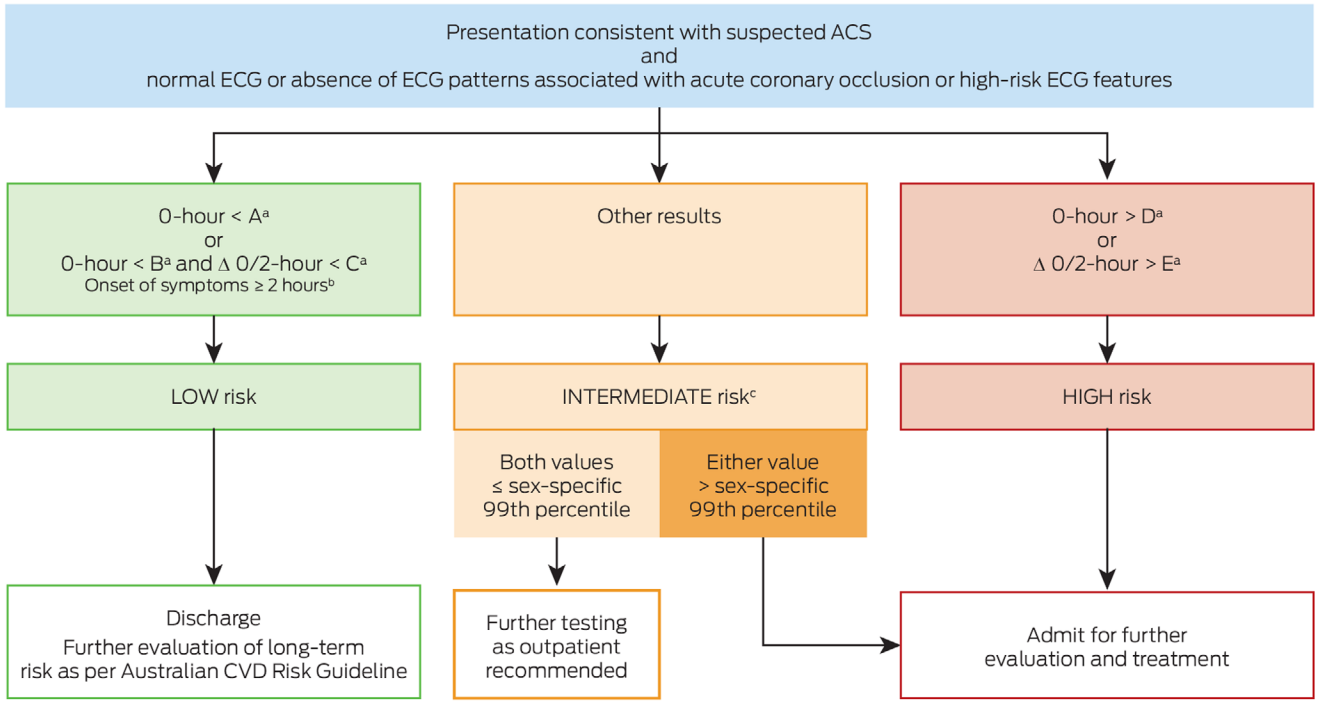


FIGURE 4 | 0/2-h testing recommendations. The 0/2-h time points are shown in this figure. If using a 0/1-h strategy, change time frames accordingly. [a]Refer to Table 2 for interpretation of cardiac troponin assay-specific values (A, B, C, D, E) and sex-specific 99th percentiles. [b]All people with symptom onset < 2 h need serial testing. People with ongoing symptoms should be assessed according to high-risk criteria. [c]See section 'Further diagnostic testing and management of people classified as intermediate risk for suspected ACS' for more details. ACS, acute coronary syndromes; CVD, cardiovascular disease; ECG, electrocardiogram.

TABLE 3 | Summary of recommendations for hospital care and reperfusion strategies.

Recommendation	Strength of recommendation	Certainty of evidence
Acute management of STEMI/ACOMI[a]		
Eligibility for reperfusion		
In people with STEMI/ACOMI within 12 h of symptom onset, perform emergency reperfusion with either primary PCI or fibrinolytic therapy.	Strong	Moderate
In people with STEMI/ACOMI, symptom onset over 12 h before presentation and evidence of continuing myocardial ischaemia (persistent ischaemic symptoms, haemodynamic compromise and/or life-threatening arrhythmias), perform emergency reperfusion with primary PCI.	Strong	Moderate
Choice of reperfusion strategy		
In people with STEMI/ACOMI within 12 h of symptom onset, primary PCI is the preferred reperfusion strategy over fibrinolysis, if it can be performed within 120 min of first medical contact.	Strong	High
In people with STEMI/ACOMI within 12 h of symptom onset, perform fibrinolysis if primary PCI cannot be delivered within 120 min of first medical contact.	Strong	Moderate
Administration of fibrinolytic therapy		
In people with STEMI/ACOMI for whom fibrinolysis is the preferred reperfusion strategy, it should be delivered within 30 min 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
Procedural recommendations in primary percutaneous coronary intervention		
For people with STEMI/ACOMI at a PCI-capable centre, deliver primary PCI within 60 min of arrival. For people with STEMI/ACOMI transferred from a non-PCI centre, deliver primary PCI within 90 min of first hospital arrival.	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 h following occlusion of an IRA, do not perform routine PCI to this artery.	Strong	Moderate
Ongoing management of fibrinolytic-treated people		
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 h of 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
Acute management of NSTEACS		
Risk stratification for people with confirmed NSTEACS		
In people with NSTEACS, consider using the GRACE risk score to determine short- and long-term cardiovascular prognosis.	Weak	High

(Continues)

TABLE 3 | (Continued)

Recommendation	Strength of recommendation	Certainty of evidence
In people with ACS undergoing coronary angiography, consider using bleeding risk scores to determine short-term bleeding risk.	Weak	Moderate
Routine versus selective invasive management for NSTEACS		
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 not at high or very high risk of adverse cardiovascular events, testing for inducible ischaemia (e.g., stress testing) may guide the need for invasive coronary angiography.	Weak	Moderate
Timing of invasive management for NSTEACS		
In people with NSTEACS with very high-risk criteria, an immediate invasive strategy within 2 h of diagnosis is recommended.	Consensus	
In people with NSTEACS with high-risk criteria, consider an early invasive strategy within 24 h of diagnosis.	Weak	High
Procedural considerations in NSTEACS		
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
Antiplatelet therapy in the acute phase		
In people with STEMI/ACOMI treated with fibrinolytic therapy, give dual antiplatelet therapy with aspirin and clopidogrel.	Strong	Moderate
In people with STEMI/ACOMI 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/ACOMI 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, but not during the first 30 days following an ACS event.	Weak	Moderate
In people with ACS with concomitant non-valvular atrial fibrillation and CHA2DS2VA[b] score > 1, give aspirin and clopidogrel, together with a non-vitamin K oral anticoagulant.	Strong	High
In people with STEMI/ACOMI undergoing primary PCI or those with NSTEACS undergoing an invasive strategy, routine glycoprotein IIa/IIIb inhibitor (GPI) is not recommended.	Consensus	
Anticoagulant therapy in the acute phase		
People treated with fibrinolytic therapy should receive anticoagulation (unfractionated heparin or enoxaparin).	Strong	Moderate

(Continues)

TABLE 3 | (Continued)

Recommendation	Strength of recommendation	Certainty of evidence
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
Acute management of ACS with cardiac arrest		
In people with return of spontaneous 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
Acute management of ACS with cardiogenic shock		
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/ACOMI and cardiogenic shock, consider left ventricular assist devices.	Weak	Moderate
Treatment for ACS with multivessel disease without cardiogenic shock		
In haemodynamically stable people with STEMI/ACOMI 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/ACOMI 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
Coronary artery bypass graft surgery in ACS		
In people with STEMI/ACOMI, 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
Treatment for spontaneous coronary artery dissection		
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

Abbreviations: ACOMI, acute coronary occlusion myocardial infarction; ACS, acute coronary syndromes; CABG, coronary artery bypass grafting; ECG, electrocardiogram; GRACE, Global Registry of Acute Coronary Events; MI, myocardial infarction; MVD, multivessel disease; NSTEACS, non-ST-segment elevation acute coronary syndromes; PCI, percutaneous coronary intervention; SCAD, spontaneous coronary artery dissection; STE, ST-segment elevation; STEMI, ST-segment elevation myocardial infarction.

[a]These recommendations apply to people with STEMI and with ECG changes that may not be recognised as STEMI but are indicative of ACOMI, specifically: high lateral MI, posterior MI, right ventricular MI, De Winter T waves, left bundle branch block with modified Sgarbossa criteria.

[b]CHA2DS2VA is a scoring system that evaluates the risk of stroke in people with atrial fibrillation.

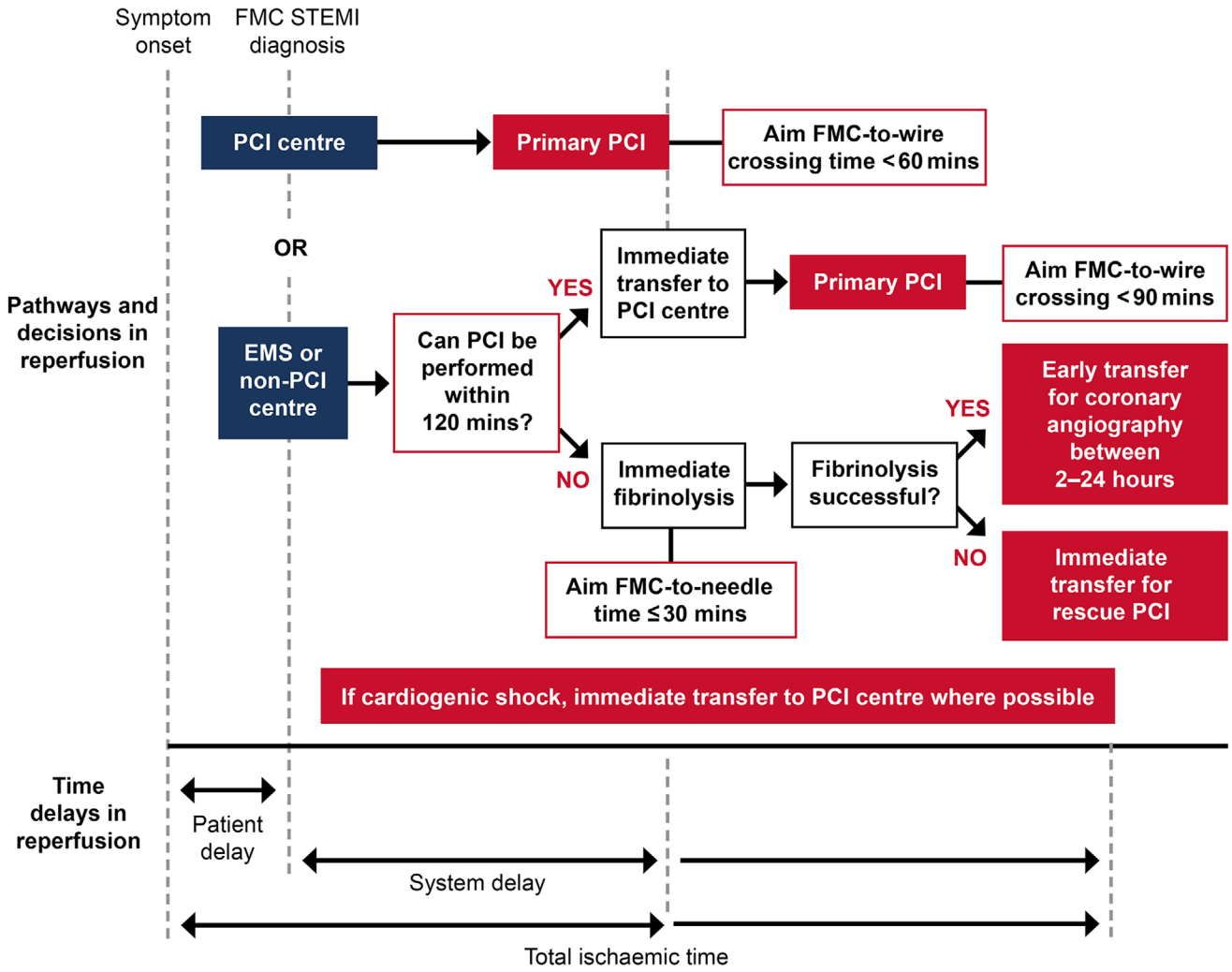


FIGURE 5 | Decision-making and organisation of reperfusion strategies within first 12 h of medical contact. Adapted from Chew et al. [11]. EMS, emergency medical service; FMC, first medical contact; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction.

Certain high-risk ECG patterns, such as Wellens T waves, diffuse ST-segment depression (STD) in multiple leads with STE in aVR, and hyperacute T waves, are associated with potential progression to ACOMI. Recognition of these patterns should prompt urgent, continuous cardiac monitoring and consideration for coronary angiography.

4.1.1.2 | Using Clinical Decision Pathways (CDPs) Incorporating High-Sensitivity Cardiac Troponin (hs-cTn) Assays for More Efficient Risk Assessment Compared With Traditional (Contemporary/Conventional) Troponin-Based Algorithms. People with suspected ACS should receive care guided by an evidence-based CDP that includes assay-specific troponin results to categorise people as high, intermediate or low risk (Figure 3). Pathways may rely solely on ECGs with hs-cTn results or combine clinical risk scores with contemporary cardiac troponin (cTn) values. The diagnostic threshold for myocardial injury is assay-specific; therefore, serial cTn testing can only be interpreted when performed with the same assay.

In the absence of ischaemic ECG findings, CDPs incorporating hs-cTn rather than contemporary troponin assays are recommended for safe, rapid risk stratification and decision-making [24, 25]. The 0/1- or 0/2-h strategy (Figure 4, Table 2), or the United Kingdom (UK) High sensitivity troponin in the evaluation of patients with acute coronary syndrome (High-STEACS) algorithm is recommended [24, 26–32].

People stratified as high risk of major adverse cardiovascular events (MACE) using hs-cTn-based CDPs should be admitted for further evaluation, while those identified as intermediate risk require further evaluation either as an inpatient or outpatient depending on their troponin results (Figure 4). Further testing to exclude AMI is not required for people classified as low risk using an hs-cTn strategy (Figure 4).

In most hospitals, standard turnaround times for laboratory-based assay results render the 0/1-h strategy impractical; therefore, a 0/2-h strategy is currently the most pragmatic option in most settings.

TABLE 4 | Summary of recommendations for recovery and secondary prevention.

Recommendation	Strength of recommendation	Certainty of evidence
Person-centred non-pharmacological secondary prevention		
For all people with ACS, refer to a multi-disciplinary exercise-based cardiac rehabilitation program prior to discharge.	Strong	Moderate
For all people with ACS, provide advice on lifestyle[a] 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 preventive medicines.	Consensus	
Vaccination against influenza and other respiratory pathogens		
In people with ACS, vaccinations for influenza and other respiratory pathogens are recommended.	Consensus	
Post-ACS pharmacotherapy		
Anti-platelet therapy		
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
Lipid-modifying therapy		
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	

(Continues)

TABLE 4 | (Continued)

Recommendation	Strength of recommendation	Certainty of evidence
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
β-Blocker therapy		
In people with ACS and LV impairment, β-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 β-blockers.	Weak	Moderate
Renin-angiotensin antagonist therapies		
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
Colchicine therapy		
In people discharged following an ACS, consider initiating colchicine (0.5 mg daily) and continuing long-term unless contraindicated or colchicine intolerant.	Weak	Moderate

Abbreviations: ACS, acute coronary syndromes; DAPT, dual antiplatelet therapy; HMG-CoA, hydroxymethylglutaryl-coenzyme A; LV, left ventricular; LVEF, left ventricular ejection fraction; OAC, oral anticoagulant; SAPT, single antiplatelet therapy.
[a]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.

The guideline includes tailored practice points to help clinicians apply hs-cTn strategies appropriately across key groups at increased risk, including women, older adults, First Nations peoples and people with renal impairment.

4.1.1.3 | Further Diagnostic Testing and Management of People Classified as Intermediate Risk for Suspected ACS. Invasive or non-invasive inpatient testing is recommended for people at intermediate risk with elevated hs-cTn above the sex-specific 99th percentile based on the relatively high rate of a cardiac event within 30 days (2%–22%) [33, 34].

If non-invasive testing is selected in these people, the guideline supports using computed tomography coronary angiography (CTCA) as a first-line investigation for those without previously known coronary artery disease (CAD) presenting with intermediate-risk ACS, if no contraindications exist.

Outpatient non-invasive testing can be considered (ideally within 30 days) for people at intermediate risk with serial troponin values ≤99th percentile, as the 30-day event rate is lower (<2%) [35].

Considerations for non-invasive test selection in First Nations peoples and those in regional and remote areas are presented in the guideline, including implementing telemedicine support and lower threshold for using CTCA when available for definitive early identification of CAD. This recognises their reduced access to services, longer wait times, and greater travel distance to access diagnostic services.

4.2 | Section 2: Hospital Care and Reperfusion

A summary of the recommendations for hospital care and reperfusion strategies is presented in Table 3.

4.2.1 | What's New in the 2025 Guideline?

4.2.1.1 | Acute Management of STEMI/ACOMI: Stronger Emphasis on the Optimal Timing of Primary Percutaneous Coronary Intervention (PCI). Timely reperfusion limits the extent of myocardial infarction and reduces mortality by minimising total ischaemic time (Figure 5) [36, 37]. The guideline emphasises the importance of adopting quality assurance

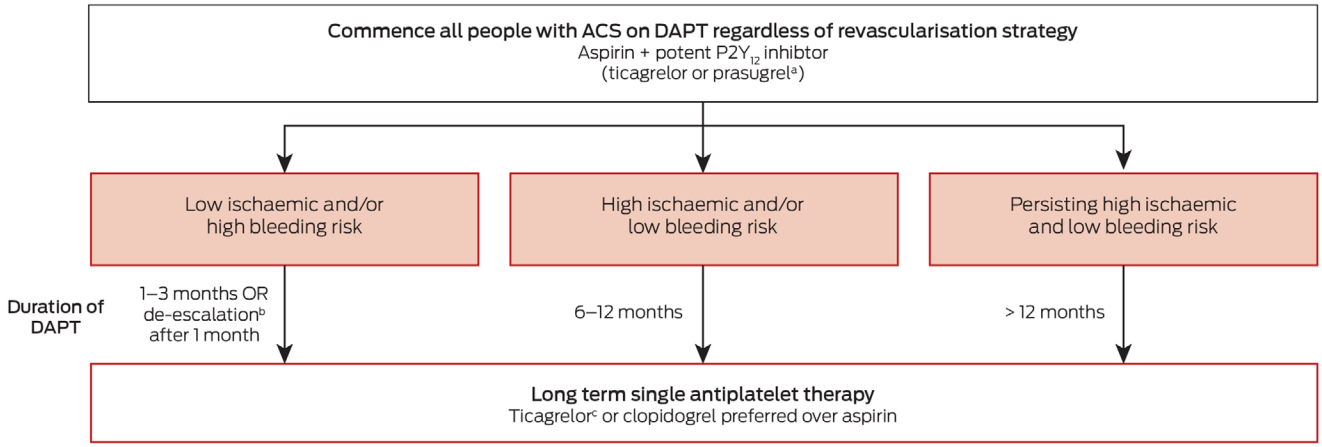


FIGURE 6 | Decision tree for dual antiplatelet therapy (DAPT) duration following an acute coronary syndrome (ACS). [a]Prasugrel is not indicated in people who do not undergo percutaneous coronary intervention. [b]Refers to the de-escalation of DAPT to aspirin and a less potent P2Y12 inhibitor (clopidogrel). [c]Current Pharmaceutical Benefits Scheme criteria preclude the prescription of ticagrelor as single therapy.

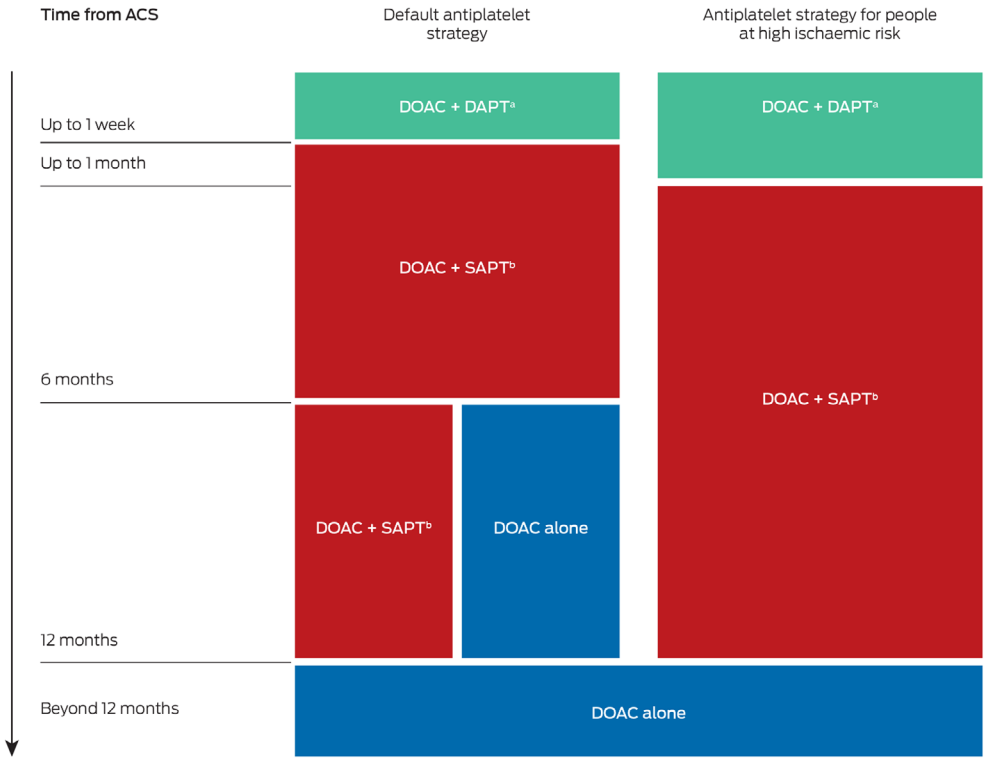


FIGURE 7 | Recommended antiplatelet treatment strategies for people with acute coronary syndromes (ACS) requiring long-term direct oral anticoagulant (DOAC) for atrial fibrillation. [a]DAPT: aspirin plus clopidogrel preferred. [b]SAPT: clopidogrel preferred. People receiving triple therapy should be given a proton pump inhibitor. DAPT, dual antiplatelet therapy; SAPT, single antiplatelet therapy.

targets to reduce delays to wire crossing if primary PCI is the chosen reperfusion strategy.

- For people with STEMI/ACOMI presenting to primary PCI-capable centres, first medical contact to wire crossing time should not exceed 60 min [38].
- For people presenting to non-PCI-capable centres, first medical contact to wire crossing time should not exceed 90 min (Figure 5) [38].

4.2.1.2 | Acute Management of Non-ST-Segment Elevation Acute Coronary Syndromes (NSTEACS): Use of Intravascular Imaging (IVI)-Guided PCI. IVI-guided PCI can be considered in people with NSTEACS undergoing an invasive approach. IVI-guided PCI, using intravascular ultrasound or optical coherence tomography, has shown superior outcomes compared with standard angiography-guided PCI in multiple randomised controlled trials and meta-analyses, including significant reductions

in risk of all myocardial infarction, target vessel myocardial infarction, cardiac mortality, all-cause mortality and target lesion repeat revascularisation [39, 40]. However, given variability in trial populations and lesion complexity, the recommendation for intracoronary imaging over angiography alone should not be applied to every PCI procedure.

4.2.1.3 | Managing ACS With Cardiogenic Shock: Use of Haemodynamic Support Devices and Left Ventricular Assist Devices. In people with ACS and cardiogenic shock, routine insertion of an intra-aortic balloon pump and venoarterial extracorporeal membrane oxygenation are not recommended due to increased risk of bleeding and vascular complications with no survival benefits, though they may be considered in select cases [41–44].

The guideline supports the use of left ventricular assist devices in select people with STEMI/ACOMI and cardiogenic shock, given potential survival benefits despite increased bleeding and vascular complication risks [45].

4.2.1.4 | Treatment of ACS With Multivessel Disease (MVD): Considerations for PCI of Non-Infarct-Related Arteries (IRAs) and Invasive Physiology Assessment. In people with STEMI/ACOMI and MVD without cardiogenic shock, complete revascularisation, especially when performed at the time of primary PCI or within 19 days of the index procedure, reduces the risk of death, non-fatal myocardial infarction, stroke, unplanned revascularisation and heart failure hospitalisation compared with staged PCI of the non-IRA [46, 47]. However, these benefits may not apply to complex MVD, where coronary artery bypass grafting (CABG) may be more appropriate. Currently, there are no dedicated trials comparing complete versus culprit-only PCI in people with NSTEACS, although observational data suggest long-term benefit with multivessel revascularisation [48].

Physiology-guided PCI of the non-IRA using fractional flow reserve (FFR) has not shown clear benefit over an angiography-guided approach in STEMI/ACOMI, but may be reasonable to use in people with NSTEACS and older adults [49–54].

4.2.1.5 | Managing ACS Due to SCAD: Considerations for Selective Revascularisation. In people with ACOMI or NSTEACS due to SCAD who are haemodynamically stable, routine revascularisation is not recommended due to its association with PCI-related complications and limited evidence of benefit [55–57]. However, in cases of haemodynamic compromise or significant ongoing ischaemia, urgent revascularisation with PCI or CABG may be required [58, 59].

4.2.1.6 | Pharmacotherapy in the Acute Phase: Timing of Platelet $P2Y_{12}$ Inhibitor Administration in STEMI/ACOMI and NSTEACS. In people with STEMI/ACOMI undergoing primary PCI, $P2Y_{12}$ inhibitor pretreatment (before angiography) has not shown clear mortality or bleeding benefits, though pre-hospital administration may reduce reinfarction [60]. Pretreatment can be considered when STEMI/ACOMI is confirmed; however, deferring $P2Y_{12}$ inhibitor administration until coronary anatomy is known is reasonable if diagnosis is uncertain or cardiothoracic surgery may be needed.

Similarly, in people with NSTEACS, $P2Y_{12}$ inhibitors can be withheld until the coronary anatomy is known and if angiography can be performed within the recommended time frames [61, 62].

4.3 | Section 3: Recovery and Secondary Prevention

A summary of the recommendations for recovery and secondary prevention is presented in Table 4.

4.3.1 | What's New in the 2025 Guideline?

4.3.1.1 | Non-Pharmacological Interventions: More Detailed Advice on Post-Discharge Care, Including Cardiac Rehabilitation and Secondary Prevention Programs, Medicine Adherence Strategies, Vaccinations and Screening for Mental Health Conditions. The guideline recommends referring all people with ACS to a multidisciplinary, exercise-based cardiac rehabilitation program before discharge, or to a flexible, cardiovascular risk management program if an exercise-based option is not available [63, 64]. Telehealth is an acceptable alternative for people with ACS in regional and remote areas [65]. Digital health interventions, such as text reminders, mobile phone applications, wearables and telehealth consultations, can enhance post-ACS cardiovascular risk management [66].

As mental health conditions are common in people post-ACS, screening for depression and other mental health conditions using validated tools and referring for appropriate mental health support is recommended [67, 68].

People with ACS should receive recommended vaccinations, including influenza, pneumococcal, respiratory syncytial virus (RSV) (for those aged ≥ 60 years) and COVID-19, as they are at increased risk of severe illness from respiratory infections [69].

Initiating guideline-recommended therapies during hospitalisation strongly predicts adherence at 6 months [70]. The guideline recommends implementing strategies to optimise long-term medicine adherence such as prescribing preventive medicines at discharge, providing person-centred medicines education, daily alerts/reminders and fixed combination medicines.

4.3.1.2 | Antiplatelet Therapy: Treatment Algorithms to Enable More Tailored Prescribing of Antiplatelet and Anticoagulation Therapies. There are new recommendations and updated guidance on dual antiplatelet therapy (DAPT) duration following an ACS (Figure 6) and antiplatelet treatment strategies for people with ACS requiring long-term anticoagulation (Figure 7).

4.3.1.3 | Lipid-Modifying Therapy: New Recommended Treatment Target for Low-Density Lipoprotein Cholesterol (LDL-C) and PCSK9 Inhibitor Initiation. People with ACS should receive the highest tolerated dose of statins,

unless contraindicated or completely statin intolerant. The guideline recommends a new initial LDL-C target of < 1.4 mmol/L and a reduction of at least 50% from baseline, as each 1.0 mmol/L reduction is associated with a 20% lower risk of major cardiovascular events [71, 72].

If LDL-C level remains above target despite statin therapy, ezetimibe can be added. For people already on intensive lipid-lowering therapy and ezetimibe but still with suboptimal LDL-C levels, the guideline recommends adding PCSK9 inhibitors (e.g., alirocumab, evolocumab and inclisiran) to further lower LDL-C and reduce cardiovascular events and mortality [73].

4.3.1.4 | Other Post-ACS Pharmacotherapy: New Recommendations on β-Blockers and Angiotensin Receptor-Neprilysin Inhibitors. In people with ACS, preserved left ventricular function, and on optimal medical therapy following revascularisation, β-blockers may be withheld, as evidence shows no reduction in death, myocardial infarction or other cardiovascular events beyond 12 months [74, 75].

Angiotensin receptor–neprilysin inhibitors are not recommended, considering no difference in cardiovascular death or heart failure compared with ACE inhibitors in those with reduced left ventricular ejection fraction (LVEF) or transient pulmonary congestion [76].

5 | Implementation

A comprehensive suite of resources to support understanding and implementation of the guideline, including infographics for both healthcare professionals and the general public, is available at www.heartfoundation.org.au/for-professionals/acs-guideline. Continuing professional development opportunities (e.g., accredited webinars) have been and will continue to be provided to healthcare professionals following the guideline publication to facilitate adoption of the new recommendations in clinical practice.

6 | Conclusion

The *Australian clinical guideline for diagnosing and managing acute coronary syndromes 2025* provides contemporary, evidence-informed recommendations to support healthcare professionals in delivering safe, accurate and efficient care across the care continuum. It reflects the growing complexity of ACS care, incorporating advances in diagnostics, therapeutics and secondary prevention, while emphasising person-centred approaches and equity in access to care. To access the full guideline, visit www.heartfoundation.org.au/for-professionals/acs-guideline.

Author Contributions

David B. Brieger: writing – review and editing. **Louise A. Cullen:** writing – review and editing. **Tom G. Briffa:** writing – review and editing. **Sarah Zaman:** writing – review and editing. **Ian A. Scott:** writing – review and editing. **Cynthia Papendick:** writing – review and editing. **Elaine Ho:** conceptualization, writing – original draft, writing – review and editing. **Victoria Leitch:** conceptualization, writing – original draft, writing – review and editing. **Dannii Dougherty:** conceptualization, writing – review and editing. **Garry Jennings:** writing – review and editing.

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Conflicts of Interest

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Data Availability Statement

Data sharing is not applicable to this article as no new data were created or analysed in this study.

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