



Guideline 14.1 - Acute Coronary Syndromes: Presentation

Summary

To whom does this guideline apply?

This guideline applies to adults.

Who is the audience for this guideline?

This guideline is for use by first responders and health professionals in the prehospital and emergency department setting.

Summary of Recommendations

The Australian and New Zealand Committee on Resuscitation (ANZCOR) makes the following recommendations:

- 1. A 12-lead electrocardiograph (ECG) should be acquired and interpreted by a qualified provider in the pre-hospital emergency setting or as soon as possible after first medical contact in patients with suspected acute coronary syndrome (ACS) to allow identification and triage of high-risk individuals (e.g. STEMI) [Good Practice Statement].
- 2. Cardiac biomarkers, specifically cardiac troponin, should be evaluated in all patients with suspected ACS [Good Practice Statement].
- 3. ANZCOR recommends coronary angiography is indicated in patients with clinical features consistent with ACS and an elevated high-sensitivity cardiac troponin (hs-cTn) to determine the need for revascularization [Good Practice Statement].
- 4. For patients at moderate or lower risk of unstable angina, computed tomography (CT) coronary angiography or functional testing (stress echo or nuclear studies) is preferred [Good Practice Statement].

1.0 | Symptoms and Signs

In ACS, acute onset chest discomfort is characterised by retrosternal pain, pressure, or

heaviness with radiation to either arm, jaw, or neck.¹ Associated symptoms may include epigastric pain, dyspnoea, sweating, nausea, and syncope. Non-specific symptoms commonly occur (including indigestion-like symptoms, isolated dyspnoea or fatigue, or isolated epigastric pain), and are more frequent in older patients, women, and in patients with diabetes, chronic renal impairment, chronic inflammatory conditions or cognitive impairment.²⁴ ACS is more likely among patients with risk factors such as older age, male sex, hyperlipidaemia, family history, diabetes, smoking, hypertension, renal impairment, and previous ACS.⁵ Certain clinical features increase the likelihood of ACS including; increased pain following physical exertion and responsiveness to nitrate administration,⁴ though response is not diagnostic. Physical signs are frequently absent in ACS presentations. Cardiac auscultation may reveal a systolic murmur relating to ischemic mitral regurgitation, aortic stenosis mimicking ACS, or more rarely a ventricular septal defect.¹ Signs of heart failure, hemodynamic instability, or arrhythmia warrant rapid diagnosis and treatment. Signs suggestive of non-coronary causes of chest pain may be elicited by physical examination, such as reproducibility of chest pain on chest wall palpation, which has a negative predictive value for ACS.⁴

Nonetheless, overall diagnostic utility of the above clinical symptoms is limited and caution should be used in their interpretation. Signs and symptoms are more useful in combination with other information such risk factors, a pre-hospital ECG, and other diagnostic tests such as biomarkers, in making triage and treatment decisions in the out-of-hospital and emergency department (ED) setting.

Clinicians should be aware of various patient related factors which can impede seeking medical help. These factors include older age, belonging to racial and ethnic minorities, female gender, diabetes, current smoking or other substance abuse, lower socioeconomic status, social isolation, and patients with English as a second language.⁸⁻¹¹ It is important to recognise these issues when providing care to Indigenous people who are at higher risk for late presentation with ACS.¹²⁻¹⁴ It is important that the health care providers are trained to expeditiously identify ACS irrespective of these factors.

2.0 | The 12 Lead Electrocardiograph

In patients with suspected ACS a 12-lead ECG should be acquired and interpreted by a qualified provider in the pre-hospital emergency setting as soon as possible after first medical contact.¹ This will allow early recognition and intervention with high-risk ACS and ST elevation myocardial infarction (STEMI), including hospital activation of the cardiac catheterisation laboratory, or consideration of pre-hospital thrombolysis if primary percutaneous coronary intervention (PPCI) cannot be facilitated within the required period (Refer to ANZCOR Guideline 14.3).¹⁵ Pre-hospital ECG interpretation may be best performed by trained clinicians at the receiving hospital after data transmission of the ECG. If this is not available, then computer assisted electrocardiograph (ECG) interpretation or pre-hospital provider interpretation are alternatives [CoSTR 2015 weak recommendation, very-low-quality evidence].¹⁵

For non-ST elevation ACS (NSTEACS), the ECG may be normal in over 30% of patients. Abnormalities may include transient ST-segment elevation, T-wave abnormalities, and ST-segment depression. When present, ST-segment depression correlates with worsened prognosis, while data regarding the prognostic impact of T wave inversion are conflicting. Several ECG patterns in NSTEACS, including but not limited to de Winter's T waves and Wellens'

syndrome, are associated with occluded vessels or critical stenosis causing ongoing ischaemia and warrant consideration of urgent angiography/revascularisation.^{20, 21} Patients with left-bundle branch block and high clinical suspicion of ongoing ischemia should be managed like STEMI presentations. Specific ECG criteria, such as Sgarbossa's criteria, may be used to assist in decision making. However, more than 50% of patients presenting with chest pain and left-bundle branch block do not have a final diagnosis of ACS.^{1, 22}

3.0 | Cardiac Biomarkers and Imaging techniques

All patients who present to the ED with suspected ACS should be evaluated with cardiac biomarkers as part of the initial evaluation [Good Practice Statement]. Cardiac specific troponin (cTnI or cTnT) has become the most widely utilized and validated diagnostic biomarker for myocardial infarction (MI). More recently, high-sensitivity cardiac troponin (hs-cTn) has become the preferred laboratory test as hs-cTn improves diagnostic accuracy for MI compared to conventional assays. Moreover, hs-cTn allows a more rapid 'rule in' and 'rule out' of MI. A clinical presentation consistent with myocardial ischemia, in combination with a dynamic elevation of cardiac troponin above the 99th percentile is diagnostic of MI.

Algorithms to exclude ACS utilising hs-cTn commonly use an initial laboratory level and a repeat level 1 or 2 hours later. These are commonly utilised alongside risk scoring systems such as the Vancouver rule, thrombolysis in myocardial infarction (TIMI) score, HEART score, Emergency Department Assessment of Chest Pain (EDACS) or North American Chest Pain rule. The positive predictive value for MI among patients meeting the 0 hour/1 hour "rule in" criteria is 70 to 75%, while the negative predictive value and sensitivity are both over 99%. ^{5, 6, 23}. These newer rapid algorithms have replaced the use of formal chest pain evaluation units in many settings.

Currently available point of care troponins have lower sensitivity, diagnostic accuracy, and lower negative predictive value. As newer higher sensitivity point of care troponins become available, diagnostic algorithms incorporating pre-hospital diagnosis may become more feasible.

In patients presenting with clinical features consistent with ACS and an elevated hs-cTn, coronary angiography is indicated to determine if revascularisation (either percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG)) is required [Good Practice Statement].¹

In patients with a very high clinical likelihood of unstable angina, invasive angiography (inpatient or outpatient) may be still the best option for diagnosis even if NSTEMI is excluded by the hs-cTn algorithm.

If high-risk ACS is not diagnosed, transthoracic echocardiography (TTE) maybe considered to identify abnormalities suggestive of ischemia or infarct, specifically segmental wall motion abnormalities. Furthermore, TTE can assist in diagnosis of aortopathies, pericardial effusion, valvular heart disease, and acute right ventricular dilatation that may suggest pulmonary embolism.

For patients at moderate or lower risk of unstable angina, CT coronary angiography or functional testing (stress echo or nuclear studies) is preferred.²⁴ Not all troponin elevations are related to acute coronary syndromes and have been described in conditions such as myocarditis,

tachyarrhythmias, pulmonary embolism, aortic dissection, acute heart failure, septic shock, valvular heart disease, hypertensive emergencies, secondary to cardiotoxic drugs as well as after therapeutic procedures like electrophysiological ablations, or electrical cardioversions.^{1, 25}

Abbreviations

Abbreviation	Meaning/Phrase
ACS	Acute coronary syndrome
ANZCOR	Australian and New Zealand Committee on Resuscitation
ARC	Australian Resuscitation Council
CABG	Coronary artery bypass graft
CoSTR	Consensus on Science with Treatment Recommendations
СТ	Computed tomography
cTnI	Cardiac troponin
ECG	Electrocardiograph
ED	Emergency department
EDACS	Emergency Department Assessment of Chest Pain
hs-cTn	High sensitivity cardiac troponin
МІ	Myocardial infarction
NSTEACS	Non ST elevation acute coronary syndrome
PCI	Percutaneous coronary intervention
PPCI	Primary percutaneous coronary intervention
STEMI	ST elevation myocardial infarction
TIMI	thrombolysis in myocardial infarction
TTE	Transthoracic echocardiography

About this Guideline

Search date/s	This review was completed in October 2023
Questions/PICOs:	This guideline has been developed from the previous ANZCOR guideline 2016 and questions included in the ILCOR 2015 and 2022 CoSTR
Method:	Literature review of the most recent acute coronary syndrome and related guidelines from the Australian Heart Foundation / Cardiac Society of Australia and NZ (2016), the European Society of Cardiology (2020) in addition to related ILCOR reviews to produce a locally relevant document.
Main Changes:	
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Other consultation:	N/A
Worksheet:	N/A
Approved:	December 2024
Guideline superseded:	Guidelines 14.0, 14.1, 14.1.2, 14.2, 14.3

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