AUSTRALIAN RESUSCITATION COUNCIL

NEW ZEALAND Resuscitation Council WHAKAHAUORA AOTEAROA

ANZCOR Guideline 14.1 – Acute Coronary Syndromes: Presentation

Guideline

Who does this guideline apply to?

This guideline applies to adult victims.

Who is the audience for this guideline?

This guideline is for use by first responders and health professionals.

1 Symptoms and Signs

While it is important that patients who are at risk and their families should be able to recognise characteristic symptoms that may be indicative of ACS, it is important to note that signs and symptoms alone are neither sufficiently sensitive nor specific. (LOE IV). (See Guideline 9.2.1 Chest Pain)

Even amongst trained health care providers, signs and symptoms alone should not be used without other information for making the diagnosis of ACS[1,2]. (LOE IV) Signs and symptoms may be useful when used in combination with other information such as biomarkers, risk factors, an ECG and other diagnostic tests, in making triage and some treatment decisions in the out of hospital and emergency department (ED) setting.

A cautionary note also applies to the use of a response to sublingual nitroglycerine therapy as a diagnostic manoeuvre[3-6]. Although it is reasonable to consider nitroglycerine in select patients without contraindications, there is really insufficient data to recommend relief of chest pain with nitroglycerine as a diagnostic test for ACS. (LOE IV)

There are various patient related factors which impede seeking medical help. These factors include older age, belonging to racial and ethnic minorities, female gender, lower social status and social isolation[7-13]. This is particularly important to recognise these issues when providing care to the Australian indigenous, Maori and Pacific Islander population. (LOE IV) It is important that the health care providers are trained to expeditiously identify ACS irrespective of these factors.

2 The 12 Lead Electrocardiograph

The acquisition and interpretation of the ECG is the critical step for the diagnosis, triage and initiation of revascularisation therapy in patients with suspected high risk ACS and STEMI. This early recognition of STEMI patients has the potential to reduce delays to reperfusion and thus improve patient survival.

Therefore in patients with suspected ACS a 12 lead ECG should be acquired and interpreted in the pre-hospital emergency setting as soon as possible after first medical contact[14-18]. Acquisition and interpretation should then result in hospital notification of suspected STEMI with prehospital activation of the cardiac catheterisation laboratory if PPCI is the planned reperfusion strategy[19] (CoSTR 2015, strong recommendation, low-quality evidence).

It is important that the system in place for the interpretation of the prehospital ECG has optimal diagnostic performance with low false positive and false negative rates. This is important to balance the risk of missing the diagnosis of STEMI against the costs of inappropriate activation of resources[19]. If the interpretation for pre-hospital usage is not available by medical staff by field transmission of the ECG for expert interpretation, then interpretation of the ECG by non-physicians trained in ECG interpretation is suggested[20-32] (CoSTR 2015, weak recommendation, very-low-quality evidence)[19].

Further computer assisted interpretation of the ECG may be used as an adjunct or in conjunction to physician or non-physician interpretation in STEMI [33-35]. Whilst computer assisted ECG interpretation is highly specific for STEMI, it is not recommended that it be used as the sole strategy for ruling out STEMI as sensitivity is poor (CoSTR 2015, weak recommendation, very-low-quality evidence)[19].

3 Cardiac Biomarkers

All patients who present to the ED with symptoms suspicious of cardiac ischaemia should be evaluated with cardiac biomarkers as part of the initial evaluation[36,37]. Cardiac specific troponin (cTnI or cTnT) has become the most widely utilized and validated diagnostic biomarker for myocardial infarction and is the preferred laboratory test. (LOE I). Given the burden of presentations of chest pain to emergency departments it is important to rule out the diagnosis of ACS to allow early appropriate discharge of patients. An acceptably low risk at 30 days is defined as a MACE event rate of <1%. To achieve such outcomes, cTn should not be used alone to exclude a diagnosis of ACS but should be combined with a validated clinical risk score (Vancouver rule, TIMI score, HEART score or North American Chest Pain rule) (CoSTR 2015, strong recommendation, very-low-quality evidence)[19].

As cTn may be initially negative if the presentation is very soon after the symptom onset, it is recommended paired biomarker testing (c TnI or c TnT) be performed at 0 and 3-6 hours after symptom onset and combined with a very low clinical risk score to reliably exclude myocardial necrosis[37-39] (CoSTR 2015, weak recommendation, low-quality evidence)[19].

Highly sensitive cardiac troponin assays (10% coefficient of cardiac variation at the 99th percentile) have been shown to have increased sensitivity and become positive at an earlier time after onset of ischaemia when compared to conventional assays[40-43]. This supports their use in the diagnosis of AMI. These assays are able to determine the presence of a positive biomarker reliably at 2 hours[19,44,45]. (LOE II). A negative hs-cTnI at 0 and 2 hours combined with a low clinical risk score (Vancouver or TIMI score) can be used to exclude the diagnosis of ACS (CoSTR 2015, weak recommendation, low-quality evidence)[19].

There has been a lack of evidence of supporting the routine use of point of care troponin testing in isolation as the primary test in a pre-hospital setting to evaluate patients with ACS[46].

It is important to note that not all troponin elevations are related to acute coronary syndromes.

Elevated troponin values have been described in a variety of conditions not at all related to acute coronary syndromes. These include myocarditis, pulmonary embolism, acute heart failure, septic shock, secondary to cardiotoxic drugs as well as after therapeutic procedures like coronary angioplasty, electrophysiological ablations, or electrical cardioversions[47].

There are a variety of biomarkers that have become available including myoglobin and brain natriuretic peptide (BNP), NT-proBNP, D-dimer, C-reactive protein, ischaemia-modified albumin, pregnancy-associated plasma protein A and interleukin 6. These tests however are not supported by sufficient evidence to allow their use in isolation to evaluate patients with symptoms or signs of myocardial ischaemia[48-51].

4 Chest Pain Observation Units

The use of Chest Pain Observation Units (CPUs) and accelerated chest pain assessment pathways using protocols outlined above are recommended in the evaluation of patients with possible ACS. CPUs usually incorporate a protocol or pathway based strategy involving the measurement of serial biomarkers, serial ECG or continuous ECG monitoring to allow for a period of clinical observation integrated with more advanced diagnostic testing[1,2,46,56-63].

This strategy involving biomarker testing with associated protocols and pathways may be recommended as a means to reduce the length of stay, reduce hospital admissions, reduce health care costs and improve diagnostic accuracy in patients who are suspected as suffering ACS[1,2,56-62]. (LOE III-1).

5 Imaging Techniques

In patients with suspected ACS there are a variety of imaging techniques which may be utilised to diagnose acute coronary syndrome. These include CT angiography, MRI, nuclear cardiography and echocardiography[64-78]. A non-invasive test may be considered in selective patients who present to the ED with chest pain and initial non-diagnostic conventional work-up. However it is important to consider both the exposure radiation and iodinated contrast when utilising these imaging modalities. (LOE II).

These non-invasive tests may help to improve the accuracy of the diagnosis and they may also, in select groups, decrease cost, length of stay and time of diagnosis. They may provide valuable short and long term prognostic information about the incidence of future major cardiac events[64-84]. (LOE II).

6 Risk Stratification

There are a number of factors determined from the patient history, physical examination, initial ECG and biomarker testing, that allow the clinician to risk stratify patients. (LOE II).

The Australian indigenous, Maori and Pacific Islander population are at high risk for ischaemic heart disease and present at a younger age with more advanced disease[85]. Features associated with high-risk, intermediate-risk and low-risk non-ST-segment-elevation acute coronary syndromes (NSTEACS).

7 High-risk Features

Presentation with clinical features consistent with acute coronary syndromes (ACS) and any of the following high-risk features[86]:

- Repetitive or prolonged (> 10 minutes) ongoing chest pain or discomfort
- Elevated level of at least one cardiac biomarker (troponin or creatine kinase-MB isoenzyme)
- Persistent or dynamic electrocardiographic changes of ST-segment depression ≥ 0.5 mm or new T-wave inversion ≥ 2 mm
- Transient ST-segment elevation (≥ 0.5 mm) in more than two contiguous leads
- Haemodynamic compromise — systolic blood pressure < 90 mmHg, cool peripheries, diaphoresis, Killip Class > I, and/or new-onset mitral regurgitation
- Sustained ventricular tachycardia
- Syncope
- Left ventricular systolic dysfunction (left ventricular ejection fraction < 0.40)
- Prior percutaneous coronary intervention within 6 months or prior coronary artery bypass surgery
- Presence of known diabetes (with typical symptoms of ACS)
- Chronic kidney disease (estimated glomerular filtration rate < 60 mL/minute) (with typical symptoms of ACS).

8 Intermediate-risk Features

Presentation with clinical features consistent with ACS and any of the following intermediate risk features AND NOT meeting the criteria for high-risk ACS:

- Chest pain or discomfort within the past 48 hours that occurred at rest, or was repetitive or prolonged (but currently resolved)
- Age > 65 years
- Known coronary heart disease — prior myocardial infarction with left ventricular ejection fraction ≥ 0.40, or known coronary lesion more than 50% stenosed
- No high-risk changes on electrocardiography (see above)
- Two or more of the following risk factors: known hypertension, family history, active smoking or hyperlipidaemia
- Presence of known diabetes (with atypical symptoms of ACS)
- Chronic kidney disease (estimated glomerular filtration rate < 60 mL/minute) (with atypical symptoms of ACS)
- Prior aspirin use.

9 Low-risk Features

Presentation with clinical features consistent with an acute coronary syndrome *without* intermediate-risk or high-risk features includes onset of angina symptoms within the last month, *or* worsening in severity or frequency of angina, *or* lowering of angina threshold.

A number of risk scores have been developed to assist in risk stratification using simple risk variables that can be calculated on information easily available to clinicians. These scores have been validated in large studies and predict major adverse cardiovascular outcomes in a robust fashion.

10 The Thrombolysis in Myocardial Infarction (TIMI) score is one such score[87] (Table 1)

Predictor Variable	Point Value of Variable
Age ≥65 years	1
≥3 risk factors for CAD	1
Risk factors ● Family history of CAD ● Hypertension ● Hypercholesterolemia ● Diabetes ● Current smoker	
Aspirin use in last 7 days	1
Recent, severe symptoms of angina • ≥2 angina events in last 24 hours	1
Elevated cardiac markers • CK-MB or cardiac-specific troponin level	1
ST deviation ≥0.5 mm	1
Prior coronary artery stenosis ≥50%	1

Calculated TIMI Risk Score:

Risk Status	Risk of ≥1 Primary End Point* in ≥14 Days
0 or 1	5% Low
2	8% Low
3	13% Intermediate
4	20% Intermediate
5	26% High

*Primary end points: death, new or recurrent MI, or need for urgent revascularization

Patients without high risk features may be managed with a conservative strategy that does not include routine invasive assessment with coronary angiography and PCI where indicated. (LOE I).

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