



ST-elevation myocardial infarction: New Zealand management guidelines

ST-Elevation Myocardial Infarction Guidelines Group and the New Zealand Branch
of the Cardiac Society of Australia and New Zealand

Glossary

ACS	Acute coronary syndromes
ACE	Angiotensin converting enzyme
CAPTIM	Comparison of Angioplasty and Prehospital Thrombolysis in Acute Myocardial Infarction (CAPTIM) study
ECG	Electrocardiogram
ED	Emergency department
FT	Fibrinolytic therapy
GRACIA	Grupo de Análisis de la Cardiopatía Isquémica Aguda (GRACIA) trial
IRA	Infarct related artery
LBBB	Left bundle branch block
LV	Left ventricular
MI	Myocardial infarction
PCI	Percutaneous coronary intervention
PRAGUE	Primary Angioplasty in AMI Patients From General Community Hospitals Transported to PTCA Units Versus Emergency Thrombolysis (PRAGUE-2) study
SHOCK	Should we Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) trial
STEMI	ST-elevation myocardial infarction
TIMI	Thrombolysis in myocardial infarction

Purpose

These guidelines apply to the management of patients with ST-elevation myocardial infarction (STEMI). The purpose of these guidelines are to provide a summary of the most up-to-date New Zealand and overseas evidence and to make recommendations based on the evidence that will lead to the best practice for patients with STEMI in New Zealand. The guidelines are aimed at all health providers who care for patients with STEMI.

For a detailed description of the levels of evidence cited in these guidelines, please see Appendix 2. These guidelines are intended for best clinical practice. Where physicians or hospitals are not able to meet the guidelines it is important that there is documentation that there have been communications between clinicians and managers clearly defining the clinical implications of any resource shortages.

Management

Sustained ST segment elevation on the electrocardiogram (ECG) in the context of an acute coronary syndrome (ACS) is usually indicative of an occluded epicardial artery. Included within this subset are those patients presenting with presumed new left bundle branch block pattern on the initial ECG.

When patients first present with ischaemic symptoms (chest pain or a surrogate) lasting for more than 20 minutes, their management (Figure 1) depends on whether

ST elevation is present on the ECG or not. If the symptoms are within 12 hours, urgent reperfusion; fibrinolytic, or catheter-based reperfusion is mandatory.1++A (See Appendix 2 for explanation)

Good nursing care is a very important component of the care of patients with STEMI.

Diagnosis

The diagnosis of STEMI is defined as ST elevation of ≥ 2 mm in chest leads V_{1-3} or ≥ 1 mm in 2 contiguous leads or presumed new left bundle branch block (LBBB).

Investigations

ECGs

An ECG should be performed and reviewed immediately on first assessment. All patients with inferior infarctions must have V_{3R} and V_{4R} leads recorded to detect right ventricular infarction. Posterior recordings (V_{7-9}) should be performed if a posterior infarction is suspected.

If the initial ECG is normal, and there is a high clinical suspicion of ongoing MI, serial ECGs should be performed at 5 to 10 minute intervals and optimally continuous ST segment monitoring should be performed. ECGs should be obtained every 6 to 8 hours in all other patients until an established diagnosis has been made.D4

The criteria required are:

- New or presumed new ST-elevation at the J point in 2 or more contiguous leads.
- ST elevation ≥ 0.2 mV (2 small squares) in leads V_1 , V_2 , or V_3 , **or** ≥ 0.1 mV (1 small square) in other leads.

Patients who present with a history consistent with acute myocardial ischaemia and have an ECG with new or presumed new LBBB should be classified and managed as a STEMI. In this context, the presence of one of three of the following ECG criteria adds independent diagnostic value:

- ST elevation ≥ 0.1 mV in leads with a positive QRS complex.
- ST depression ≥ 0.1 mV in V_1 to V_3 .
- ST elevation ≥ 0.5 mV in leads with a negative QRS complex.

The ECG leads in which ST segment changes occur are helpful in localising the regions of ischaemia of the left ventricular myocardium and this in turn can help predict the culprit coronary artery involved:

- Anterior wall ischaemia $\rightarrow V_2$ to V_4 .
- Anteroseptal ischaemia \rightarrow Leads V_1 to V_3 .
- Apical or lateral ischaemia \rightarrow Leads V_4 to V_6 .
- Inferior wall ischaemia \rightarrow Leads II, III, and aVF.

- Posterior wall ischaemia → ST depression in leads V₁ and V₂ with upright T waves or (more sensitively) presence of ST elevation in posterior chest leads (V₇-V₉).

Q waves:

- Development of any Q wave in leads V₁ through V₃, **or**
- The development of a Q wave ≥30 ms (0.03 s) in leads I, II, aVL, AVF, V₄, V₅, V₆.
- Q-wave changes must be present in 2 contiguous leads and be ≥1 mm in depth.
- “Q-wave equivalent” in a posterior MI is the presence of a dominant R wave in leads V₁ and V₂.

The absence of ST elevation or a new LBBB pattern does not exclude the presence of complete epicardial coronary artery occlusion, but the benefit of reperfusion has not been demonstrated among these patients. With a posterior MI due to circumflex artery occlusion there may be marked ST segment depression in leads V₁ to V₄ associated with tall R waves and upright T waves in the right precordial leads (V₁ to V₃).

In patients with STEMI, initiation of reperfusion therapy based on the initial ECG should take priority over cardiac marker analysis. Subsequent confirmation of MI can be determined by initial and subsequent biomarker levels.

Cardiac biomarkers

Blood samples for measurement of troponin levels, which are the preferred cardiac markers for ACS,¹ should be obtained within 10 minutes of presentation.**D4**

Measurement should be repeated 6 to 8 hours later particularly if the baseline level was normal. Troponin levels assessed 8 hours after admission will detect most MIs but requires 12 hours after the onset of symptoms to detect all MIs.

Many patients who present within 3 to 6 hours of the onset of symptoms will have normal troponin and CKMB levels. Myoglobin levels will usually be elevated in these patients and maybe of clinical value to guide management when there is uncertainty about the diagnosis of STEMI particularly in patients with LBBB.

Due to its rapid rise and fall CKMB is preferred over troponin T or I (which may remain elevated for 2 weeks) for the diagnosis of re-infarction.

Troponins (which are the most specific cardiac markers), however, may be elevated in conditions other than an ACS.

Other blood tests

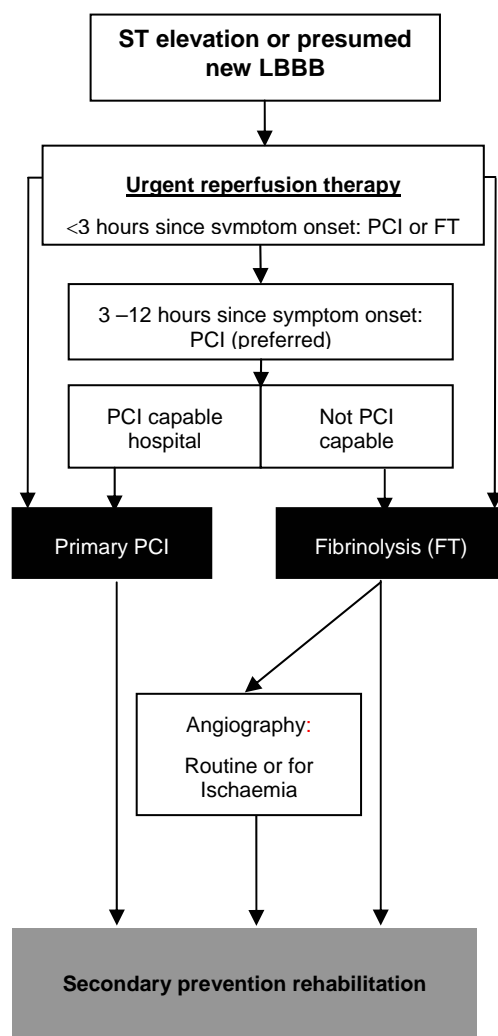
Blood should also be obtained for FBC, electrolytes, glucose, liver function, renal function and lipids. A CXR should be performed (but in the absence of clinical features suggesting aortic dissection or other differential diagnoses), not before initiation of treatment.**D4**

Echocardiography

On occasions acute echocardiography demonstrating a regional wall motion abnormality may be a useful adjunct for diagnosis and assessment of complications

such as ventricular septal defect, sub acute rupture and LV thrombus. In situations where left bundle-branch block is present on the initial electrocardiogram an early rising cardiac marker such as myoglobin may aid diagnosis.**D4**

Figure 1. Management of ST-elevation acute coronary syndromes



Management

Early risk stratification

There are a number of risk scores. The TIMI STEMI risk score for early risk stratification in patients with STEMI is used most commonly for predicting mortality 30 days after the MI (Table 1).² Risk assessment plays an important role in predicting patient prognosis and initiating appropriate evidence-based therapies in patients who are most likely to benefit from them.^{D4}

Reperfusion therapy in patients with ST elevation

Urgent reperfusion of the ischaemic myocardium by restoration of flow in the occluded epicardial coronary artery is the primary therapeutic goal in patients with STEMI who present within 12 hours of symptom onset. If reperfusion therapies are initiated early after symptom onset, the infarctions are smaller, complications are

reduced and survival benefit is greater. When epicardial flow is restored within 30 minutes of occlusion infarction can be aborted.

If flow is achieved within 3 hours, considerable myocardial salvage can occur with beneficial effects on ventricular function and mortality with additional long-term benefit from the presence of an open infarct related artery (IRA). When reperfusion is achieved after 3 hours myocardial salvage is progressively reduced and recovery of ventricular function is dependant on established collateral flow. Beyond 6 hours myocardial salvage is minimal or absent with the major benefit being that related to an open IRA.³ Reperfusion can be achieved using a strategy of fibrinolysis or primary PCI. Door to balloon times should be <90 minutes.1++A

Primary PCI vs fibrinolysis

Infarct artery patency rates at 90 minutes with PCI are superior to fibrinolysis (90% vs 60%). In a recent meta-analysis of 23 trials comparing PCI to fibrinolysis (which included the SHOCK trial which compared stabilisation with immediate revascularisation for cardiogenic shock).^{4,5} PCI appeared superior in reducing short-term mortality, reinfarction, and stroke. However, for patients presenting very early after symptom onset the outcomes with fibrinolysis may be superior.

In the PRAGUE⁶ study for patients randomised to receive streptokinase within 3 hours of symptom onset, the 30-day mortality was similar to that with primary PCI. Theoretically, based on the GUSTO-1⁷ results, where patients treated with tPA had a 1% absolute reduction in mortality compared with patients treated with streptokinase, an accelerated tPA regimen could achieve a 1% lower (absolute) mortality than PCI. In the CAPTIM⁸ study patients who received (pre-hospital) fibrinolysis within 2 hours of symptom onset had improved outcomes compared with primary PCI. Thus for patients presenting <3 hours after symptom onset fibrinolytic therapy may be the treatment of choice for mortality reduction. For patients presenting >3 hours and <12 hours after symptom onset primary PCI appears to be superior and is the reperfusion therapy of choice.

The choice of strategy adopted at any given institution, however, depends on a number of factors (Table 2).

Elderly patients

The optimal reperfusion strategy in the elderly is not defined.^{7,9} In a recent re-analysis of the Fibrinolytic Trialists Enrollment Group overview of patients >75 years there was a significant 15% relative reduction in mortality equating to 34 lives saved per 1000 patients treated; larger than the 16 lives saved per 1000 in patients aged <55 years.¹⁰ Although the risk of intracranial haemorrhage increases with age, most elderly patients who suffer an intracranial haemorrhage die (and are not counted in the mortality benefit) and the risk of non-fatal strokes with major disability occurring is small.¹⁰ In patients up to the age of 84 tPA has been shown to be superior to streptokinase for reducing the composite of mortality and non-fatal disabling stroke.⁷

Choice of fibrinolytic

In the absence of contraindications (Table 3) a fibrin specific agent (tPA, TNK, rPA (see Table 4) is most effective in patients <84 years,1++A⁷ or patients previously

administered streptokinase because of formation of antibodies and concerns about lack of efficacy.^{2++B¹¹} Streptokinase is cheaper but less effective than fibrin specific agents (Table 4). If cost is an issue, streptokinase is a suitable alternative for some patients.

Streptokinase is a vasodilator and may cause hypotension in 10% of patients. This should be managed by head down tilting, with consideration to giving iv sodium chloride 0.9% 250 mL boluses x 2-3. Allergic or febrile reactions to streptokinase may also occur and should be treated with hydrocortisone 100 mg iv and/or promethazine 12.5–25 mg iv stat. For severe anaphylaxis adrenaline sc/iv should be administered (which is otherwise absolutely contraindicated in the setting of an acute MI because of the risk of VF).

If major haemorrhage occurs with either streptokinase or tPA: apply local pressure and if appropriate consider reversing the effects of heparin with protamine and administering 1–3 units fresh frozen plasma.

Monitoring

Continuous ECG monitoring should be performed during infusion of fibrinolytic therapy and 12-lead ECGs should be recorded to give assessment of ST segment recovery. These are recommended to be performed at 90 minutes and 3 hours after first starting the infusion of fibrinolytic therapy.^{D4} To obtain a peak troponin level as a guide to infarct size troponins should be measured at least twice in the 24 hours and at 24–36 hours CKMB should be measured at ≈24 hours to aid detection of reinfarction as troponin T levels remain elevated for 10–14 days.^{D4}

Adjunctive therapies

Oxygen should be administered to keep the saturations around 96% (higher doses of oxygen increase afterload via arterial vasoconstriction. Sublingual GTN and morphine should be administered for pain relief (observe BP and RR) or fentanyl. IV antiemetics should be given with morphine (metoclopramide 10 mg or cyclizine 25 mg).^{D4}

Aspirin

All patients should immediately receive aspirin 150–300 mg which should be chewed if enteric-coated and 75–150 mg continued indefinitely (if there are no contraindications). This recommendation is based on the collaborative meta-analysis of randomised trials of antiplatelet therapy showing no relation of dose with efficacy¹² and information from other studies showing increased bleeding with increasing aspirin doses.^{1++A¹³}

Clopidogrel

Administration of clopidogrel should be considered at presentation in all patients with STEMI. For patients treated with fibrinolysis who are <75 years it is recommended that a loading dose of 300 mg followed by 75 mg daily be given for 2 weeks.¹⁴ For patients over 75 years receiving fibrinolysis or patients of any age not receiving fibrinolysis, consideration should be given to commencing 75 mg of clopidogrel at presentation and continued for 2 weeks.^{1++A¹⁵}

For patients undergoing primary PCI clopidogrel can be given (with a loading dose of 300 mg–600 mg) at presentation or after defining the coronary anatomy and continued for up to 12 months depending on the type of stent used.^{1+A}

Clopidogrel is relatively expensive but several studies have shown it to be cost effective.^{16–18} It is not currently funded except for patients undergoing stenting.

Table 1. Risk stratification of STEMI patients using the TIMI – STEMI risk score² (*Referenced to average mortality [95% confidence intervals])

TIMI Risk Score for STEMI	
History	
Age 65–74	2 points
≥75	3 points
Diabetes, hypertension or angina	1 point
Examination	
SBP <100	3 points
HR >100	2 points
Killip II-IV	2 points
Weight <67 kg	1 point
Presentation	
Anterior STE or LBBB	1 point
Time to treatment >4 hours	1 point
Risk score = Total	(0-14)
Risk Score	Odds of death by 30D*
0	0.1 (0.1-0.2)
1	0.3 (0.2-0.3)
2	0.4 (0.3-0.5)
3	0.7 (0.6-0.9)
4	1.2 (1.0-1.5)
5	2.2 (1.9-2.6)
6	3.0 (2.5-3.6)
7	4.8 (3.8-6.1)
8	5.8 (4.2-7.8)
>8	8.8 (6.3-12)

Table 2. Preferred reperfusion strategy for STEMI

Primary PCI preferred	Thrombolysis preferred
Primary PCI capable catheterisation laboratory available (ED to balloon time <90 minutes, appropriate operator and team experience) Duration of symptoms \geq 3hours Cardiogenic shock Significant heart failure Killip \geq 3 Contraindications to fibrinolysis (Table 3)	Primary PCI capable catheterisation laboratory not available Duration of symptoms <3hours (and delay to laboratory) Difficult vascular access

Table 3. Contraindications to fibrinolysis

<p>Absolute contraindications</p> <ul style="list-style-type: none"> • Any prior intracranial haemorrhage • Known structural cerebral vascular lesion • Known malignant intracranial or spinal neoplasm or arteriovenous malformation • Ischaemic stroke within 6 months • Neurosurgery within 6 months • Suspected aortic dissection • Active bleeding or bleeding diathesis (excluding menses) • Significant closed-head or facial trauma within 3 months • Uncontrolled hypertension on presentation (SBP >180 mmHg or DBP >110 mmHg) • Recent internal bleeding within 6 weeks • Major surgery or major trauma <2 weeks
<p>Relative contraindications</p> <ul style="list-style-type: none"> • Transient Ischaemic Attack <6 months • Traumatic cardiopulmonary resuscitation <2 weeks • Non compressible vascular puncture • Pregnancy • Active peptic ulcer • Current use of anticoagulants with an international ratio >2: the higher the INR, the higher the risk of bleeding

Table 4. Fibrinolytic agents

Variable	Streptokinase	Alteplase tPA*	Reteplase rPA	Tenecteplase TNK-tPA
Molecular weight	47,000	70,000		
Administration	Infusion (1.5 MU over 30 minutes)	Infusion (weight based up to 100 mg over 90 minutes)	10 units over 2 min repeated after 30 minutes	Weight adjusted bolus 30-50 mg over 5-10 seconds
Fibrin specific	No	Yes++	Yes+	Yes+++
Systemic fibrinogen depletion	+++	+	++	Minimal
Bleeding (non-cerebral)	+++	++	++	+
Haemorrhagic stroke	+	++	++	++
Antigenic	Yes	No	No	No
Hypotension with administration	Yes	No	No	No
TIMI grade 3 flow at 90 minutes (%)	32	54	60	63
Cost	+	++	+++	++++

*Bolus 15 mg infusion 0.75 mg/kg over 30 minutes (maximum 50 mg), then 0.5 mg/kg not to exceed 35 mg over the next 60 minutes to an overall maximum of 100 mg

Antithrombotic therapy

Heparin

Adjunctive heparin therapy is recommended to be administered immediately with tPA and as an option with streptokinase. (Some hospitals may prefer not to use heparin with streptokinase particularly if patients are elderly and/or have a small inferior MI).

The dose of heparin has recently been adjusted downwards because of concerns about the risks of intracranial haemorrhage with fibrinolytic therapy and a bolus of UFH of 60 units/kg (maximum 4000 units) followed by an infusion of 12 units/kg/h (maximum 1000 units/h) with the infusion rate adjusted according to the APTT at 3h and the heparin infusion adjusted to achieve an APTT of 50-70sec is used.¹⁹ The infusion should continue for 48 hours.

Bedside APTTs are strongly preferred because of the immediate availability of the result. Other antithrombotics such as enoxaparin, fondaparinux and bivalirudin are currently being tested in clinical trials.

Glycoprotein IIb/IIIa inhibitors

These are not recommended with fibrinolytic therapy. For PCI and stenting abciximab is recommended.²⁰

β -blockers

IV β -blockers are recommended to be considered for administration immediately or following initiation of fibrinolysis in patients who are haemodynamically stable without heart failure (Killip III or IV) and without contraindications (asthma, systolic BP <110 mmHg, heart rate <50 minutes, Mobitz Type II 2nd degree or 3rd degree heart block)²¹ and oral therapy continued indefinitely—e.g. metoprolol 5 mg IV bolus every 2 minutes up to 15 mg followed 15 minutes later by 50 mg orally or atenolol 10 mg IV bolus followed 15 minutes later by 50 mg orally. For patients with heart failure it is recommended that β -blockers (carvedilol or metoprolol) be begun when the patient is stable for 24-48 hours.^{1++A}^{22,23}

ACE inhibitors

All patients with evidence of heart failure, anterior infarction or a history of previous infarction should be considered to receive oral ACE inhibitors beginning 2 hours after admission if the systolic BP is >100mmHg usually commencing with a low dose of a short acting drug and then increasing over several days to maximally tolerated doses.^{1++A}^{24,25} In all other patients ACE inhibitors are recommended to be begun on day 1 and continued long term.^{1++A}

If a choice has to be made between β -blockers and ACE inhibitors because of hypotension, ACE inhibitors are the preferred initial therapy (because of their effect on remodelling). If patients are intolerant of ACE inhibitors they should be started on an angiotensin receptor blocker.²⁶

Lipid modifying therapy

Initiation of statin therapy should be begun in-hospital (e.g. simvastatin 40 mg or atorvastatin 80 mg) with an aim to reduce the LDL to 1.6 mmol/L.1++A²⁷⁻²⁹

Nitrates

Nitrates are appropriate for the control of angina and hypertension.D4

Calcium channel blockers

There is no evidence that calcium antagonists improve prognosis following myocardial infarction, but they can be used for symptomatic angina in combination with a beta-blocker. Heart rate limiting calcium channel blockers are preferred if patients cannot tolerate a beta-blocker e.g. verapamil or diltiazem.1-B³⁰

Warfarin

Patients with pedunculated or mobile left ventricular thrombus should be anticoagulated for 3-6 months with repeat echocardiography at this time to determine if continuation of anticoagulation is appropriate. Anticoagulation of patients with low ejection fractions and no LV thrombus is controversial and treatment should be individualised. There is no need to anticoagulate patients with transient AF (<24 hours). Anticoagulation and rate control may be preferred for patients with AF and other risk factors (over 60, hypertension, diabetes, heart failure, EF ≤35%, prior thromboembolism, persistent atrial thrombus) rather than anticoagulation for 3 weeks, and cardioversion (or TOE guided), although for younger patients this may be appropriate. Ongoing warfarinisation may still be appropriate for some patients who are initially returned to sinus rhythm.1++A

Failed fibrinolysis

In patients with continuing ischaemia or haemodynamic instability, after lytic therapy, rescue PCI should be undertaken as soon as possible.D4 Lack of resolution of ST segment elevation by 30% of baseline at 90 minutes is also considered to be indicative of failed reperfusion but has less evidence for its support as an indication for rescue PCI.

Stenting and adjunctive devices for PCI

Stenting is recommended. Drug eluting stents are indicated in small vessels, long lesions, bifurcation or ostial lesions, bypass grafts and patients with diabetes.1++A

Rotablation is recommended for fibrotic or heavy calcified lesions that cannot be adequately dilated before stenting.D4

Distal protection devices are indicated for use in saphenous vein grafts and where there is a high thrombus load.1++A Randomised trials are testing these devices. Other approaches such as aspiration systems are also being tested.

Facilitated PCI

Facilitated PCI is defined as planned PCI soon after fibrinolysis. A number of trials are investigating various fibrinolytic and glycoprotein IIb/IIIa regimens. These approaches cannot be recommended at present.

Angiography in patients who have received thrombolytic therapy

There are two approaches. One is ischaemia-driven if ischaemic symptoms or ischaemic ECG changes occur despite medical therapy or ischaemic changes occur during exercise stress testing or pharmacologic or stress echo imaging. This is the most common approach in New Zealand.

The other approach is to perform angiography in all patients who would be candidates for revascularisation. Routine angiography and PCI as appropriate on significant stenoses may provide prognostic information and enable patients to be discharged home early and reduce ischaemic events post discharge.⁴ This approach is supported by the GRACIA 1 trial where an invasive strategy within 24 hours (median 16.7 hours) of thrombolysis, compared with an ischaemia guided strategy resulted in a lower incidence (9% vs 21%) of the primary end-point of the combined rate of death, reinfarction or revascularisation at 1 year and there was a trend for a reduction in death and MI, 7% vs 12% (RR 0.59, 0.33–1.05).³¹ There are a number of ongoing trials evaluating the most appropriate timing for angiography.

Surgical revascularisation (CABG)

The success of PCI and fibrinolysis for STEMI has meant that the need for urgent surgical reperfusion is limited to a very few select circumstances although it may be used as the primary reperfusion strategy in 2-5% of patients with STEMI:

- Failed PCI (primary or rescue) with ongoing symptoms and/or haemodynamic compromise.
- In patients who require surgical management of severe mitral regurgitation due to ischaemic papillary muscle rupture or repair of ventricular septal rupture, or sub acute rupture.
- Patients who are unsuitable for fibrinolysis or PCI who have persistent or recurrent ischaemia refractory to medical therapy.
- Patients <75 years (and selected older patients without important comorbidity) who develop cardiogenic shock within 36 hours of STEMI, have left main or severe 3 vessel coronary artery disease and can undergo CABG within 18 hours of the development of shock.⁵
- Patients with $\geq 50\%$ stenosis of the left main stem. This may change with increasing experience with left main stem PCI.

Diabetes mellitus

Diabetics are a high-risk group and all patients with a blood glucose >11 mol/L and a suspected or definite MI or unstable angina with an Ischaemic ECG should be treated aggressively with an insulin, glucose and potassium regimen.³² There are several

appropriate regimens. At this stage without a clear optimal strategy, local units are advised to discuss this therapy with colleagues, and to institute a local policy.1-A

Complications of myocardial infarction

The majority of deaths in hospitalised patients with STEMI are due to LV pump failure and mechanical complications. Compared to the pre-reperfusion era, ventricular tachyarrhythmias are now less common.

Mechanical complications

A number of mechanical complications may occur including Mitral regurgitation, ventricular septal defect and free wall rupture—all of which require urgent echocardiography, and may require urgent insertion of an intra aortic balloon pump and often an urgent surgical consultation.

Arrhythmias

Ventricular or atrial arrhythmias are frequent. Local CCUs have standard protocols for treatment.

Ongoing ischaemia

If patients have ongoing ischaemia (an ECG should be obtained during symptoms to document the degree and extent of ischaemia) expeditious angiography should be considered.

Other complications

Delayed complications include post myocardial infarction syndrome and Deep Vein Thrombosis/Pulmonary Embolism.

Cardiogenic shock

The presence of shock due to left ventricular dysfunction following MI implies ischaemia/infarction of a large area of myocardium and is associated with 70–80% in-hospital mortality. Shock is defined as hypotension (BP \leq 90 mmHg or requiring inotropes to keep the BP $>$ 90 mmHg for 30 minutes) unresponsive to fluid loading and usually with associated decreased tissue reperfusion and decreased urine output. The SHOCK trial has shown that mortality is reduced to ~50% when aggressive support measures including administration of fibrinolytic therapy, intra aortic balloon counter-pulsation, mechanical ventilation and early revascularisation are performed.⁵ In patients without important comorbidity the interventional team, including an anaesthetist should be contacted immediately and oxygen, appropriate ventilation and inotropic support should be begun immediately and emergency angiography should be undertaken.1++A For patients where PCI is not appropriate, surgery should be considered.1++A³³

Heart failure

Frusemide should be given to decrease breathlessness. All patients should be placed on evidence based therapies including ACE inhibitors,²⁴ spironolactone^{34,35} and β -blockers.^{22,23} If patients are intolerant of ACE inhibition an ARB should be prescribed.1++A Enoxaparin 50 mg/day should be given to prevent deep vein

thrombosis and pulmonary embolism.1+A Digoxin should be considered for patients in sinus rhythm who continue to be symptomatic as it has been shown to decrease rehospitalization.1+A³⁶

Right ventricular infarction

Right ventricular infarction is usually diagnosed clinically or by ST elevation in right precordial ECG leads or on echocardiography. Patients may have raised jugular venous pressure, hypotension and clear lung fields. It is important that all patients get adequate fluids—i.e. at least 2 litres in the first 24 hours. If patients are hypotensive a fluid challenge should be given—e.g. 200 mL of IV saline over 10–15 minutes. Swan Ganz catheterisation may help monitor volume status.4D

Reinfarction

Approximately 2–6% of patients experience reinfarction in hospital and this is associated with increased mortality and more frequent heart failure, cardiogenic shock and ventricular arrhythmias. Urgent PCI should be considered. In hospitals without PCI facilities readministration of a fibrin-specific agent (50% of dose in first 24 hours) should be considered, followed by urgent transfer for PCI.1+A³⁷

Testing for inducible ischaemia

All patients who have not had angiography or have had incomplete revascularisation should undergo testing for inducible ischaemia—e.g. by treadmill, stress echo, or nuclear imaging prior to hospital discharge.D4

Echocardiography

Pre-discharge assessment of left ventricular function is necessary to assess left ventricular function in all patients if this has not been assessed by other means.D4

Holter monitoring

Routine Holter monitoring is not recommended.D4

Resource availability

It is recognised that in New Zealand there are limitations on resources. Pharmacoeconomic analyses are planned to determine the appropriate levels of funding in New Zealand for patients with STEMI.

Rehabilitation

All patients should be referred to the Rehabilitation Service and be encouraged to attend rehabilitation programmes, to stop smoking, undergo regular exercise, (30 minutes of brisk walking or equivalent on most days of the week), to achieve ideal weight, and to have a cardioprotective diet, and to aid compliance with medications.1++A

Advice about return to work and sexual activities should be tailored to the individual patient. The National Heart Foundation of New Zealand has several excellent patient information brochures. For driving guidelines, refer to page 56 of the “Medical Aspects of Fitness to Drive” book issued by the Land Transport Safety Authority.

Conclusion

It is very important that patients with STEMI receive some form of reperfusion therapy as quickly as possible along with the other evidence based therapies and that access across New Zealand be equitable, particularly for Maori. These guidelines are the evidence base for best practice management of patients with ST-elevation acute coronary syndromes.

Author information: ST-Elevation Acute Coronary Syndrome Guidelines Group (refer to Appendix 1 below), nationwide; The New Zealand Branch of The Cardiac Society of Australia and New Zealand, Wellington.

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Appendix 2

LEVELS OF EVIDENCE	
I++	High quality of meta-analysis, systematic reviews of RCTs, or RCTs with a very low risk of bias.
I+	Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias.
I-	Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias.

2++	High quality systematic reviews of case-control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is casual.
2+	Well-conducted case-control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is casual.
2-	Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not casual.
3	Non-analytic studies—e.g. case reports, case series.
4	Expert opinion.

Following assessment of the level of evidence for individual papers, recommendations were given a grade from A to D as below. This grading system departs from the Scottish Intercollegiate Guidelines Network (SIGN) system which was derived primarily for treatment guidelines and revises ranking according to therapy or prognosis. Questions relating to prognosis were considered a feature of this guideline to determine how to tailor cardiac rehabilitation services according to individual patient needs. For further details on the SIGN system see www.sign.ac.uk.

Grades of Recommendation	
A	<p>For therapy: At least one meta-analysis, systematic review, or RCT rated as 1++, and directly applicable to the target population, OR</p> <p>A body of evidence consisting principally of studies rated as 1+, directly applicable to the target population and demonstrating overall consistency of results.</p> <p>For prognosis: At least one meta-analysis, systematic review, or large high quality cohort study rated as 2++ and directly applicable to the target population, OR</p> <p>A body of evidence consisting principally of studies rated as 2+, directly applicable to the target population and demonstrating overall consistency of results.</p>
B	<p>For therapy: A body of evidence including studies rated as 2++, directly applicable to the target population, and demonstrating overall consistency of results, OR</p> <p>Extrapolated evidence from studies rated as 1++ or 1+.</p> <p>For prognosis: A body of evidence including studies rated as 2+, directly applicable to the target population and demonstrating overall consistency of results.</p>
C	<p>For therapy: A body of evidence including studies rated as 2+, directly applicable to the target population and demonstrating overall consistency of results, OR</p> <p>Extrapolated evidence from studies rated as 2++</p>
D	<p>Evidence levels 3 or 4, OR</p> <p>For therapy: Extrapolated evidence from studies rated as 2+, or expert opinion.</p>

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