This guideline is meant to be a guide for clinical practice, based on the best available evidence at the time of development. Adherence to this guideline may not necessarily guarantee the best outcome in every case. Every health care provider is responsible for the management of his/her unique patient based on the clinical picture presented by the patient and the management options available locally.

**Period of validity**
This update was issued in 2014 and will be reviewed in 2019 or sooner if new evidence becomes available.

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Electronic version available on the following website:
http://www.moh.gov.my
http://www.acadmed.org.my

*This document is an update and supersedes the previous CPG on STEMI (2007).*
Message from the Director General of Health

ST segment myocardial infarction (STEMI) is the most deadly among the clinical presentations of acute coronary syndrome (ACS). Unlike many of medical conditions, STEMI is associated with high mortality and morbidity in its early stages, including sudden death. The National Cardiovascular Disease (NCVD) ACS Registry revealed that there was a 10% mortality rate for patients who were admitted to hospital with STEMI in 2006-2008.

Since the publication of the 2nd Edition of the Malaysian Clinical Practice Guidelines (CPG) for the management of STEMI in 2007, there have been advances in the way STEMI is managed by the medical community, and more specialised centres providing leading-edge care for this condition. Therefore, it is timely for the publication of this 3rd edition of the Malaysian CPG for STEMI.

It is testament to the commitment of the team of experts involved who are continuing to pursue the update of this and other CPGs. The pace of innovation in medicine has relentless in recent years, especially so in the area of cardiovascular medicine, in terms of both medical and interventional management.

There is a continual effort to not only update the CPGs but also improve the process of selecting and evaluating evidence. In addition, the incorporation of local data will make Malaysian CPGs more relevant document in the local context. I envisage many young physicians, generalists and allied health professionals will use these CPGs as a useful reference.

I acknowledge that this is not an insignificant effort given other clinical and leadership responsibilities of all those directly involved, and thank the team of authors and reviewers for their time and valuable contributions. I believe this CPG will be an invaluable document for healthcare providers involved in the management of STEMI and subsequently to improve health outcomes associated with this deadly condition.

(Datuk Dr. Noor Hisham bin Abdullah)
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Rationale:
Acute Myocardial Infarction (AMI) continues to be a major health problem for Malaysians due to a more sedentary lifestyle compared to past years, increasing trends of hypertension, diabetes, obesity and dyslipidaemia. There is also a high prevalence of smoking.

The 1st CPG on STEMI was published in 2001 with a 2nd edition in 2007. Rapid further developments have taken place since then.

This 3rd edition of the CPG on STEMI was developed to provide a clear and concise approach based on current evidence. We have summarised and adapted relevant clinical trial data and published literature to the local practice.

This CPG has been prepared by a panel of committee members from the National Heart Association of Malaysia (NHAM) and Ministry of Health (MOH). The committee members comprised of cardiologists, general and family physicians from the government, private sector and universities.

Objectives:
These guidelines are intended to provide awareness and education in order to reduce the morbidity and mortality associated with STEMI by:

- Early recognition and appropriate timely reperfusion strategies.
- Evidence-based management.
- Effective secondary prevention measures to prevent recurrence.

Process:
The previous CPG published in 2007 was used as a base. In addition to the previous clinical questions that needed to be updated, the Expert Panel formulated new questions that needed to be addressed. These clinical questions were then divided into sections and each member was assigned one or more topics.

A review of current medical literature on STEMI from 2007 (the date of the last CPG) until 31st August 2013 was performed. Literature search was carried out using the following electronic databases – PubMed and Cochrane Database of Systemic Reviews. The following MeSH terms or free text terms were used either singly or in combination:

“Myocardial Infarction”, “Acute Myocardial Infarction”, “STEMI”, “ST Elevation Myocardial Infarction” [MeSH]

The search was filtered to clinical trials and reviews, involving humans and published in the English language. The relevant articles were carefully selected from this huge list. In addition, the reference lists of all relevant articles retrieved were searched to identify further studies. Experts in the field were also contacted to obtain further information. International guidelines on STEMI- the American Heart Association (AHA)/American Heart Association, European Society of Cardiology etc. were also reviewed.
College of Cardiology (ACC) and European Society of Cardiology (ESC) were also studied. All literature retrieved were appraised by members of the Expert Panel and all statements and recommendations made were collectively agreed by the group. The grading of evidence and the level of recommendation used in this CPG was adapted from the American College of Cardiology/ American Heart Association and the European Society of Cardiology (Page vii).

After much discussion, the draft was then drawn up and submitted to the Technical Advisory Committee for CPGs, Ministry of Health Malaysia and key health personnel in the major hospitals of the Ministry of Health and the private sector for review and feedback.

**Clinical Questions Addressed**

- How do you diagnose STEMI?
- What is the best strategy to treat STEMI patients based on the current evidence available?
  - How to improve early diagnosis and pre-hospital care level?
  - How do you identify high-risk STEMI patients at diagnosis?
  - How to evaluate reperfusion strategies?
  - When to transfer from non-percutaneous coronary intervention (PCI) to PCI centres?
  - How to identify complications and manage appropriately?
  - How to risk stratify post-STEMI patients?
- How to reduce the risk of a subsequent cardiovascular event (secondary prevention)?
  - What is the role of cardiac rehabilitation?
- How to treat the following special groups?
  - Elderly
  - Diabetics
  - Women
  - Chronic Kidney Disease (CKD)
  - What are the follow-up strategies for these patients?

**Target Group:**

These guidelines are developed for all healthcare providers involved in the management of STEMI in adults.

**Target Population:**

These guidelines are developed to treat all adults with STEMI.

**Dr. Robaayah Zambahari**

Chairperson,

*CPG for STEMI, 3rd edition*
Table 1: Levels of evidence and grades of recommendation

<table>
<thead>
<tr>
<th>GRADES OF RECOMMENDATION</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Conditions for which there is evidence and/or general agreement that a given procedure/therapy is beneficial, useful and/or effective.</td>
</tr>
<tr>
<td>II</td>
<td>Conditions for which there is conflicting evidence and/or divergence of opinion about the usefulness/efficacy of a procedure/therapy.</td>
</tr>
<tr>
<td>II-a</td>
<td>Weight of evidence/opinion is in favour of its usefulness/efficacy.</td>
</tr>
<tr>
<td>II-b</td>
<td>Usefulness/efficacy is less well established by evidence/opinion.</td>
</tr>
<tr>
<td>III</td>
<td>Conditions for which there is evidence and/or general agreement that a procedure/therapy is not useful/effective and in some cases may be harmful.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LEVEL OF EVIDENCE</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Data derived from multiple randomised clinical trials or meta analyses.</td>
</tr>
<tr>
<td>B</td>
<td>Data derived from a single randomised clinical trial or large non-randomised studies.</td>
</tr>
<tr>
<td>C</td>
<td>Only consensus of opinions of experts, case studies or standard of care.</td>
</tr>
</tbody>
</table>

Adapted from the American College of Cardiology Foundation / American Heart Association and the European Society of Cardiology

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SUMMARY

- Cardiovascular disease (CVD) is an important cause of death in Malaysia.
- The diagnosis of STEMI is made in the presence of ischaemic type chest pain/ chest pain equivalent and characteristic evolutionary ST elevation in the resting electrocardiogram (ECG) or new onset left bundle branch block (LBBB).
- It should be confirmed by a rise and fall in cardiac biomarkers.
- TIME LOST IS MYOCARDIUM LOST, thus early diagnosis and treatment is important.
- Early management of STEMI involves pain relief, stabilisation of haemodynamics and assessment for reperfusion.
- The occluded infarct-related artery (IRA) should be opened as soon as possible. The appropriate and timely use of some form of reperfusion therapy is more important than the choice of therapy.
- Primary PCI is the reperfusion strategy of choice if it can be done in a timely manner by an experienced operator.
- If primary PCI cannot be performed, then fibrinolytic therapy should be administered with a DNT of less than 30 minutes.
- Concomitant pharmacotherapy includes aspirin, clopidogrel (or prasugrel or ticagrelor), β-blockers, ACE-Is/ARBs and statins.
- Complications of STEMI include arrhythmias, left ventricular (LV) dysfunction and shock.
- High-risk patients should have early coronary angiography with a view to revascularisation. The others should be risk stratified according to the presence or absence of ischaemia, arrhythmias and LV function.
- Secondary prevention is important and includes the use of aspirin, -blockers, Angiotensin converting enzyme inhibitors (ACE-I)/ Angiotensin II receptors blocker (ARB) and statins.
- All patients should be encouraged to undergo cardiac rehabilitation.
**Flow chart 1: Management of patients presenting with STEMI**

**CHEST PAIN/ CHEST PAIN EQUIVALENT**

- Electrocardiography
- Cardiac Biomarkers

**Concomitant initial management includes:**

- Continuous ECG monitoring
- Sublingual glyceryl trinitrate (GTN) (if no contraindication)
- Aspirin
- Clopidogrel
- Analgesia
- Oxygen [if oxygen saturation (SpO₂) < 95%]

**Assessment for reperfusion:**

- **Onset of symptoms:**
  - < 3 hours
  - 3-12 hours
  - > 12 hours

**Preferred option:**

- < 3 hours: Primary PCI** or Fibrinolytic Therapy
- 3-12 hours: Primary PCI***
- > 12 hours: Medical Therapy ± Antithrombotics

**Second option:**

- < 3 hours: Fibrinolytics
- > 12 hours: Primary PCI*

**Subsequent management:**

- Consider PCI within 3-24 hours if fibrinolytics are administered as part of the pharmacoinvasive strategy
- PCI if ongoing ischaemia or haemodynamic instability

**Concomitant Therapy:**

- Antithrombotics*
- β-blockers
- ACE-Is/ ARBs
- Statins
- Nitrates*
- Calcium antagonists*

---

*When clinically indicated

**Preferred option in:**
- high-risk patients
- presence of contraindications to fibrinolytic therapy and/or
- PCI time delay [DBT – DNT] is less than 60 minutes

***If DBT is within 90 minutes
Flow chart 1: Management of patients presenting with STEMI

**Electrocardiography**

**Cardiac Biomarkers**

Concomitant initial management includes:
- Continuous ECG monitoring
- Sublingual glyceryl trinitrate (GTN) (if no contraindication)
- Aspirin
- Clopidogrel
- Analgesia
- Oxygen if oxygen saturation ($\text{SpO}_2$) < 95%

Assessment for reperfusion:
- Onset of symptoms:
  - Preferred option: **Primary PCI**
  - Second option: Fibrinolytic Therapy
- Subsequent management:
  - Concomitant Therapy:
    - *When clinically indicated*
    - **Preferred option in:**
      - High-risk patients
      - Presence of contraindications to fibrinolytic therapy and/or PCI time delay [DBT – DNT] is less than 60 minutes
      - ***If DBT is within 90 minutes

**Table 2: Level of evidence and grade of recommendation for acute therapy of STEMI**

<table>
<thead>
<tr>
<th>RECOMMENDATION</th>
<th>GRADE OF RECOMMENDATION</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary PCI</strong></td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td><em>Strategy of choice if:</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- DBT &lt; 90 minutes.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>- There are contraindications to fibrinolysis.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>- High-risk patients.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td><strong>Fibrinolytic therapy</strong></td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td><em>Strategy of choice if:</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- DBT &gt; 90 minutes.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>- No contraindications to fibrinolysis.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td><strong>Concomitant Pharmacotherapy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Aspirin:</strong> Loading dose of 300 mg followed by maintenance dose of 75 mg – 150 mg daily.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td><strong>Clopidogrel:</strong> Loading dose of 300 mg followed by maintenance dose of 75 mg daily (for at least 1 month).</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td><strong>Prasugrel:</strong> Loading dose of 60 mg followed by maintenance dose of 10 mg (to be administered only prior to primary PCI).</td>
<td>II-a</td>
<td>B</td>
</tr>
<tr>
<td><strong>Ticagrelor:</strong> Loading dose of 180 mg followed by maintenance dose of 90 mg twice daily (bd) to be administered to patients undergoing primary PCI.</td>
<td>II-a</td>
<td>B</td>
</tr>
<tr>
<td><strong>Antithrombotic (heparin/enoxaparin/fondaparinux)</strong> to be given to patients:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Who received fibrin selective lytic agents.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>- With atrial fibrillation (AF).</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>- With mural thrombus.</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>- Routine administration to patients following fibrinolysis.</td>
<td>II-a</td>
<td>B</td>
</tr>
<tr>
<td><strong>β-blockers:</strong> For all patients if no contraindications.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td><strong>ACE-Is:</strong> For all patients with no contraindications.</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td><strong>Statins:</strong> For all patients if no contraindications.</td>
<td>I</td>
<td>A</td>
</tr>
</tbody>
</table>

*Please refer to flow chart 1 for details.*
Table 3: Level of evidence and grade of recommendation for secondary prevention post-STEMI

<table>
<thead>
<tr>
<th>STRATEGY</th>
<th>GRADE OF RECOMMENDATION</th>
<th>LEVEL OF EVIDENCE</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking Cessation</td>
<td>I</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>Exercise</td>
<td>I</td>
<td>B</td>
<td>At least 30-60 minutes most days of the week.</td>
</tr>
</tbody>
</table>

**CONCOMITANT PHARMACOTHERAPY**

<table>
<thead>
<tr>
<th>STRATEGY</th>
<th>GRADE OF RECOMMENDATION</th>
<th>LEVEL OF EVIDENCE</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>I</td>
<td>A</td>
<td>Maintenance dose: 75-150 mg daily.</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>I</td>
<td>A</td>
<td>Maintenance dose 75 mg daily to be given for 1 month following fibrinolytic therapy and for longer periods post-primary PCI.</td>
</tr>
<tr>
<td>β-blockers</td>
<td>I</td>
<td>A</td>
<td>Consider long-term therapy for all patients if no contraindications.</td>
</tr>
<tr>
<td>ACE-Is</td>
<td>I</td>
<td>A</td>
<td>Started on first day and continued long-term for all patients if no contraindications.</td>
</tr>
<tr>
<td>ARBs</td>
<td>I</td>
<td>B</td>
<td>For ACE-I intolerant patients, consider valsartan.</td>
</tr>
<tr>
<td>Statins</td>
<td>I</td>
<td>A</td>
<td>Aim for low density lipoprotein-cholesterol (LDL-C) &lt; 2.0 mmol/L (preferably &lt; 1.8)</td>
</tr>
</tbody>
</table>
## Table 4: Indications for PCI in STEMI

<table>
<thead>
<tr>
<th>INDICATIONS</th>
<th>GRADE OF RECOMMENDATION/LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary PCI</strong> in patients presenting &lt; 12 hours of ischaemic symptoms:</td>
<td></td>
</tr>
<tr>
<td>• &lt; 3 hours and PCI time delay is &lt; 60 minutes.</td>
<td>I, A</td>
</tr>
<tr>
<td>• 3-12 hours in a PCI centre or PCI transfer delay &lt; 2 hours.</td>
<td>I, A</td>
</tr>
<tr>
<td><strong>Primary PCI</strong> in patients presenting 12 to 24 hours of symptom onset with evidence of ongoing ischaemia.</td>
<td>II-a, B</td>
</tr>
<tr>
<td><strong>Primary PCI</strong> in patients who have:</td>
<td></td>
</tr>
<tr>
<td>• High-risk features – section 4.2 (C).</td>
<td>I, B</td>
</tr>
<tr>
<td>• Contraindications to fibrinolytics – section 4.2 (B).</td>
<td>I, B</td>
</tr>
<tr>
<td><strong>Rescue PCI</strong> in patients who have evidence of failed reperfusion of the IRA diagnosed by persistent ST elevation and/or recurrent/ongoing chest pain.</td>
<td>I, A</td>
</tr>
<tr>
<td><strong>Facilitated PCI</strong> is a strategy of immediate PCI &lt; 1 hour after an initial pharmacological regimen (fibrinolytics ± Gp IIb/IIIa inhibitors).</td>
<td>III, A</td>
</tr>
<tr>
<td><strong>Post-fibrinolysis</strong> and:</td>
<td></td>
</tr>
<tr>
<td>• Routine angiography with a view to PCI and stenting between 3-24 hours in all STEMI patients (pharmacoinvasive therapy).</td>
<td>II-a, B</td>
</tr>
<tr>
<td>• Delayed selective angiography depending on presence of haemodynamic instability or residual ischaemia.</td>
<td>I, A</td>
</tr>
<tr>
<td><strong>PCI</strong> of totally occluded vessel 3-28 days after MI and no reversible ischaemia.</td>
<td>III, B</td>
</tr>
</tbody>
</table>
INTRODUCTION

CVD remains an important cause of mortality in Malaysia, accounting for 20-25% of all deaths in public hospitals. This has remained unchanged for the last 10 years. In Malaysia, patients with ACS present at a mean age of 59 ± 12 years, 6 years younger than those in the Global Registry of Acute Coronary Events (GRACE). The in-hospital and 30-day mortality following STEMI is also high at 10% and 14% respectively.

Since the publication of the last guidelines, the following efforts have been intensified in Malaysia:

- Educating healthcare providers and the public on the importance of early diagnosis and treatment of STEMI.
- Increasing the number of cardiovascular treatment centres within the public and private sectors.
- Early institution of reperfusion therapy in the emergency department, with a reduction in DNT (from 60 minutes in 2006 to 45 minutes in 2009 & 2010).
- Increasing the number of primary PCI interventions in PCI-capable hospitals.
- The use of evidence based medicine post-STEMI – almost 90% usage of statins and aspirin.

Despite these efforts, the in-hospital mortality still remains high. This is due to late presentation and occasional misdiagnosis leading to missed opportunity for appropriate reperfusion therapy. In addition, there is a lack of risk stratification resulting in non-recognition of high-risk patients who would otherwise benefit from revascularisation. There should be greater use of dual antiplatelet therapy (DAPT) post-STEMI.

Guidelines are intended to help in the management of patients. All the recommendations stated in this guideline may not be available to all eligible patients. Patient care should be individualised and sound clinical judgement still plays an important role in decision-making.
2 DEFINITION AND PATHOGENESIS

ACS is a clinical spectrum of ischaemic heart disease ranging from unstable angina, non-ST segment elevation myocardial infarction (NSTEMI) to STEMI depending upon the degree and acuteness of coronary occlusion (See Figure 1). In unstable angina, myocardial injury is absent and cardiac biomarkers are normal. In myocardial infarction (MI) [both NSTEMI and STEMI] cardiac biomarkers are raised.

MI is a clinical diagnosis based on the presence of myocardial injury or necrosis as indicated by a rise and fall of serum cardiac biomarkers. In addition, there should be at least one of the following:\(^4\)

i. Clinical history consistent with chest pain of ischaemic origin.
ii. ECG changes of ST segment elevation or presumed new LBBB.
iii. Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
iv. Identification of an intracoronary (IC) thrombus by angiography or autopsy.

This new comprehensive definition of MI which utilises newer cardiac biomarkers and imaging techniques is more sensitive in diagnosing MI.\(^5\)

‘Reinfarction’ is used for MI that occurs within 28 days of the incident event while recurrent MI occurs after 28 days.\(^4\)

![Figure 1: Clinical spectrum of ACS. Adapted from Antman EM, Anbe DT, Armstrong PW et al. “ACC/AHA Guidelines for the management of patients with ST Elevation Myocardial Infarction” at www.acc.org](image-url)
The Task Force for the Universal Definition of MI has classified MI into various types based on pathological and clinical features. \(^4\) (Table 5, page 3)

The current CPG will primarily focus on spontaneous MI (MI type 1) with ST segment elevation on ECG.

**Table 5: Clinical classification of MI\(^4\)**

<table>
<thead>
<tr>
<th>Type 1: Spontaneous MI</th>
</tr>
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<tbody>
<tr>
<td>Spontaneous MI related to atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection with resulting intraluminal thrombus in one or more of the coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis. The patient may have underlying severe coronary artery disease (CAD) but on occasion non-obstructive or no CAD.</td>
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<table>
<thead>
<tr>
<th>Type 2: MI secondary to an ischaemic imbalance</th>
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</thead>
<tbody>
<tr>
<td>In instances of myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or demand, e.g. coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachy/bradyarrhythmias, anaemia, respiratory failure, hypotension, and hypertension with or without left ventricular hypertrophy (LVH).</td>
</tr>
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<table>
<thead>
<tr>
<th>Type 3: MI resulting in death when biomarker values are unavailable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac death with symptoms suggestive of myocardial ischaemic and presumed new ischaemic ECG changes or new LBBB, but death occurring before blood samples could be obtained, before cardiac biomarker could rise, or in rare cases cardiac biomarkers were not collected.</td>
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<table>
<thead>
<tr>
<th>Type 4a: MI related to PCI</th>
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</thead>
<tbody>
<tr>
<td>MI associated with PCI is arbitrarily defined by elevation of cardiac troponin (cTn) values &gt; 5 x 99(^{th}) percentile upper reference limits (URL) in patients with normal baseline values (≤ 99(^{th}) percentile URL) or a rise of cTn values &gt; 20% if the baseline values are elevated and are stable or falling. In addition, either (i) symptoms suggestive of myocardial ischaemia, or (ii) new ischaemic ECG changes or new LBBB, or (iii) angiographic loss of patency of a major coronary artery or a side branch or persistent slow-or no-flow or embolisation, or (iv) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality is required.</td>
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<table>
<thead>
<tr>
<th>Type 4b: MI related to stent thrombosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>MI associated with stent thrombosis is detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac biomarkers values with at least one value above the 99(^{th}) percentile URL.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type 5: MI related to coronary artery bypass surgery (CABG)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MI associated with CABG is arbitrarily defined by elevation of cardiac biomarker values 10 x 99(^{th}) percentile URL in patients with normal baseline cTn values (99(^{th}) percentile URL). In addition, either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.</td>
</tr>
</tbody>
</table>

Adapted from Thygesen K et al. Third universal definition of myocardial infarction. Journal of the American College of Cardiology. 2012; 60(16):1581-98.
2.1 Diagnosis

The diagnosis of STEMI is based on the presence of evolutionary changes of ST elevation in the resting ECG in patients presenting with chest pain or its equivalent and supported by the presence of raised cardiac biomarkers.

2.1.1 History

A thorough, targeted history is important in making the diagnosis of STEMI. Chest pain of STEMI begins abruptly and lasts for more than thirty minutes. It is usually located in the centre of the chest, and may radiate to the jaw or down the left arm. It may occur at rest or with activity. The pain may just be a tightness or heaviness in the chest, but it is usually described as a pressure, squeezing or a severe crushing pain with a sense of impending doom associated with sweating, nausea, vomiting and shortness of breath. The pain may be of a burning quality and localised to the epigastria or interscapular region resulting in a misdiagnosis.

In the elderly, females and patients with diabetes, the index of suspicion has to be high because they may present with atypical symptoms such as unexplained fatigue, shortness of breath, dizziness, lightheadedness, unexplained sweating and syncope. They may not necessarily have chest pain.

Other important points to note in the history are the presence of:

- Previous history of ischaemic heart disease, PCI or CABG.
- Risk factors for atherosclerosis.
- Symptoms suggestive of previous transient ischaemic attack (TIA) or other forms of vascular disease.

Upon clinical suspicion of ACS, a 12-lead ECG should be performed and interpreted immediately within 10 minutes of first medical contact (FMC).

2.1.2 Electrocardiographic changes

The diagnosis of STEMI depends upon the presence of characteristic ECG changes. The presence of ST elevation in two contiguous leads in patients with symptoms of ischaemia is the cardinal feature of STEMI.

The cut-off points for new or presumed new ST segment elevation (in the absence of LVH and LBBB) is the presence of \( \geq 0.1 \text{ mV} \) ST segment elevation in all leads except leads V2-V3. In leads V2-V3, a cut-off point of \( \geq 0.25 \text{ mV} \) (in males < 40 years), \( \geq 0.2 \text{ mV} \) (in males \( \geq 40 \text{ years} \)) and \( \geq 0.15 \text{ mV} \) in females is used.\(^4\)
The presence of a new onset or presumed new LBBB in a patient with typical chest pain of ischaemia may indicate an infarct and should be treated as STEMI.7

Occasionally the ECG may be non-diagnostic. Cardiac imaging techniques and cardiac biomarkers can help make the diagnosis in these difficult situations. (See Appendix I, page 51).

In the early stages of MI, the initial ECG may be normal, equivocal or show hyperacute T-wave changes only. In these patients if the index of suspicion of STEMI is high, the ECG should be repeated at close intervals of at least 15 minutes to look for progressive ST changes. Comparison with previous ECG’s may also be helpful.

Patients with inferior STEMI should have an ECG recording of the right praecordial lead (V4R) to identify concomitant right ventricular (RV) involvement.8 In those with ST segment depression in leads V1-V3, it is advisable to have an ECG recording of the posterior chest wall (V7-V9) to identify a true infero-basal (formerly known as infero-posterior) STEMI. The cut-off point for ST segment elevation in the posterior leads is ≥ 0.05 mV (≥ 0.1 mV in men < 40 years).4

The presence of ST elevation in lead AVR may be a predictor of left main/3 vessel CAD and carries an adverse prognosis.9,10

Table 6: ECG patterns of various STEMI locations11

<table>
<thead>
<tr>
<th>Location</th>
<th>Leads</th>
<th>ECG findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteroseptal</td>
<td>V1 – V3</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Extensive anterior</td>
<td>V1 – V6</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Posterior</td>
<td>V7 – V8</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Posterior</td>
<td>V1 – V2</td>
<td>ST depression, Tall R wave</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>I, AVL, V5 – V6</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>Inferior</td>
<td>II, III, AVF</td>
<td>ST elevation, Q wave</td>
</tr>
<tr>
<td>RV</td>
<td>V4R</td>
<td>ST elevation</td>
</tr>
</tbody>
</table>

2.1.3 Serum cardiac biomarkers

The history and ECG is of paramount importance in making the diagnosis of STEMI and determining the reperfusion strategy. A rise and fall in the levels of serum cardiac biomarkers support the diagnosis of STEMI. One should not however, wait for the results of these biomarkers before initiating reperfusion therapy.

These cardiac biomarkers include:4

- Cardiac Troponin T (cTnT) and Cardiac Troponin I (cTnl).
- Creatine Kinase-Myocardial Band (CK-MB).
- Creatine Kinase (CK).
For the relative timing, rate of rise, peak value, duration of elevation and properties of these cardiac biomarkers following STEMI, see Figure 2 (page 6) and Table 7 (page 7). In patients with clinical features and ECG diagnostic of STEMI, the preferred cardiac biomarker is CK-MB. In this situation, troponins are not necessary since there is already ECG evidence of myocardial injury.

If the clinical features and ECG are suspicious but non-diagnostic of MI, then the preferred biomarkers are troponins - cTn (I or T). The absence of ST elevation on the resting ECG and elevated troponin levels indicates NSTEMI (See Malaysian CPG on the Management of Unstable Angina/NSTEMI, 2011).

Troponins have near absolute specificity and high clinical sensitivity for myocardial necrosis. It rises within 3-4 hours of the onset of MI and is more likely to be positive 6 hours after symptom onset. Troponins are not useful for the detection of reinfarction because it remains elevated for 10-14 days and sometimes longer.

High sensitivity cTn has a negative predictive value of 95% as a single test on admission and almost 100% when repeated after 3 hours. For the usefulness of troponins in patients with CKD, refer to section 8.4. Troponins may also be raised in other conditions (See Appendix II, page 52).

CK-MB (measured by mass assay) is the next best alternative. Values differ between the genders. The measurement should be made at the time of first assessment and repeated 6-9 hours later:

- To document the rise and/or fall exceeding the 99th percentile URL for the diagnosis of MI.
- If the first measurement is non-diagnostic and the clinical suspicion of MI is high.

CK-MB measurements are useful for the diagnosis of reinfarction. In a patient with recurrent chest pain following STEMI, a ≥ 20% increase in the value from the last sample suggests reinfarction.

To ensure the reliability of these tests, each individual laboratory should maintain high quality laboratory practice and confirm the range of reference values in their specific setting.
Aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) levels are not sensitive or specific for acute MI with frequent false positive elevations. The use of these biomarkers should be discontinued. Total CK measurement is also not recommended owing to its poor specificity and large distribution in skeletal muscles.

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Figure 2: Time Course of Elevation of Serum Cardiac Biomarkers after STEMI

Table 7: Properties of serum cardiac biomarkers

<table>
<thead>
<tr>
<th>Protein</th>
<th>First detection*</th>
<th>Duration of detection</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>CK-MB</td>
<td>2 – 3 hours</td>
<td>1 – 2 days</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Troponin I</td>
<td>3 – 4 hours</td>
<td>7 – 10 days</td>
<td>++++</td>
<td>+++++</td>
</tr>
<tr>
<td>Troponin T</td>
<td>3 – 4 hours</td>
<td>7 – 14 days</td>
<td>+++++</td>
<td>+++++</td>
</tr>
<tr>
<td>CK</td>
<td>4 – 6 hours</td>
<td>2 – 3 days</td>
<td>++</td>
<td>++</td>
</tr>
</tbody>
</table>

* Hours after symptom onset.

2.1.4 Other diagnostic modalities
Echocardiography is a particularly useful bedside imaging technique. It is useful in detecting:
- New regional wall motion abnormalities in difficult diagnostic situations.
- Mechanical complications of acute MI e.g. free wall rupture, acute ventricular septal defect (VSD), mitral regurgitation.

Other imaging techniques such as chest radiography, multi-slice computed tomography (MSCT), magnetic resonance imaging (MRI) and radionuclide techniques may be useful investigations in the patient presenting with acute chest pain in difficult diagnostic situations.

They help to:
- Rule out or confirm the presence of acute MI or ischaemia.
- Identify non-ischaemic conditions causing chest pain such as valvular heart disease, peri-myocarditis, pulmonary embolism, aortic dissection and pneumothorax.
3 PRE-HOSPITAL MANAGEMENT
Public awareness about heart disease should be increased so that individuals will seek appropriate treatment early thus reducing time from symptom onset to FMC. Most deaths following STEMI occur in the pre-hospital phase.

In STEMI, it is important to reduce total ischaemic time i.e. the time from symptom onset to the time of institution of reperfusion strategies. Total ischaemic time is a combination of:

- Time from arterial occlusion to symptom onset.
- Time from symptom onset to FMC.
- FMC to initiation of reperfusion strategies (DBT or DNT).

The public should be educated about:

- Symptoms of ACS.
- The importance of seeking early treatment at the nearest hospital.
- The benefits of early treatment – opening the blocked coronary artery as soon as possible so as to limit myocardial damage to the minimum and preserve heart function. “TIME IS MYOCARDIUM.”

Immediate measures to be taken in suspected cases of ACS.

3.1 For the general public
- Seek immediate medical attention at the nearest hospital.
- Call for an ambulance (dial 999, hospital call centre or hospital direct line if known) or get someone to take you immediately to the nearest hospital.
- Do not drive yourself.
- If not on regular aspirin and with no history of allergy, chew and swallow one 300 mg tablet of aspirin immediately. Regular aspirin is preferred over enteric coated aspirin in this situation because of its faster onset of action.

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<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Troponin I</td>
<td>3-4 hours</td>
<td>7-10 days</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Troponin T</td>
<td>4-6 hours</td>
<td>7-14 days</td>
<td>++</td>
<td>+++</td>
</tr>
</tbody>
</table>

* Hours after symptom onset.
3.2 For patients with known coronary heart disease (CHD)

- If the chest pain is suggestive of ACS (See section 2.1.1), take one dose of sublingual GTN and be rapidly transported to the nearest hospital.
- GTN should not be taken within 48 hours of taking phosphodiesterase inhibitors [sildenafil (Viagra), tadalafil (Cialis), vardenafil (Levitra)]. It can result in severe vasodilatation, hypotension and even death.

3.3 For patients with known CHD and history of previous PCI and/or CABG

- Go as soon as possible preferably to a PCI capable hospital provided there is no undue delay.

3.4 For the general practitioner / family physician

- Ask patient to chew and swallow one 300 mg tablet of (non-enteric coated) aspirin.\(^\text{18}\)

- Give sublingual GTN if systolic blood pressure (SBP) is more than 90 mmHg.

- If the ECG shows ischaemic changes, give 300 mg of clopidogrel if available.\(^\text{19, 20}\)

- Wherever possible, set up intravenous (IV) access.

- Give oxygen by mask/nasal prongs if the patient is dyspnoeic and/or SpO2 is < 95%.

- Pain relief with titrated IV opiates [IV morphine 3-5 mg slowly]. IV antiemetic may be administered to prevent opioid induced vomiting.

- Avoid intramuscular injections since this could result in intramuscular haematomas if fibrinolytic agents are subsequently administered.

- Call an ambulance or ask the patient’s relative or friend to send the patient immediately to the nearest hospital.

- Wherever possible, contact the doctor at the hospital so that the patient can be treated promptly on arrival.
3.5 For allied health care personnel

Immediate measures to be taken when there is an ambulance call:

- Note nature of complaint.
- Obtain name of caller, address, important landmarks and telephone number.
- If possible, request that a relative or friend wait at a strategic place to help locate the patient.
- Dispatch an adequately equipped ambulance with trained paramedics immediately.
- Patient should be given oxygen if breathless, sublingual GTN and aspirin (if he/she has not taken) and transported to hospital. The cardiac rhythm should be continuously monitored.

Allied health care personnel should be trained:

- To identify patients at high-risk of developing ACS such as those with prior heart disease, the elderly, presence of multiple cardiovascular risk factors - diabetes, smoking, hypertension, dyslipidaemia, and a family history of premature heart disease.
- To identify patients with STEMI based on history and characteristic ECG changes.
- On strategies to reduce DNT and DBT.
- In basic and advanced cardiopulmonary resuscitation (CPR).

Key Messages:

- The public and allied health care personnel should be educated on the importance of early diagnosis and the benefits of early treatment.
- Patients with suspected STEMI should be given aspirin and clopidogrel.
- These patients should be rapidly transported to the hospital for early institution of reperfusion strategies.
4 IN-HOSPITAL MANAGEMENT

Early management of STEMI is directed at:
• Pain relief.
• Establishing early reperfusion.
• Treatment of complications.

4.1 Initial recognition and management

When the patient with suspected STEMI reaches the emergency department, evaluation and initial management should be prompt (FAST TRACK - RED ZONE) because the benefits of reperfusion therapy are greater the earlier it is instituted.\(^{21-25}\)

A quick targeted history should be taken and vital signs noted. The diagnosis should be confirmed with an ECG, which should be done as soon as possible, preferably within 10 minutes of the patient’s arrival in the emergency department. It is important to monitor the patient continuously for arrhythmias.

Pain should be relieved with titrated IV morphine.

The patient’s suitability for reperfusion by either fibrinolytic therapy or primary PCI should be quickly assessed.

The following should be done immediately and concomitantly in the emergency department (See flow chart 1, page x):

- Assessment and stabilisation of the patient’s haemodynamics.
- Sublingual GTN if chest pain persists (avoid if SBP < 90 mmHg).
- Continuous ECG monitoring.
- 300 mg of non-enteric coated aspirin chewed and swallowed if not given earlier.\(^{18}\)
- Clopidogrel at a dose of 300 mg should be given, if not given earlier.\(^{19, 20}\)
- Alternatively, ticagrelor at a loading dose of 180 mg may be given if primary PCI is being considered.\(^{26, 27}\) Ticagrelor has not been tested with fibrinolytic therapy.
- Oxygen by nasal prongs/facemask if SpO2 is less than 95%.\(^{28}\)
- Venous access established and blood taken for cardiac biomarkers, full blood count, renal profile, glucose and lipid profile. Preferably two IV lines should be set up.
• Pain relief - morphine should be administered IV at 2-5 mg by slow bolus injection every 5-15 minutes as necessary. Watch for adverse events – hypotension and respiratory depression. Anti-emetics (IV metoclopramide 10 mg or promethazine 25 mg) should be given with morphine and 8-hourly as necessary.

• Intramuscular injections should be avoided.

• Assessment for reperfusion strategy.

A patient’s immediate and long term prognosis following STEMI can be predicted by using the Thrombolysis in Myocardial Infarction (TIMI) risk score (Appendix III, page 53) or the GRACE risk score (Appendix IV, pages 54 & 55). The TIMI risk score was specifically developed for patients with STEMI while the GRACE risk score predicts in-hospital and 6-month mortality in patients with ACS. Risk assessment is a continuous process that should be repeated throughout hospitalisation and at the time of discharge.

4.2 Reperfusion strategies
The appropriate and timely use of some form of reperfusion therapy is more important than the choice of therapy.

Early and prompt reperfusion is crucial as TIME LOST is equivalent to MYOCARDIUM LOST.22, 32, 33

Overwhelming data has shown that prompt reperfusion therapy improves survival.22, 32 The local NCVD-ACS registry data show that 73% of eligible patients in Malaysia receive fibrinolytic therapy and 7% receive primary PCI.3 The main reasons for not getting reperfusion is late presentation or having missed the diagnosis of MI.

Primary PCI is superior to fibrinolytic therapy as a reperfusion strategy.34, 35

However in patients who present within 3 hours of symptom onset and are at low-risk, both treatment strategies appear to have similar benefits.36, 37

In the majority of our hospitals, fibrinolytic therapy is more readily available and constitutes the main reperfusion strategy.

If both choices are available, the reperfusion strategy of choice is still primary PCI if it can be done in a timely manner by experienced operators in PCI capable centres.
The following factors are important considerations:

• Time from symptom onset to FMC.
• Time to PCI (time from hospital arrival to balloon dilatation i.e. DBT).
• Time to hospital fibrinolysis (time from hospital arrival to administration of fibrinolytic therapy i.e. DNT).
• Contraindications to fibrinolytic therapy.
• High-risk patients.

The best reperfusion strategy will depend upon:

A) Time from onset of symptoms to FMC

• Early presentation (within 3 hours of symptom onset)

I,A If both treatment options are readily available, they have been shown to be equally effective except for the following situations where primary PCI is the preferred strategy:36, 37

- Fibrinolytic therapy is contraindicated.
- In high-risk patients.
- PCI time delay (DBT minus – DNT) is more than 60 minutes.38

• Late presentation (3 to 12 hours of symptom onset)

I,A Primary PCI is preferred. The DBT should be within 90 minutes if the patient presents at a PCI capable facility.

II-a,B If transferred from a centre with no PCI facilities, DBT should be less than 2 hours (including transfer delay).39

II-a,A If the time delay to primary PCI is longer than as mentioned, then the next best option is to give fibrinolytic therapy and make arrangements to transfer the patient to a PCI capable centre for pharmacoinvasive treatment.40, 41

• Very late presentation (> 12 hours)

I,A Both primary PCI and fibrinolytic therapy are not routinely recommended in patients who are asymptomatic and haemodynamically stable.42,43

I,C However, reperfusion therapy would still be beneficial in patients with persistent ischaemic symptoms, haemodynamic or electrical instability. In this subgroup, primary PCI is the preferred strategy.
B) Contraindications to fibrinolytic therapy

Absolute contraindications

- Risk of intracranial haemorrhage
  - History of intracranial bleed.
  - History of ischaemic stroke within 3 months.
  - Known structural cerebral vascular lesion (e.g. arteriovenous malformation).
  - Known intracranial neoplasm.

- Risk of bleeding
  - Active bleeding or bleeding diathesis (excluding menses).
  - Significant head trauma within 3 months.
  - Suspected aortic dissection.

Relative contraindications

- Risk of intracranial haemorrhage
  - Severe uncontrolled hypertension on presentation (blood pressure (BP) > 180/110 mmHg)*.
  - Ischaemic stroke more than 3 months.
  - History of chronic, severe uncontrolled hypertension.

- Risk of bleeding
  - Current use of anticoagulation in therapeutic doses [International Normalised Ratio (INR) > 2].
  - Recent major surgery < 3 weeks.
  - Traumatic or prolonged CPR > 10 minutes.
  - Recent internal bleeding (e.g. gastrointestinal or urinary tract haemorrhage) within 4 weeks.
  - Non-compressible vascular puncture.
  - Active peptic ulcer.

- Others
  - Pregnancy.
  - Prior exposure (> 5 days and within 12 months of first usage) to streptokinase (if planning to use same agent).44

* The BP should be reduced prior to institution of fibrinolytic therapy
C) **High-risk patients**

High-risk patients include:

- Large infarcts.
- Anterior infarcts.
- Hypotension and cardiogenic shock.
- Significant arrhythmias.
- Elderly patients.
- Post-revascularisation (post-CABG and post-PCI).
- Post-infarct angina.

Primary PCI is the preferred strategy in patients in Category B and C.45-47

The goals of time to reperfusion therapy should be within:

- 30 minutes DNT, 22, 32
- 90 minutes DBT.38

### 4.2.1 Fibrinolytic therapy

Fibrinolytic therapy has been shown to reduce mortality when given within the appropriate time frame.22, 25 When given within 2 hours from time of onset of symptoms (the “golden hour”), it is most beneficial and has been shown to be able to abort the infarction and reduce mortality by up to 50%.22, 36, 48, 49

The DNT should be within 30 minutes.22, 32 Strategies should be put in place to achieve this target. Fibrinolytic therapy should be made available in all hospitals and there should be protocols to initiate it in the emergency department. At present, achieving DNT < 30 minutes is one of the key performance indicators for emergency department personnel in public hospitals.

Pre-hospital fibrinolytic therapy has been shown to achieve faster reperfusion.36, 37

#### 4.2.1.1 Indications

Fibrinolytic therapy should only be given to patients with STEMI. It has no role and may even be detrimental in patients with NSTEMI.50, 51

#### 4.2.1.2 Contraindications

See 4.2 (B).
4.2.1.1.3 Choice of fibrinolytic agent

Presently the agents available in Malaysia are:

Streptokinase

1A This is the most widely used agent. It is not fibrin specific and it results in a lower patency rate of the occluded vessel at 60 minutes than fibrin specific agents.\textsuperscript{52-55} Despite having a lower risk of intracranial haemorrhage, the reduction in mortality is less than with fibrin specific agents.\textsuperscript{52, 55}

1B Streptokinase is antigenic and promotes the production of antibodies. Thus the utilisation of this agent for reinfarction is less effective if given between 3 days and 1 or even 4 years after the first administration.\textsuperscript{44} PCI or fibrin specific agents should then be considered.

Regimen:
- 1.5 mega units in 100 ml normal saline or 5\% dextrose over 1 hour.

Tenecteplase (TNK-tPA)

1B The benefit of using TNK-tPA is that it causes more rapid reperfusion of the occluded artery than streptokinase and is given as a single bolus dose.\textsuperscript{56, 57}

Regimen: TNK-tPA  

<table>
<thead>
<tr>
<th>Weight Range</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 60 kg</td>
<td>30 mg</td>
</tr>
<tr>
<td>60 to &lt; 70 kg</td>
<td>35 mg</td>
</tr>
<tr>
<td>70 to &lt; 80 kg</td>
<td>40 mg</td>
</tr>
<tr>
<td>80 to &lt; 90 kg</td>
<td>45 mg</td>
</tr>
<tr>
<td>&gt; 90 kg</td>
<td>50 mg</td>
</tr>
</tbody>
</table>

This is a weight based regimen and thus there is a risk of bleeding if the weight has been overestimated. In patients over the age of 75, the dose should be reduced by 50\%.\textsuperscript{58}

Heparin or enoxaparin should be given immediately after the completion of fibrinolysis and needs to be given for 48 hours. Subcutaneous (SC) fondaparinux 2.5 mg daily may be given as an alternative for 8 days or till discharge.\textsuperscript{59}

4.2.1.1.4 Indicators of successful reperfusion

There is no sensitive bedside clinical method to reliably detect successful reperfusion. Some useful guides are:

- Resolution of chest pain (may be confounded by the use of narcotic analgesics).
• Early return of ST segment elevation to isoelectric line or a decrease in the height of the ST elevation by 50% (in the lead that records the highest ST elevation) within 60-90 minutes of initiation of fibrinolytic therapy.\textsuperscript{60}

• Early peaking of CK and CK-MB levels.

• Restoration and/or maintenance of haemodynamic and/or electrical stability.

The occurrence of ‘reperfusion arrhythmias’ is not a reliable indicator of successful reperfusion. An exception is accelerated idioventricular rhythm and sudden sinus bradycardia which have been correlated with a patent infarct-related coronary artery after fibrinolytic therapy or primary PCI.\textsuperscript{60}

\textbf{4.2.1.1.5 Failed fibrinolysis}

Failure of fibrinolytic agents to open the occluded IRA is manifested as continuing chest pain, persistent ST segment elevation and haemodynamic instability. These patients are more likely to develop complications such as heart failure (HF) and arrhythmias.

The treatment of choice for these patients is rescue PCI.\textsuperscript{61, 62}

They should not be given a second dose of a fibrinolytic agent. This is because there has been no difference in event free survival demonstrated whether these patients are given a repeat dose of a fibrinolytic agent or are treated conservatively.\textsuperscript{63}

\textbf{4.2.2 PCI}

\textbf{4.2.2.1 Primary PCI}

Primary PCI is the preferred reperfusion strategy in patients with ischaemic symptoms < 12 hours when it can be performed in a timely manner and promptly by experienced operators in centres performing a sufficient number of primary PCI procedures.\textsuperscript{34, 39, 64, 65}

\textbf{4.2.2.2 Transfer of patient}

Transfer of patients with STEMI to PCI capable centres should be considered in the following situations:

• Onset of ischaemic symptoms < 12 hours and fibrinolytic therapy is contraindicated irrespective of time delay from FMC.\textsuperscript{66, 67}

• Cardiogenic shock irrespective of time delay.\textsuperscript{45, 68}
STEMI presenting with acute HF. These patients should be stabilised rapidly and ventilated if necessary prior to transfer. Another option is to give fibrinolytic therapy and transfer the patient within 24 hours for a pharmacoinvasive strategy.

When symptoms have been present between 3 and 12 hours and PCI can be performed within 2 hours (preferably as soon as possible). 39, 64, 65, 69, 70

Failed fibrinolytic therapy or reocclusion post-fibrinolysis (See Rescue PCI under chapter 4.2.2.3). 60-63, 71

As part of a pharmacoinvasive strategy in stable patients who have been given fibrinolytics and an elective PCI can be performed within 3 and 24 hours. 41, 72-76

4.2.2.3 PCI post-fibrinolysis or patients who did not receive fibrinolysis

Following fibrinolysis, or in patients who did not receive fibrinolysis, PCI may be performed in the following situations:

Failed reperfusion or reocclusion after fibrinolytic therapy. 60-63, 71

Cardiogenic shock or acute pulmonary oedema that develops after initial presentation. 67, 68, 77, 78

Stable patients within 3-24 hours post-fibrinolysis as part of a pharmacoinvasive strategy. 42, 72-76, 79

Intermediate or high-risk findings on pre-discharge non-invasive ischaemia testing. 80, 81

Spontaneous or easily provoked myocardial ischaemia such as recurrence of chest pains and/or dynamic ECG changes.

Failed fibrinolytic therapy is manifested as one or more of the following:

- Ongoing chest pains.
- Persistent hyper-acute ECG changes (< 50% resolution of ST elevation in the lead showing the greatest degree of ST elevation at presentation). 60
- Haemodynamic and electrical instability.

Rescue PCI is initiated very early (1 to 2 hours) after failed fibrinolytic therapy.

It is associated with a reduction in HF, reinfarction and a trend towards reduction in mortality but with increased risk of bleeding and stroke. Hence these patients should be individually evaluated. 62, 63
4.2.2.4 Facilitated PCI
This refers to a strategy of planned immediate PCI (< 1 hour) after an initial pharmacologic regimen consisting of a reduced dose of a fibrinolytic agent, glycoprotein (Gp) IIb/IIIa inhibitor or a combination of these agents. The purpose of facilitated PCI was to achieve earlier reperfusion but retain the benefits of primary PCI.

This strategy was associated with increased mortality and major bleeding. It is thus not recommended.82-86

4.2.2.5 Routine angiography and PCI after thrombolysis (pharmacoinvasive therapy)
This refers to stable patients routinely undergoing angiography and PCI 3-24 hours post-fibrinolysis, irrespective of the absence or presence of ongoing myocardial ischaemia and reperfusion status.

Given the advances in PCI and antithrombotic therapy, recent studies show that routine angiography with the intent to perform PCI with stenting between 3 and 24 hours after fibrinolysis improved patient outcomes as compared to symptom or ischaemia guided delayed intervention. This strategy has resulted in a significant reduction in mortality and reinfarction rates without an increase in adverse events.64, 70, 71, 75, 87-89

4.2.2.6 Delayed selective angiography and PCI
Patients with STEMI who have not had coronary angiography within 24 hours should be considered for delayed selective angiography. This refers to a strategy of doing angiography and PCI only if there is spontaneous or inducible ischaemia.

Stable patients who are not at high-risk (Refer to section 4.2 (C) for high risk) and who did not undergo early (<24 hours) angiography should undergo non-invasive ischaemia testing.80, 81 If spontaneous or inducible ischaemia is present, then angiography and appropriate revascularisation should be performed.

Routine PCI of totally occluded coronary arteries 3-28 days after STEMI is not recommended unless there is ischaemia demonstrated.90

4.2.3 Technical considerations and pharmacotherapy during primary PCI
For a favourable outcome, it is important to obtain good TIMI 3 epicardial flow as well as optimum reperfusion of the myocardial microvasculature91, 92 (TIMI myocardial perfusion grade – TMP) (Refer to the Malaysian CPG on the Management of PCI, 2009).
4.2.3.1 Antiplatelet therapy to support primary PCI for STEMI

- Oral aspirin 300 mg should be given before primary PCI.\textsuperscript{93-95}
- In addition, a P2Y12 receptor inhibitor should also be given:
  - Clopidogrel 300-600 mg loading dose to be given as early as possible\textsuperscript{95-97, or}
  - Prasugrel 60 mg loading dose to be given after the coronary angiogram has been performed \textsuperscript{98, 99, or}
  - Ticagrelor 180 mg loading dose to be given as early as possible.\textsuperscript{26, 27}
- Gp IIb/IIIa inhibitors may be considered in selected patients:
  - Abciximab.\textsuperscript{100-102}
  - Tirofiban.\textsuperscript{103, 104}

4.2.3.2 Antithrombotic therapy to support primary PCI for STEMI

- IV unfractionated heparin (UFH) with additional bolus to maintain activated clotting time (ACT) above 275.\textsuperscript{105}
- IV low molecular weight heparin (LMWH) – enoxaparin.\textsuperscript{106}
- IV fondaparinux is not recommended because of the risk of catheter thrombosis.\textsuperscript{59}
- Bivalirudin infusion.

4.2.3.3 PCI access site

- Both femoral and radial access is feasible depending on operator experience.\textsuperscript{107,108}
- Radial access has the advantage of reducing bleeding complications. However when larger devices are necessary and the use of intra-aortic balloon pump (IABP) is anticipated, femoral access may be preferable.

4.2.3.4 Technical tips during procedure

- Primary PCI should be performed on the IRA.
- Complete revascularisation may be attempted on critical lesions in non-culprit vessels in the same procedure when patient is in cardiogenic shock.
- PCI is indicated in a non-infarct artery at a later time when there is evidence of myocardial ischaemia.\textsuperscript{80, 81,109}
4.2.3.5 Drug eluting stents (DES) versus bare metal stents (BMS) for STEMI
Both DES and BMS are effective in the setting of STEMI. Randomised trials have not shown any mortality advantage of DES over BMS. However, DES is associated with lower target vessel revascularisation (TVR) without any effect on all-cause mortality.110, 111

4.2.3.6 Distal embolisation and the use of adjunctive devices and pharmacotherapy
Thrombus burden is usually large if the patient presents late or the IRA is ectatic. Predictors of slow flow and no-reflow (TIMI 0) of the IRA are:112-114
• Vessel diameter ≥ 3.5 mm.
• Treatment of the right coronary artery.
• Higher TIMI thrombus score.
• Angiographic findings such as:
  o “Cut-off” sign (i.e. abrupt occlusion of the epicardial vessel) seen on the coronary angiogram.
  o Persistent contrast stasis just proximal and/or distal to the obstruction.
  o Longer lesions.
  o Thrombus of > 5 mm proximal to occlusion.
  o Floating thrombus.

The following steps can be taken to prevent distal embolisation:
• Aspiration catheter - aspiration of thrombus prior to PCI was associated with improved tissue reperfusion and medium term survival when compared with conventional PCI.115-117 A recent study however did not show any reduction in 30-day mortality with routine IC thrombus aspiration prior to primary PCI.118
• Distal embolic protection - meta-analysis showed that these devices had a neutral effect on mortality.115
• Gp IIb/IIIa inhibitors - abciximab therapy during primary PCI showed short term benefit especially in high-risk patients.119-122 The data on its effect on long-term survival is however conflicting.

4.2.3.7 Management of no reflow
No reflow (TIMI 0) or slow reflow (TIMI 1 and 2) may occur transiently or may persist after primary PCI.

No-reflow may occur as a consequence of:
• Microvascular dysfunction from vasospasm.
• Distal embolisation.
• Intimal dissection/intramural haematoma.
It is associated with poor recovery of LV function and a higher incidence of post-MI complications.

Management includes:
- **IC nitroglycerin.** 100 - 200 µg boluses.
- **IC verapamil** 100 – 200 µg boluses.
- **IC adenosine** 100 – 200 µg boluses.
- **IC nitroprusside** 50 – 100 µg boluses.
- **Others:** IC papaverine, IC nicorandil.

### Key Messages

- Primary PCI is superior to fibrinolysis for STEMI when performed in a timely manner (DBT < 90 minutes and PCI time delay < 60 minutes) at experienced centres.
- When fibrinolytic therapy is administered, the DNT should be less than 30 minutes.
- Whenever possible, patients given fibrinolytic therapy should be considered for a pharmacoinvasive approach (elective angiogram within 3-24 hours post fibrinolysis).

### 4.3 Cardiac Care Unit (CCU) management

#### 4.3.1 General measures

All STEMI patients should be admitted to a CCU or equivalent unit equipped with adequate monitoring facilities.

Following successful reperfusion, uncomplicated cases may be kept for a minimum of 24 hours before transfer to a step-down unit. They can sit out of bed and undertake self-care the next day. Patients with STEMI complicated by significant myocardial damage and arrhythmias need longer bed rest and may need to be kept in the CCU longer.

Sedatives may be useful. Titrated IV opioids may be administered to relieve pain.

Use of bedside commode and assisted bedside washing should be safe in most patients.

The Valsalva manoeuvre has been shown to precipitate dangerous haemodynamic and electrocardiographic changes. Prevention of constipation with stool softeners is encouraged.

#### 4.3.2 Monitoring

The general condition of the patient, vital signs, pulse oximetry and the cardiac rhythm should be continuously monitored following STEMI.
4.3.3 Concomitant therapy

4.3.3.1 Oxygen

- Oxygen is indicated in the presence of hypoxaemia (SpO2 < 95%).\(^{28}\) It is reasonable to administer it to all patients within the first 6 hours.\(^{28}\) Oxygen, via nasal prongs, at 2-4 litres/minute is usually adequate. One should aim to maintain the SpO2 above

4.3.3.2 Antiplatelet agents

A) Aspirin

- Aspirin is indicated in all patients at diagnosis and should be continued indefinitely unless contra indicated.
- The initial dose of 100-300 mg should be followed by a maintenance dose of 75 – 150 mg daily.\(^{18, 97}\)

B) Clopidogrel

- Clopidogrel, when given together with aspirin and fibrinolytic therapy in STEMI, has been shown to reduce the odds of an occluded IRA, death or reinfarction without increasing the risk of bleeding or cerebrovascular accidents.\(^{19, 20, 97}\)
- In patients less than 75 years of age given fibrinolysis, a loading dose of 300 mg may be administered followed by a maintenance dose of 75 mg daily. In older patients, a loading dose of 75 mg may be adequate.\(^{20}\)
- In patients considered for primary PCI, a higher loading dose of 300-600 mg may be necessary.
- DAPT is recommended for at least:
  - 1 month after fibrinolytic therapy.\(^{20}\)
  - 1 month and preferably up to a year following PCI with BMS.\(^{124}\)
  - 12 months following PCI with DES.\(^{124, 125}\)

C) Prasugrel

- It may be preferred as an alternative to clopidogrel in patients with STEMI undergoing primary PCI, particularly diabetics.\(^{98}\)
- The loading dose is 60 mg, and should be given after the coronary angiogram. The maintenance dose is 10 mg/day.\(^{98, 99}\)
- It is not recommended for patients > 75 years old, < 60 kg weight, have a past history of TIA or stroke due to a higher risk of major bleeding.

D) Ticagrelor

- Ticagrelor was shown to significantly reduce cardiovascular endpoints when compared to clopidogrel in patients with STEMI undergoing primary PCI.\(^{26, 27}\) Patients given fibrinolytic therapy were excluded from the trial.
• The loading dose is 180 mg and given early after the diagnosis. The maintenance dose is 90 mg bd.

• This agent is short acting and thus can be used in patients who may need surgery without increasing the risk of bleeding.

• Potential drawback is dyspnoea and transient ventricular pauses during the first week. This was rarely associated with symptoms or need for a pacemaker. Caution should be exercised in patients with heart block.

• There was also a small increase in non-CABG related major bleeding.\textsuperscript{27}

In the trials, both ticagrelor and prasugrel were given for 1 year.\textsuperscript{27, 98}

\textbf{4.3.3.3 Antithrombotic therapy}

These agents are administered to patients who received fibrinolytic therapy and did not undergo PCI.\textsuperscript{59, 126-128} They include: (Table 8, page 24)

• UFH\textsuperscript{126}

• LMWH - enoxaparin\textsuperscript{127, 128}
  o In patients > 75 years of age and with renal impairment (serum creatinine (Scr) > 200 µmol/L in women and > 250 µmol/L in men), UFH is preferable to LMWH.\textsuperscript{128}

• Anti Xa inhibitor - fondaparinux
  o May be given to those patients treated medically including those not receiving fibrinolytic therapy.\textsuperscript{129} In STEMI patients not undergoing primary PCI and not receiving fibrinolytic therapy, fondaparinux was shown to reduce mortality and reinfarction without increasing bleeding and stroke rates when compared to UFH or placebo.\textsuperscript{129}
  o It is associated with an increase in catheter-related thrombus and coronary angiographic complications. Thus, fondaparinux is not recommended as the sole anticoagulant during PCI.\textsuperscript{59}

Currently, newer oral anti Xa inhibitors (rivaroxaban, apixaban) are being evaluated in ACS patients receiving standard therapy. At present, no specific recommendation can be made on these new agents.\textsuperscript{130, 131}

• Gp IIb/IIIa inhibitors - used mainly in the setting of primary or emergency PCI.\textsuperscript{100-104} It is administered IC and may be followed by an infusion in the presence of thrombus or slow/no reflow.\textsuperscript{132, 133}

The dose of antithrombotics should be reduced in the presence of CKD(See section 8.4, page 43).
The benefits of ACE-Is are greatest in patients with:

- HF.
- Anterior infarcts.
- Asymptomatic LV dysfunction [LV ejection fraction (LVEF)] < 40% on echocardiography.

### 4.3.3.5 ACE-Is and ARBs

Early use of ACE-Is (within 24 hours) following STEMI has been shown to improve survival. ACE-Is should be started when the BP is stable and SBP remains above 100 mmHg.

The benefits of ACE-Is are greatest in patients with:

- HF.
- Anterior infarcts.
- Asymptomatic LV dysfunction [LV ejection fraction (LVEF)] < 40% on echocardiography.

### Table 8: Recommended dosages of antithrombotics in STEMI

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
<th>Duration of therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enoxaparin</td>
<td>&lt; 75 years: 30 mg IV bolus, SC 1.0 mg/kg bd</td>
<td>Until hospital discharge</td>
</tr>
<tr>
<td></td>
<td>≥ 75 years: no bolus, SC 0.75 mg/kg bd</td>
<td></td>
</tr>
<tr>
<td>UFH</td>
<td>60 U/kg bolus (max 4000 U)</td>
<td>≥ 48 hours</td>
</tr>
<tr>
<td></td>
<td>Infusion 12 U/kg/h (max 1000 U/h) – to maintain an activated partial</td>
<td></td>
</tr>
<tr>
<td></td>
<td>thromboplastin time (APTT) of 1.5 – 2.5 x control</td>
<td></td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>IV 2.5 mg bolus, followed by SC 2.5 mg once daily (od)</td>
<td>8 days or until hospital</td>
</tr>
</tbody>
</table>

### 4.3.3.4 β-blockers

Current recommendations are to use oral β-blockers in all stable patients without specific contraindications. Patients who cannot tolerate β-blockers initially should be evaluated for suitability prior to discharge.

Contraindications to β-blockers:
1) Bradycardia < 60/minute.
2) SBP < 100 mmHg.
3) Pulmonary congestion with crepitations beyond the lung bases.
4) Signs of peripheral hypoperfusion.
5) Second or third degree atrio-ventricular (AV) block.
6) Asthma or chronic obstructive airway disease
7) Severe peripheral vascular disease.

### Table 9: Recommended dosages of β-blockers in STEMI

<table>
<thead>
<tr>
<th>Type</th>
<th>Initiation dose</th>
<th>Target dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metoprolol</td>
<td>25 mg bd</td>
<td>100 mg bd</td>
</tr>
<tr>
<td>Atenolol</td>
<td>25 mg od</td>
<td>100 mg od</td>
</tr>
<tr>
<td>Propranolol</td>
<td>5 mg tds</td>
<td>80 mg tds</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>3.125 mg bd</td>
<td>25 mg bd</td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>1.25 mg od</td>
<td>10 mg od</td>
</tr>
</tbody>
</table>
In patients who cannot tolerate ACE-Is, the ARB, valsartan, has been shown to have a similar survival benefit in patients post-STEMI with LV dysfunction.\textsuperscript{142}

Contraindications to ACE-I and ARB therapy:
- SBP < 100 mmHg.
- Established contraindications e.g. bilateral renal artery stenosis.

The dose of ACE-I/ARB should be reduced or stopped if there is:
- An increase in \( \text{Scr} \) of \( \geq 30\% \) from baseline within 2 weeks after initiation.
- Persistent hyperkalaemia (> 5.6 mmol/L).

(Refer to 4\textsuperscript{th} edition Malaysian CPG for Management of Hypertension, 2013)

**Table 10: Recommended dosages of ACE-Is and ARBs in STEMI**

<table>
<thead>
<tr>
<th>Type</th>
<th>Initiation dose</th>
<th>Target dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>6.25 mg bd – tds</td>
<td>25 – 50 mg tds</td>
</tr>
<tr>
<td>Ramipril</td>
<td>2.5 mg bd</td>
<td>10 mg od</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5 – 5 mg od</td>
<td>10 mg od</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>5 mg od</td>
<td>10 mg od</td>
</tr>
<tr>
<td>Perindopril</td>
<td>2 mg od</td>
<td>8-10 mg od</td>
</tr>
<tr>
<td>Valsartan</td>
<td>40 mg – 80 mg od</td>
<td>160 mg bd</td>
</tr>
</tbody>
</table>

**4.3.3.6 Mineralocorticoid receptor antagonists**

Mineralocorticoid receptor antagonists when added to \( \beta \)-blockers and ACE-Is, have been shown to reduce mortality and hospitalisations when given to patients post-MI with impaired LV function and mild HF.\textsuperscript{143}

**4.3.3.7 Statins**

All patients with STEMI should be prescribed statins regardless of their baseline cholesterol level. Early and intensive high dose statins in ACS have been proven to produce superior benefits in reduction of major adverse cardiac events.\textsuperscript{144-147}

In a number of studies, high loading or re-loading dose of statins have been shown to be beneficial in preventing peri-procedural MI in ACS patients undergoing PCI.\textsuperscript{147-151}

**4.3.3.8 Nitrates**

The routine use of nitrates has not been shown to have a survival benefit.\textsuperscript{152, 153}

Nitrates can be considered in patients with:
- Continuing chest pain and / or ischaemia.
- HF.
- Hypertension.
In the acute stage, IV nitrates are recommended because of their rapid onset of action, ease of titration and potential for prompt termination in the event of side effects. After the first 48 hours, oral or topical nitrates may be continued in patients with persisting ischaemia and/or HF.

Contraindications to nitrate therapy:
- Hypotension (SBP < 90 mmHg).
- RV infarction (RVI).
- History of phosphodiesterase-5 inhibitor ingestion depending upon the half-life of

Table 11: Recommended doses of nitrates

<table>
<thead>
<tr>
<th>Compound</th>
<th>Route</th>
<th>Dosage</th>
<th>Time of onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerine, GTN</td>
<td>IV</td>
<td>5 – 200 µg/minute*</td>
<td>1 minute</td>
</tr>
<tr>
<td></td>
<td>Sublingual</td>
<td>0.3 - 0.6 mg, can repeat up to 3 times at 5 minute intervals</td>
<td>2 minutes</td>
</tr>
<tr>
<td></td>
<td>GTN spray</td>
<td>0.4 - 0.8 mg per metered dose, no more than 3 sprays at 5 minute intervals</td>
<td>2 minutes</td>
</tr>
<tr>
<td></td>
<td>Transdermal patch</td>
<td>0.2 - 0.8 mg over 12 hours on, then 12 hours off</td>
<td>1 - 2 hours</td>
</tr>
<tr>
<td>Isosorbide dinitrate</td>
<td>IV</td>
<td>1.25 – 5 mg/hour</td>
<td>1 minute</td>
</tr>
<tr>
<td></td>
<td>Transdermal patch</td>
<td>2.5 – 10 mg</td>
<td>3 - 4 minutes</td>
</tr>
<tr>
<td></td>
<td>Oral</td>
<td>10 – 20 mg, bd/tds</td>
<td>30-60 minutes</td>
</tr>
<tr>
<td>Isosorbide mononitrate extended release</td>
<td>Oral</td>
<td>30-60 mg, od</td>
<td>60 minutes</td>
</tr>
</tbody>
</table>

*The dose of IV nitrates should be titrated every 5 - 10 minutes until symptoms and/or ischaemia is relieved and the desired haemodynamic response is obtained.

4.3.3.9 Calcium channel blockers

There is no data to support the routine use of calcium channel blockers post-STEMI.154,155

However they may be used as adjunctive therapy in patients with hypertension and/or on-going ischaemia despite β-blockers and nitrates.

In patients who cannot tolerate β-blockers, verapamil or diltiazem may be used for secondary prevention.156

Calcium channel blockers should be avoided in patients with LV dysfunction and pulmonary congestion. Verapamil and diltiazem should not be given in the presence of bradycardia and AV block.
4.3.3.10 Others – magnesium, lignocaine, glucose – insulin potassium infusions

Magnesium and lignocaine are not recommended for routine use in patients with STEMI. \(^{153, 157}\)

Although earlier studies and meta-analysis seem to indicate that glucose-insulin-potassium infusions are beneficial, more recent studies have not shown reduction in mortality or infarct size. \(^{158, 159}\)

Key Messages:

- All patients should receive 300 mg aspirin and 75-300 mg clopidogrel followed by a maintenance dose of 75-150 mg of aspirin long-term and 75 mg of clopidogrel daily.
- Patients who underwent PCI require DAPT for up to a year.
- All patients should be on β-blockers if there are no specific contraindications.
- Other medications that have been shown to improve survival if given early are ACE-IIs (or ARBs if ACE-I intolerant) and statins.

4.4 Complications of STEMI

These are:
- Arrhythmias.
- LV dysfunction and shock.
- Mechanical complications.
- RV infarction.
- Others e.g. pericarditis.

4.4.1 Arrhythmias

These include:

A) Tachyarrhythmias

- **Pulseless ventricular tachyarrhythmias**
  This is either pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF). Defibrillate immediately. Early VF occurs within the first 48 hours and is due to electrical instability. Late VF is associated with large infarcts and poor pump function and carries a poor prognosis (refer to algorithm 1, page 49).

- **Ventricular tachycardia (VT)**
  VT in the setting of STEMI may arise from either ischaemia (usually within 48 hours) or from myocardial scar due to the infarct (late onset). Treatment of ischaemia may result in the termination of the tachycardia (refer to algorithm 2, page 50).

- **Ventricular premature contractions (VPC)**
  These are often benign and do not require treatment. Correct underlying ischaemia, hypoxia and electrolyte disturbances.
• **Accelerated idioventricular rhythm**
  These do not require any treatment. This is a sign suggestive of successful reperfusion.

• **Atrial fibrillation (AF)**
  This is more commonly seen in the elderly and is associated with large infarcts. It denotes a poorer prognosis and carries an increased risk of thromboembolism (refer to algorithm 3, page 51).

B) **Bradyarrhythmias**

• **Sinus bradycardia**
  This does not require treatment unless associated with symptoms and/or hypotension.

• **Atrio-ventricular (AV) block**
  First degree and second degree type 1 (Mobitz 1) do not need treatment. Patients with second degree type 2 (Mobitz 2) and complete AV block may not require treatment if haemodynamically stable. If unstable, urgent temporary pacing is necessary. Atropine may be given in the interim (maximum 3 mg) (refer to algorithm 4, page 52).

  Patients with anterior infarcts who develop second degree (Mobitz 2) and complete AV block carry a worse prognosis. Even if haemodynamically stable, these patients require temporary pacing.

  In inferior MI, the inferior wall of the RV may be friable. Insertion of a temporary wire may lead to RV perforation. Thus extra care is required to prevent this complication.

• **Asystole and pulseless electrical activity (PEA)**
  Rhythms which require defibrillation (pulseless VT/VF) are called shockable rhythms while asystole and PEA are non-shockable rhythms.

  For the management of asystole and PEA, refer to algorithm 1, page 49.
4.4.2 LV dysfunction and cardiogenic shock

LV dysfunction is the single strongest predictor of mortality following STEMI. The mechanisms responsible for acute LV dysfunction include myocardial necrosis, myocardial stunning, atrial and ventricular arrhythmias and valvular dysfunction (pre-existing and/or new).

Co-morbidities such as infection, pulmonary disease, renal dysfunction, diabetes, anaemia and drugs may aggravate HF.

4.4.2.1 Presentation

The clinical manifestation of LV dysfunction varies from asymptomatic to cardiogenic shock. An important prognostic indicator is LVEF which can be assessed objectively using echocardiography.

A useful clinical classification of LV dysfunction is the Killip’s Classification. 42,160 (Table 12, page 29)

Table 12: Clinical classification of LV dysfunction in STEMI (Killip’s Classification)

<table>
<thead>
<tr>
<th>KILLIP CLASS160</th>
<th>CLINICAL FEATURES</th>
<th>APPROXIMATE PROPORTION OF PATIENTS WITH AMI (%)42</th>
<th>30 DAY-MORTALITY (%)42</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No signs of LV failure</td>
<td>71</td>
<td>7</td>
</tr>
<tr>
<td>II</td>
<td>S3 gallop, bibasal crackles</td>
<td>23</td>
<td>20</td>
</tr>
<tr>
<td>III</td>
<td>Acute pulmonary oedema</td>
<td>3.7</td>
<td>39</td>
</tr>
<tr>
<td>IV</td>
<td>Cardiogenic shock</td>
<td>2.4</td>
<td>70</td>
</tr>
</tbody>
</table>

4.4.2.2 Investigations

Echocardiography is an essential tool and needs to be performed early to assess LV function and volumes, valvular function, extent of myocardial damage and to detect mechanical complications.

Other investigations that may be helpful in the management include:

- Chest radiograph (to assess extent of pulmonary congestion).
- ECG (for the detection of arrhythmias, ischaemia or reinfarction).
- Arterial blood gases.

4.4.2.3 Management

A) Acute Heart Failure

Acute management includes the following:

- Oxygen – by nasal prongs/face mask to maintain SpO2 above 95%. Consider non-invasive ventilation [bi-level positive airway pressure (BiPAP) or continuous positive airway pressure (CPAP)] early if SpO2 cannot be maintained with high flow face mask.
• Diuretics – IV frusemide.
• IV nitrates.
• Inotropes if hypotensive.
  o Noradrenaline should be the first choice agent. It is started at the lowest dose and titrated till the SBP is more than 80 mmHg.\textsuperscript{161}
  o Dopamine should be refrained as it has been associated with a higher mortality when used in patients with cardiogenic shock.\textsuperscript{161}
• In patients who are hypoxic or exhausted and are unable to achieve satisfactory SpO2 despite non-invasive ventilation, endotracheal intubation and ventilatory support may be required.

B) Cardiogenic shock

This condition occurs in 6–10% of all cases of STEMI and remains a leading cause of death, with hospital mortality rates approaching 70%.\textsuperscript{162}

The typical presentation is of a low cardiac output state (hypotension with SBP < 90 mmHg, resting tachycardia, altered mental status, oliguria, cool peripheries) and pulmonary congestion. The haemodynamic criteria are an elevated central filling pressure [pulmonary central wedge pressure (PCWP)] of $> 18$ mmHg and cardiac index is $< 2.2$ L/min/m$^2$. Insertion of a pulmonary artery catheter may be helpful in the diagnosis and management of these patients.

Emergency PCI may be life-saving and should be considered early irrespective of the time delay from onset of MI. The use of IABP has not shown a definite benefit and its use should be individualised.\textsuperscript{163}

LV assist device may be considered for patients who do not respond to conventional therapies.

When cardiogenic shock is due to a mechanical defect, urgent surgical repair is indicated. Concomitant CABG surgery in these patients remains an issue of debate. The decision must be individualised.

4.4.3 Mechanical complications

These include the following:
• Free wall rupture - it is usually fatal and presents with sudden cardiovascular collapse and haemopericardium.
• Ventricular septal rupture.
• Mitral regurgitation.

The diagnosis should be suspected in patients with sudden clinical deterioration and suggested by the presence of new murmurs or diminished heart sounds. The diagnosis can be confirmed by echocardiography in these patients early surgery should be considered.
Ventricular septal rupture requires urgent surgical repair, but there is no agreement on the optimal timing for surgery.

4.4.4 Right Ventricular Infarct (RVI)

Patients with RVI may have varying clinical presentation, from asymptomatic to cardiogenic shock. Haemodynamically significant RVI complicates approximately 5-10% of all STEMI. It occurs in 30 – 50% of patients with infero basal (formerly known as infero-posterior) MI and is associated with a significantly higher mortality. RVI can also occur in patients with extensive anterior STEMI.

4.4.4.1 Clinical diagnosis

The presence of RVI should be sought in all patients with inferior STEMI. The clinical triad of hypotension, clear lung fields and elevated jugular venous pressure in the setting of inferior STEMI is suggestive of RVI.

ST elevation in the right praecordial leads (V4R) is the most specific finding in diagnosing RVI. However, this ECG finding may be transient, often resolving within 8-10 hours.

4.4.4.2 Management

Treatment strategies depend on the severity of peripheral hypoperfusion and the degree of co-existing LV dysfunction. Drugs that reduce the preload, such as nitrates and diuretics should be avoided.

Management includes:

- Optimisation of IV fluids (saline or colloids) is the key therapy to correct the hypotension.
- Inotropes.

Failure to respond to these measures usually indicates concomitant LV dysfunction. These patients require more aggressive management with afterload reducing agents such as nitroprusside and IABP.

4.4.5 Others

4.4.5.1 Chest pain post-STEMI

Chest pain post-STEMI may be due to reinfarction, ischaemia or pericarditis. Non cardiac causes must also be considered.

A) Reinfarction

Reinfarction occurs in about 3-4% of patients who had undergone fibrinolytic therapy and received aspirin. Reinfarction may be diagnosed by:

- Recurrence of ischaemic type chest pain.
• Recurrence of ST segment elevation of at least 0.1 mv in at least two contiguous leads and/or
• Re-elevation of serum cardiac biomarkers - CK-MB (≥ 20% increase in the value from the last sample).4

Death, severe HF and arrhythmias are more common in these patients. They should be considered for rescue PCI.

B) Post-infarct angina
Early recurrent angina, especially after successful reperfusion with fibrinolytic therapy may occur in up to 18% of patients.166, 167 The ECG in these patients may show ST segment changes or pseudo-normalisation of inverted T-waves. These patients should be sent for early coronary angiography with a view to revascularisation.

C) Pericarditis
Pericarditis secondary to STEMI may produce pain as early as the first day and as late as 6 weeks.168 The pain classically becomes worse on deep inspiration and may be relieved when the patient sits up and leans forward. A pericardial rub may be detected.

Dressler’s syndrome (post-MI syndrome) usually occurs 2-10 weeks after STEMI. This is immunologically mediated.169 It is treated with aspirin 600 mg 3-4 times a day.

Acetaminophen or narcotic analgesics may be reasonable if aspirin, even in higher doses, is not effective.

Glucocorticoids and non-steroidal anti-inflammatory drugs (NSAIDs) are potentially harmful for treatment of pericarditis after STEMI and are best avoided in the first 4 weeks of STEMI.170, 171

4.4.5.2 LV thrombus and arterial embolism
The prevalence of LV mural thrombus has been reduced from 20% to 4% in the era of primary PCI.172,173 The majority of these occur following anterior or large infarcts. Anticoagulation therapy is recommended for 3-6 months or till the LV thrombus disappears or organises on echocardiography.174

4.4.5.3 Deep venous thrombosis (DVT)
In high-risk patients (prolonged bed rest, HF, unable to mobilise), prophylactic anti-coagulation therapy (SC heparin 5000 units bd, LMWH – e.g. enoxaparin 40 mg od) may be considered until the patient is ambulant.
4.5 Urgent/Emergent CABG surgery:
Urgent/emergent CABG surgery should be considered in the following situations:
• At the time of surgical repair of post-infarction VSD or mitral valve regurgitation (See section 4.4.3).
• Patients with failed reperfusion whose coronary anatomy and clinical profile are suitable.

In general, CABG surgery in this group of patients carries a very high in hospital mortality rate.

Key Messages:
• Important complications following STEMI are arrhythmias and HF.
• HF may be due to extensive myocardial damage or mechanical complications.

5 RISK STRATIFICATION POST-STEMI

Risk stratification of patients post-STEMI serves to prognosticate and identify appropriate treatment strategies. Risk stratification starts from admission and is a continuing process. It is especially important in patients treated medically and those with multivessel disease who underwent PCI of the IRA only. The GRACE score predicts in-hospital and 6-month mortality (Appendix IV, page 56 & 57).

Ideally, all patients with poor prognostic indicators who did not undergo primary PCI should have a coronary angiogram during the index hospitalisation.

These include:
• Older persons (> 75 years).
• Female gender.
• Previous MI.
• Anterior MI.
• Inferior MI with RV involvement.
• Diabetes mellitus (DM).
• Persistent or recurrent ischaemia as manifested by post-infarction angina or ST segment depression at rest.
• Hypotension.
• HF.
• AF and late (after 48 hours) ventricular arrhythmias.
• Presumably new LBBB.
• Previous CABG or PCI.
All other patients who did not undergo coronary angiography should be risk stratified early. This may be done by assessing:

- **LV function**
  - Clinical, chest X-ray, echocardiogram, radionuclide studies or cardiac MRI.

- **Presence of myocardial ischaemia**
  - Clinical (recurrent angina).
  - Exercise stress testing in asymptomatic patients.

- This may be done from day 5 post-STEMI (sub-maximal stress test with a target heart rate of 70% of maximum predicted heart rate) up to 6 weeks post-STEMI (maximal with a target heart rate of 90% of maximum predicted heart rate for age or symptom limited).

- If the pre-discharge sub-maximal stress test is negative, the patient should be subjected to a maximal or symptom limited stress test within 6 weeks after discharge.

- For those who cannot exercise, consider dobutamine stress echocardiogram, radionuclide perfusion studies or cardiac MRI.

- **Presence of malignant ventricular arrhythmias.**

**I.A** In the presence of angina, inducible ischaemia or late ventricular arrhythmia early coronary angiography with a view to revascularisation is indicated.80

**I.C** In patients with poor LV function, myocardial viability studies (dobutamine stress echocardiogram, radionuclide perfusion studies or cardiac MRI) would help to differentiate scarred from viable ischaemic myocardium.

**II-a,B** Patients with severe CAD, angina, viable myocardium and reversible ischaemia with mild LV dysfunction (LVEF > 35%) are more likely to benefit from revascularisation.175

Patients with palpitations, near faints and syncope require comprehensive evaluation to determine the cause of their symptoms and risk of sudden cardiac death. This includes:

- Serum electrolytes.
- Resting ECG.
- 24-hour ambulatory ECG recording.
- Evaluation of LV function.
- Assessment for reversible myocardial ischaemia.
- Coronary angiography.
In these patients, reversible causes such as electrolyte disturbances and ischaemia should be corrected.

The following medications have been shown to reduce the incidence of sudden death:

- **β-blockers**.\(^{176, 177}\)
- **ACE-Ils**.\(^{178}\)
- **Mineralocorticoid antagonists**.\(^{143}\)
- **Statins**.\(^{179, 180}\)

In addition, the following patients should be considered for an implantable cardioverter-defibrillator (ICD):

- **Secondary prevention** in patients with resuscitated sudden cardiac death.\(^{181-183}\)
- **Primary prevention** in patients with LV dysfunction [ejection fraction (EF) < 30%]. ICD may be considered 40 days post-STEMI and 3 months post-revascularisation.\(^{184-186}\)

### Key Messages:

- All patients post-STEMI should be risk stratified either clinically or by using GRACE risk score.
- High-risk patients should be referred to cardiology centres.

### 6 DURATION OF HOSPITALISATION

The duration of hospital stay following STEMI will depend on the extent of myocardial damage, presence of complications and comorbidities.

Early (same day) transfer to the referring centres may be considered in selected low-risk uncomplicated patients after successful primary PCI.

Asymptomatic patients with uncomplicated STEMI may be discharged after 3-5 days particularly if the IRA has been successfully reperfused.\(^{187, 188}\) Patients with significant LV dysfunction or other complications may require a longer hospital stay.
7 SECONDARY PREVENTION

Patients who survive the initial course of STEMI are at increased risk of morbidity and mortality because of reinfarction and the development of other complications such as HF and late onset arrhythmias. Thus, it is important that efforts be made to reduce this risk.

Secondary prevention should be initiated in-hospital. Patients should be advised on the benefits and importance of lifestyle changes. A close collaboration between the cardiologist, the general/family physician, the rehabilitation specialist, pharmacists and dietician is important.

These lifestyles measures include:

7.1 Cessation of smoking

In patients with known CHD, smoking cessation reduced CHD mortality by 36% as compared to those who continued smoking. Following MI, smoking cessation reduced subsequent mortality by nearly 50%.

This lifestyle change confers a risk reduction which is at least as great as other pharmacological interventions. Stopping smoking is potentially the most effective of all secondary prevention measures.

Trials of nicotine replacement therapy using either transdermal nicotine patch or nicotine chewing gum have proven to greatly increase abstention rates after cessation. Nicotine patches have been demonstrated to be safe in ACS patients. Such pharmacological therapy, as well as physician-guided counselling and nurse-directed programmes are cost-effective and should be encouraged.

The safety of electronic cigarettes is uncertain at this time.

7.2 Diet and weight control

Dietary intervention has been shown to reduce cardiac event rates post-STEMI.

Recommendations include:

- Total calorie intake should be tailored to the desirable body weight.
- Substituting saturated and trans fat with monounsaturated and polyunsaturated fat from vegetables and marine sources.
- Reducing total fat to 30% of total calorie intake.
- More high fibre food and whole grains which have low glycaemic index instead of more rapidly digested carbohydrates.
- More fruits, nuts and vegetables.
- Increasing intake of omega 3-fatty acids by eating fish at least twice a week.
- Reducing salt intake – no added table salt.
- Increasing intake of oat β-glucan to at least 3 g a day will lower total and LDL-C by 5-10%.
7.3 Regular exercise

Exercise training as part of coronary rehabilitation programmes was associated with a reduction in cardiac mortality in patients with CHD. Exercise-based rehabilitation has been shown to be effective at reducing all-cause mortality and the risk of reinfarction, as well as improving risk factors, exercise capacity and health-related quality of life after STEMI. Thirty minutes of moderate intensity aerobic exercise at least five times per week is recommended. It should be supplemented with an increase in daily lifestyle activities such as walking up stairs whenever possible.

7.4 Control of hypertension

After STEMI, prognosis is affected by both the pre-existing and the subsequent BP. The higher the pre-existing BP, the higher the fatality rate. After ACS, the BP goal should be systolic < 140 mmHg but not < 110 mmHg. Drugs of choice include ß-blockers, ACE-Is and ARB (if ACE-I intolerant). In addition, lifestyle modification (reduced salt intake, increased physical activity and weight loss) usually helps achieve these goals.

7.5 Good glycaemic control

Good control of the blood glucose is important and should be individualised.

7.6 Antiplatelet agents

- Aspirin – low dose aspirin 75-100 mg daily is indicated indefinitely after STEMI unless contraindicated.
- In patients who cannot tolerate aspirin, alternatives include:
  - Clopidogrel, 75 mg daily.
  - Ticopidine, 250 mg b.d.

DAPT should be given for at least one month post-fibrinolytic therapy for secondary prevention.

Following primary PCI, DAPT with combination of aspirin and clopidogrel or ticagrelor or prasugrel is recommended. DAPT with aspirin and oral adenosine diphosphate (ADP) receptor antagonist is recommended to be continued for up to 12 months after STEMI, with a strict minimum:

- 1 month for patients receiving BMS.
- 6 months for patients receiving DES.
7.7 **β-blockers**

**IA** The benefit of long-term treatment with β-blockers after STEMI is well established from clinical trials conducted in the pre-thrombolytic era.216

**IA** Oral treatment with β-blockers is indicated in patients with HF or LV dysfunction.217-220 When patients are haemodynamically stable, β-blockers may be initiated.

7.8 **ACE-Is and ARBs**

**IA** ACE-Is are indicated starting within the first 24 hours of STEMI in high-risk patients (LVEF < 40% or who have experienced HF in the early phase, DM or an anterior infarct) and should be continued indefinitely if there are no contraindications.137, 221

**II-a,A** In all other patients, ACE-Is should be considered.222-224

**IB** In ACE-I intolerant patients, an ARB may be used preferably valsartan.142, 225

7.9 **Mineralocorticoid antagonists**

**IB** Mineralocorticoid antagonists, e.g. eplerenone should be considered in patients with LVEF < 40% and HF in the absence of renal failure or renal impairment.143

7.10 **Lipid-lowering therapy**

**IC** A lipid profile should be taken as soon as possible after presentation.

**IA** Statins at high dose should be initiated early after admission in patients with STEMI who do not have contraindication or history of intolerance, regardless of initial cholesterol values and continued indefinitely.144, 145, 226, 227

**IB** Target LDL-C should be < 2.0 mmol/L.228-231

**IB** Recent studies indicate that lowering LDL-C even further (< 1.8 mmol/L) confers greater benefits.145, 232-234

7.11 **Oral anticoagulants (warfarin)**

**IC** In patients with a clear indication for oral anticoagulation e.g. LV thrombus, AF with CHA₂DS₂-VASC score ≥ 2 or mechanical prosthetic valve, oral anticoagulation must be implemented in addition to antiplatelet therapy (Appendix V, page 58).

**IC** The optimal duration of triple therapy is unknown and clinical judgement should consider the risk of bleeding versus the risk of stent thrombosis.

**IC** There is insufficient data on the use of new oral anticoagulant agents at this time.

**II-a,B** In patients with LV thrombus, warfarin should be given for 3-6 months or till the thrombus disappears or organises.172, 235
7.12 Others

- Hormone replacement therapy (HRT) is not beneficial for secondary prevention.\textsuperscript{236, 237}
- Vitamin E and antioxidants have no clinical benefit.\textsuperscript{238-240}
- Garlic, lecithin, Vitamin A and C are not beneficial.

**Key Messages:**
- Secondary prevention interventions such as smoking cessation, statin therapy, control of diabetes, hypertension and lifestyle changes can reduce mortality and cardiac event rate post-STEMI.
- Healthcare providers must provide patient education and encourage compliance.

8 SPECIAL GROUPS

8.1 STEMI in the elderly

Patients above the age of 75 years have a much higher in-hospital as well as 1-year mortality.\textsuperscript{241-243} This may be explained by their atypical and delayed presentations, co-morbidities and under-utilisation of reperfusion strategies.\textsuperscript{244, 245} Diagnosis may be delayed because of:

- Atypical symptoms such as dyspnoea, syncope and acute confusion.
- Non-diagnostic and difficult to interpret ECGs.
- Non-diagnostic cardiac biomarkers. The baseline troponin levels may be elevated in as many as 22\% of the elderly without an MI because of pre-existing cardiac and renal disease.\textsuperscript{246, 247} Thus, a high index of suspicion must be present to make a diagnosis of ACS in the elderly. The trend of a rise and fall cardiac biomarker levels is critical to the diagnosis.

**Management**

Elderly patients were excluded from most of the large trials and data is mainly derived from post-hoc analysis.\textsuperscript{242} One should consider the biological age rather than the chronological age of the patient when making management decisions. The elderly are a heterogenous group and the risk-benefit ratio of each intervention should be individualised.\textsuperscript{248}

CKD is common in these patients and may increase the risk of bleeding. Most drugs are excreted by the kidneys and doses need to be adjusted. Creatinine clearance (CrCL) should be estimated using the Cockcroft-Gault (Appendix VI, page 59), Modification of Diet in Renal Disease (MDRD) equations (Appendix VII, page 59) or CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) equations (Appendix VIII, page 59).
In the management of STEMI in the elderly:

- **Primary PCI** - this is the preferred reperfusion strategy if facilities are available and the patient is eligible for PCI.\(^{249-252}\) Procedural success is highly variable. Elderly patients are more likely to have PCI related complications especially bleeding.\(^{253}\)

- **Fibrinolytic therapy** - there is an increased risk of intracranial haemorrhage in the elderly and the risks of bleeding have to be carefully considered in those older than 75 years.\(^{254-256}\)

- **Adjunctive therapy** - when compared with younger patients, the elderly have a greater absolute reduction with most evidence based medications.\(^{257}\)
  - **Aspirin** - at a dose of 75 to 150 mg in the absence of contraindications.\(^{242, 258}\)
  - **Clopidogrel** - the absolute benefits of clopidogrel are similar, but relative benefits are less in the elderly. Patients undergoing PCI with higher TIMI risk scores or prior revascularisation are more likely to benefit.\(^{124, 259, 260}\) A loading dose when compared to a conventional dose of clopidogrel did not result in an increased bleeding risk in the elderly.\(^{261}\) The need for a loading dose needs to be individualised.
  - **Ticagrelor** – ticagrelor has a similar efficacy as clopidogrel in patients aged ≥ 75 years of age and those < 75 years of age. There was no increased risk of bleeding.\(^{262}\)
  - **Prasugrel** - should be avoided in patients > 75 years of age.\(^ {98}\)
  - **Enoxaparin** was found to be more effective than UFH in the majority of subgroups except in patients > 75 years of age in whom there is no available data.\(^{242, 265}\) The dose however needs to be adjusted.\(^{129, 264}\) (Table 8, page 24)

- **Secondary prevention** - the benefit associated with the use of β-blockers, ACE-Is and statins is similar to, and often greater than, that observed in younger patients.\(^{135, 265-271}\)

- **The elderly benefit from cardiac rehabilitation and exercise training.**\(^{272, 273}\)

- **Risk stratification** - this has to be individualised and patient preferences are important in determining further management. The presence of ongoing ischaemia, symptomatic malignant arrhythmias and a depressed LV function are poor prognostic indicators and would generally necessitate a more aggressive approach. Both PCI and CABG, when indicated, can be carried out in the elderly with acceptable morbidity and mortality by experienced operators. The risks are however higher than in younger patients.
8.2 STEMI in diabetics

Blood glucose level was associated with adverse outcomes independent of prior diabetic status in patients following STEMI.274, 275 This poor prognosis in diabetics occurred despite successful reperfusion of the infarct-related vessel.275 Local NCVD registry data indicate that diabetics with STEMI did worse than would be expected from their TIMI risk score.29

Management

Diabetic patients should be treated in a similar manner as non-diabetics. Primary PCI is the reperfusion strategy of choice in these high-risk patients.276

Adjunctive therapy includes:

- Antiplatelet agents – aspirin and clopidogrel or prasugrel or ticagrelor.18-20, 26, 27, 98
- Prasugrel has been found to be more effective in diabetics.98
- Gp IIb/IIIa inhibitors – Meta-analysis and registry data indicate that early administration of abciximab in diabetic patients resulted in infarct-related artery patency before and after primary PCI, and a trend towards an improvement in 1-year survival.277, 278

Diabetes is a well-recognised risk factor for contrast induced nephropathy (CIN).279 In the setting of STEMI, an elevated pre-procedural glucose level is associated with a greater risk for CIN even in patients without known diabetes.280 For prevention of CIN, see Appendix IX and X, pages 60 & 61)

There is still a lack of consensus on the optimal management of blood sugars during the acute event.281, 282 Intensive insulin therapy to achieve normoglycaemia in the acute setting has not been shown to reduce mortality and is associated with an increase in the episodes of hypoglycaemia. A low blood sugar level (< 4.5 mmol/L) has also been associated with adverse outcomes. A general consensus is to keep blood sugars between 6-10 mmol/L in the acute setting and then aim for optimal control following discharge.282, 283

8.3 STEMI in women

Women have a higher mortality following STEMI.284-289 They are generally about 10 years older when they develop CHD and have more comorbidities.285

Women tend to have atypical symptoms and generally do not present with classical ischaemic-type chest pain. They are more likely to have fatigue, neck pain, syncope, nausea, right arm pain, dizziness and jaw pain.286 Subjective reports of sleep disturbance preceding MI seem to be common in women.287 Women are more likely to present with cardiogenic shock.288-290
Management

In general, women should be treated the same as men taking into consideration the following:258, 291, 292

- Primary PCI is the preferred reperfusion strategy. Women however, have higher early all-cause and cardiac mortality after primary PCI and they also have higher bleeding risk.288 Women are less likely to undergo coronary angiography and reperfusion and those who underwent primary PCI tend to have longer DBT.288, 293

- Women given fibrinolytics [recombinant tissue plasminogen activators (r-TPA)] had a higher incidence but lower mortality from bleeding than men.294

- Adjunctive therapy is similar in both genders.264, 295, 296 Women however have higher bleeding risk.

- Cardiac rehabilitation – women, especially older women, are under-referred for cardiac rehabilitation. Efforts should be initiated to overcome these barriers.297

- Continuing HRT after STEMI does not confer a benefit nor pose a worrisome increase in risk. When considering the need for HRT for menopausal symptoms, clinical judgement is necessary.298, 299

Key messages:

- Diagnosis of STEMI in the elderly, diabetics and women is difficult and a high index of suspicion is important.

- Treatment is the same although the elderly and women tend to have higher bleeding risk.

8.4 STEMI in renal disease

Patients with all grades of CKD (Appendix VI, VII & VIII, page 59) have a worse prognosis after an MI compared to those with normal renal function; those on dialysis may have as high as 74% mortality at 2 years.29, 300, 301 The S\text{cr}\ at admission predicts long-term mortality even after successful primary PCI. 1 in 6 survivors develop worsening renal function during the admission and this is also associated with increased mortality even in patients with normal renal function at baseline.302

Patients with CKD tend to be older, have more comorbidities such as diabetes and be on more cardio-protective medications. They are more likely to present without chest pain, be in Killip Class III or IV and present as NSTEMI.303 Troponins may be elevated in patients with CKD even in the absence of ACS. A rise and fall in cardiac biomarkers is essential to make a diagnosis of MI. Small studies seem to indicate that Troponin I may be more specific for myocardial necrosis than Troponin T in CKD.304

The optimal mode of reperfusion in patients with CKD presenting with STEMI has not been addressed in large prospective trials. Registry data
indicate that 30-day and 1-year mortality were higher in those treated with primary PCI as compared to those given fibrinolytics and those who were not reperfused. This suggests that fibrinolytics may be the preferred strategy in these patients especially in those with glomerular filtration rate (GFR) < 30 ml/minute. \(^{305, 306}\)

The success rate of emergency PCI in patients with CKD is generally lower and an unsuccessful primary PCI can result in worsening renal function due to haemodynamic instability and CIN. \(^{307}\) Patients with CKD tend to have more extensive atherosclerosis and more complex calcified lesions. \(^{307}\) In some patients with multivessel coronary involvement and haemodynamic instability, complete revascularisation by CABG may be superior to PCI.

CIN is higher among patients undergoing primary PCI as compared to elective procedures. It occurs in as high as 20-30% of cases and is associated with adverse 1-year mortality. \(^{308}\) Half of these patients may go on to develop persistent renal failure. \(^{309}\) For the prevention of CIN, see Appendix IX & X, pages 60-61. Recent studies have shown high-dose statin therapy to reduce the incidence of CIN. \(^{310-312}\)

Management

Patients with CKD were excluded from most clinical trials and most of the available data is derived from post-hoc analyses. These patients have higher rates of bleeding and the doses of antithrombotic agents need to be adjusted accordingly. (Table 13, page 44)

- Fibrinolytic therapy may be the preferred strategy particularly in patients with GFR < 30 ml/minute. \(^{305, 306}\)

### Table 13: Dosages of Antithrombotics in CKD

<table>
<thead>
<tr>
<th>Antithrombotics</th>
<th>LOADING DOSE</th>
<th>MAINTENANCE DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>UFH</td>
<td>No change</td>
<td>No change</td>
</tr>
<tr>
<td>Enoxaparin</td>
<td>30 mg IV</td>
<td>1 mg/kg SC every 24 hours if CrCL &lt; 30 ml/minute</td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>Avoid if CrCL &lt; 30 ml/minute</td>
<td>Avoid if CrCL &lt; 30 ml/minute</td>
</tr>
<tr>
<td>Eptifibatide</td>
<td>180 mcg/kg</td>
<td>IV infusion 1.0 mcg/kg/minute if CrCL &lt; 50 ml/minute</td>
</tr>
<tr>
<td>Tirofiban</td>
<td>IV infusion 0.4 mcg/kg/minute for 30 minutes</td>
<td>IV infusion 0.05 mcg/kg/minute if CrCL &lt; 30 ml/minute</td>
</tr>
</tbody>
</table>


Adjunctive therapy:
• Standard care: aspirin 75-100 mg and clopidogrel 75 mg.
  
  o This regime was found to be non-inferior to Gp IIb/IIIa inhibitors or loading dose of clopidogrel. Standard care was associated with less bleeding.313
  
  o In patients with mild to moderate CKD who underwent primary PCI, a 600 mg loading dose of clopidogrel was not found to be beneficial. This dose however did not increase the in-hospital major bleeding rate.314-316
  
  o Ticagrelor reduced mortality and major cardiovascular events more than clopidogrel.26, 317, 318

• Antithrombotics:
  
  o Fondaparinux - in patients with mild to moderate CKD, fondaparinux was more effective than enoxaparin largely explained by the lower rates of bleeding.319 However, it should be avoided in patients with CrCL <30 ml/minute.
  
  o Bivalirudin was not superior to heparin and Gp IIb/IIIa inhibitors but was associated with less bleeding.320-322

• Adjunctive therapy: meta-analyses and post-hoc analyses of studies in patients with mild to moderate CKD have noted benefits with aspirin, β-blocker and statin therapy.323, 324

In patients on dialysis, there is a lack of evidence concerning the cardiovascular benefits of ACE-Is and ARBs, β-blockers and statins.325, 326 Aspirin may result in greater bleeding risks in these patients.

Key messages:
  
  • Treatment of STEMI in patients with CKD should be individualised.
  
  • Fibrinolytic therapy may be the preferred reperfusion strategy.
  
  • In view of bleeding risk, lower doses of aspirin (75 – 100 mg) and clopidogrel (75 mg) should be given.

9 CARDIAC REHABILITATION IN STEMI

All post-STEMI patients (including those post-PCI or CABG) should undergo comprehensive cardiac rehabilitation. The mortality benefits of cardiac rehabilitation persist even in the era of more aggressive treatment strategies of STEMI.200, 327

This programme aims at improving the long-term prognosis and optimising the physical, psychological and social well-being of the patient. It comprises prescribed exercise training and education, counselling, risk factor modification and behavioural interventions. Cardiac rehabilitation should start in the CCU, continue in the outpatient setting and extend to community care. It is a proven
effective intervention and every effort must be made to ensure minimal dropouts so as to maximise beneficial effects of the programme.

The components are as follows:

- Patient assessment for appropriateness of programme.
- Ongoing medical surveillance (BP/ lipids/ diabetes control).
- Nutritional counselling.
- Smoking cessation.
- Psychosocial counselling.
- Physical activity counselling - resumption of sexual activity (after 1 week*), driving, work, exercise and air travel (after 2 weeks*). A simple rule of thumb is to resume sexual activity if the patient can climb 2 flights of stairs comfortably.
- Exercise training.
- Pharmacological treatment (optimum up-titration or maximum tolerable dose).

*stable uncomplicated cases

Patient education regarding lifestyle modification is vital and enhances compliance and commitment to therapy. Family members should be encouraged to learn basic CPR.

Structure of a cardiac rehabilitation programme (CRP) consists of 4 phases:
- Phase 1: Inpatient CRP: focus on patient education.
- Phase 2: Outpatient CRP.
- Phase 3: Extended CRP (for patients with impaired functional status initially, requiring an extended course before returning to usual activity in the community).
- Phase 4: Lifetime CRP.

10 FUTURE DEVELOPMENTS

Since the publication of the 2nd CPG, newer technologies have emerged in the diagnosis and management of STEMI. While some of these technologies are currently being subjected to clinical trials, others that have proven effective, are being gradually integrated in the care of patients with STEMI.

New technological advances include:

- Diagnostics – new biomarkers e.g. high sensitivity troponins and point-of-care measurements.
- Risk stratification – genetic testing to identify individuals at high-risk of developing ACS and reinfarction following STEMI.
- Bioinformatics - advanced ECG diagnostics allowing diagnosis of STEMI at FMC and relaying this information to healthcare personnel thus resulting in earlier reperfusion strategies.
- Imaging techniques – high resolution hand-held echocardiography to assist in the diagnosis of STEMI in the presence of non-diagnostic ECGs.
- Therapeutics – newer antithrombotics, novel lipid lowering agents
and myocardial preservation in the acute phase of STEMI utilising stem cells, myocytes, growth factors and other agents. \(^{334,335}\)

- Interventional - bioresorbable scaffolds in STEMI, mechanical thrombectomy, distal embolic protection devices, and other adjunctive technologies. \(^{336-338}\)
- Regenerative medicine - stem cell therapies.

### 11 CHECKLISTS FOR FOLLOW-UP VISIT

The following should be assessed at each follow-up visit:

- Assess the presence or absence of cardiac symptoms and determine the functional class of the patient.
- Evaluate patients’ psychosocial (anxiety & depression) status and the social integration and support network.
- Review pre-discharge risk assessment and evaluate:
  - Presence of residual ischaemia.
  - LV function.
  - Current medications and optimise their doses.
- Treat to target.
  - BP: < 140/90 mmHg.
  - Lipids: LDL-C < 2.0 mmol/L, preferably < 1.8 mmol/L.
  - Diabetic control: targets should be individualised.
  - Achieve and maintain ideal body weight and waist circumference.

### 12 CLINICAL AUDIT INDICATORS

Performance measures should be used with the goal of improving quality of care for STEMI.

Process performance measures focus on aspects of care that are delivered to a patient, while outcome measures focus on end-points such as mortality or hospitalisation.

Suggested process performance indicators include:

- % of patients with suspected ACS who had an ECG done within 10 minutes of arrival in the emergency department
- % of patients with STEMI who received fibrinolytic therapy within 30 minutes (DNT) or
- % of patients with STEMI who received primary PCI within 90 minutes (DBT)
- % of patients discharged on DAPT
- % of patients discharged on β-blockers
- % of patients discharged on statins
- % of patients discharged on ACE-I/ARB
- % of patients who received an echocardiogram prior to discharge
- % of patients referred for cardiac rehabilitation
- % of smokers receiving quit smoking advice
In non-PCI capable hospitals,
• % of patients who received ischaemic testing (e.g. sub-maximal stress test, stress echocardiogram) prior to discharge

In PCI capable hospitals,
• % of patients receiving primary PCI
• % of patients undergoing coronary angiography during index admission

Suggested outcome measures indicators include:
• In-hospital mortality

Refer to Appendix XI for calculation of the above.

13 IMPLEMENTING THE GUIDELINES AND RESOURCE IMPLICATIONS

STEMI is one of the common causes of death in Malaysia and patients can present at all levels of healthcare. To ensure that patients receive good evidence based care, the information in this CPG must be successfully disseminated.

In improving outcomes, time is essence and early diagnosis assumes paramount importance. Patients should be educated to seek early treatment if they have symptoms of suspected ACS. Healthcare personnel need to have a high index of suspicion of ACS, make the diagnosis promptly and institute timely treatment.

This can be done by ensuring:
• regular public education via forums, mass media etc.
• continuous medical education of healthcare providers via regular seminars, lectures and roadshows.
• widespread availability of this CPG to healthcare providers via printed copies, electronic websites etc.
• regular audit of the performance and outcome indicators mentioned in section 12.
• availability of the drugs mentioned in this CPG. Most of these are already available in the Ministry of Health drug formulary. Healthcare personnel need however, to be educated on their appropriate and timely usage.
• a work-in-progress towards providing 24 hour coverage for primary PCI or pharmacoinvasive therapy.
• seamless care i.e. coordinated linkage between primary healthcare personnel and the tertiary cardiac centres.
ALGORITHM 1: PULSELESS ARRHYTHMIAS

ADULT CARDIAC ARREST
SHOUT FOR HELP/ACTIVATE EMERGENCY RESPONSE

1. Start CPR
   - Give oxygen
   - Attach monitor/defibrillator

2. Yes
   - Rhythm shockable?

3. VF/VT
   - Shock

4. CPR 2 min
   - IV/I/O access

5. Yes
   - Rhythm shockable?

6. CPR 2 min
   - Epinephrine every 3-5 min
   - Consider advanced airway, capnography

7. Yes
   - Shock

8. CPR 2 min
   - Amiodarone
   - Treat reversible causes

9. No
   - Asystole/PEA

10. CPR 2 min
    - IV/I/O access
    - Epinephrine every 3-5 min
    - Consider advanced airway, capnography

11. Yes
    - Rhythm shockable?

12. No
    - CPR 2 min
      - Treat reversible causes

13. If no signs of return of spontaneous circulation (ROSC), go to 10 or 11
    - If ROSC, go to post-cardiac arrest care

Adapted from 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science. Circulation.2010; 122:S729-S767
ALGORITHM 2: STABLE VENTRICULAR TACHYCARDIA
(if unstable for immediate synchronised cardioversion)

- Assess and support ABCs*
- Give oxygen
- Monitor ECG

Haemodynamically stable

Monomorphic VT
- IV amiodarone 150 mg over 10 minutes
- Repeat as needed up to 2.2 g/24 hours

Successful pharmacological CV**

Oral amiodarone

Failed pharmacological CV**

Electrical CV**

Polymorphic VT
- Check electrolytes and correct accordingly
- Stop all anti arrhythmic drugs (if any)
- IV magnesium
- Overdrive pacing

*ABC: airway, breathing, circulation
** CV: cardioversion
ALGORITHM 3: ATRIAL FIBRILLATION

Search and treat identifiable underlying causes

Haemodynamic Stability

Unstable

Electrical CV*

Successful CV*

Unsuccessful CV*

Oral amiodarone

IV amiodarone

Stable

Normal LV function

Rate or rhythm control
• Rate control
  o β-blockers
  o Calcium blockers
• Rhythm control
  o IV amiodarone followed by oral amiodarone
• Anticoagulation
  o if persistent after 48 hours or if CV* is contemplated

Impaired LV function

Rate or rhythm control
• Rhythm control preferably IV amiodarone followed by oral amiodarone
• Anticoagulation
• Electrical CV*

Pre medicate for CV*
• Anticoagulation

*CV: cardioversion
**ALGORITHM 4: BRADYCARDIA**

- **Yes**
  - Type II second degree AV block or third degree AV block?
  - Haemodynamically stable
  - Anterior MI

- **No**
  - Assess ABC*
  - Vital signs monitoring
  - Search for underlying reversible causes (e.g. electrolytes, drugs) and treat accordingly **myocardial ischaemia**

- **Serious symptoms due to bradycardia?**
  - **Yes**
    - Intervention sequence:
      - Atropine 0.5 to 1 mg, max 3 mg
      - Dopamine 2 to 10 mcg/kg/minute
      - Epinephrine 2 to 10 mcg/minute
      - Transcutaneous pacing if available
  - **No**

- **Observe**
  - Transvenous pacing

*ABC: airway, breathing and circulation
APPENDICES

APPENDIX I: DIFFICULTIES IN ECG DIAGNOSIS OF MI

The following conditions may cause ECG changes that may be confused with that of STEMI:

- Prior MI with Q-waves and/or persistent ST elevation.
- Early repolarisation.
- LBBB.
- Right ventricular pacing.
- Pre-excitation.
- Peri-/myocarditis.
- Pulmonary embolism.
- Subarachnoid haemorrhage.
- Metabolic disturbances such as hyperkalaemia.
- Cardiomyopathy.
- Cholecystitis.
- Tricyclic antidepressants or phenothiazines.
- J point elevation syndromes, e.g. Brugada syndrome.

In these difficult situations where the ECG is non-diagnostic, cardiac imaging techniques such as echocardiogram looking for presumed new wall motion abnormalities or elevation of cardiac biomarkers will help in making the diagnosis.

Adapted from Thygesen K et al. Third universal definition of myocardial infarction. Journal of the American College of Cardiology. 2012;60(16):1581-98.
**APPENDIX II: ELEVATIONS OF CARDIAC TROPONIN IN THE ABSENCE OF OVERT ISCHAEMIC HEART DISEASE.**

<table>
<thead>
<tr>
<th>Damage related to secondary myocardial ischaemia (MI type 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachy- or bradyarrhythmias</td>
</tr>
<tr>
<td>Aortic dissection and severe aortic valve disease</td>
</tr>
<tr>
<td>Hypo- or hypertension, e.g. haemorrhagic shock, hypertensive emergency</td>
</tr>
<tr>
<td>Acute and chronic HF without significant concomitant CAD</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Coronary vasculitis, e.g. systemic lupus erythaematosus, Kawasaki syndrome</td>
</tr>
<tr>
<td>Coronary endothelial dysfunction without significant CAD e.g. cocaine abuse</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Damage not related to myocardial ischaemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac contusion</td>
</tr>
<tr>
<td>Cardiac incisions with surgery</td>
</tr>
<tr>
<td>Radiofrequency or cryoablation therapy</td>
</tr>
<tr>
<td>Rhabdomyolysis with cardiac involvement</td>
</tr>
<tr>
<td>Myocarditis</td>
</tr>
<tr>
<td>Cardiotoxic agents, e.g. anthracyclines, herceptin, carbon monoxide poisoning</td>
</tr>
<tr>
<td>Severe burns affecting &gt; 30% of body surface</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indeterminant or multifactorial group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apical ballooning syndrome</td>
</tr>
<tr>
<td>Severe pulmonary embolism or pulmonary hypertension</td>
</tr>
<tr>
<td>Peripartum cardiomyopathy</td>
</tr>
<tr>
<td>Renal failure</td>
</tr>
<tr>
<td>Severe acute neurological disease e.g. stroke, trauma</td>
</tr>
<tr>
<td>Infiltrative disease e.g. amyloidosis, sarcoidosis</td>
</tr>
<tr>
<td>Extreme exertion</td>
</tr>
<tr>
<td>Sepsis</td>
</tr>
<tr>
<td>Acute respiratory failure</td>
</tr>
<tr>
<td>Frequent defibrillator shocks</td>
</tr>
</tbody>
</table>

### APPENDIX III: TIMI RISK SCORE FOR STEMI

<table>
<thead>
<tr>
<th>Categories</th>
<th>Options</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>□ &lt; 65</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>□ 65 - 74</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>□ ≥ 75</td>
<td>3</td>
</tr>
<tr>
<td>Weight &lt; 67 kg</td>
<td>□ Yes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>□ No</td>
<td>0</td>
</tr>
<tr>
<td>SBP &lt; 100 mmHg</td>
<td>□ Yes</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>□ No</td>
<td>0</td>
</tr>
<tr>
<td>Heart rate &gt; 100 bpm</td>
<td>□ Yes</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>□ No</td>
<td>0</td>
</tr>
<tr>
<td>Killip Class II-IV</td>
<td>□ Yes</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>□ No</td>
<td>0</td>
</tr>
<tr>
<td>Anterior ST segment elevation or LBBB</td>
<td>□ Yes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>□ No</td>
<td>0</td>
</tr>
<tr>
<td>Diabetes, history of hypertension, history of angina</td>
<td>□ Yes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>□ No</td>
<td>0</td>
</tr>
<tr>
<td>Time to treatment &gt; 4 hours</td>
<td>□ Yes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>□ No</td>
<td>0</td>
</tr>
</tbody>
</table>

**Note:**
TIMI Risk Score: 0 – 14 plausible points
- Low and moderate risk: 5 points and below
- High-risk: 6 points and above
APPENDIX IV: GRACE ACS RISK MODEL

A) At Admission (in-hospital/to 6 months)

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<td>□ 150 – 199</td>
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<td></td>
<td>□ ≥ 200</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>□ &lt;80</td>
</tr>
<tr>
<td></td>
<td>□ 80 – 99</td>
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<tr>
<td></td>
<td>□ 100 – 119</td>
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<td>□ 120 – 139</td>
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<td>□ 140 – 159</td>
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<td></td>
<td>□ 160 – 199</td>
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<tr>
<td></td>
<td>□ ≥ 200</td>
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<tr>
<td>Creatinine (mg/dL)</td>
<td>□ 0 – 0.39</td>
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<td></td>
<td>□ 0.4 – 0.79</td>
</tr>
<tr>
<td></td>
<td>□ 0.8 – 1.19</td>
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<tr>
<td></td>
<td>□ 1.2 – 1.59</td>
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<tr>
<td></td>
<td>□ 1.6 – 1.99</td>
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<tr>
<td></td>
<td>□ 2.0 – 3.99</td>
</tr>
<tr>
<td></td>
<td>□ ≥ 4</td>
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<tr>
<td>CHF (Killip Class)</td>
<td>□ I (No CHF)</td>
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<tr>
<td></td>
<td>□ II (Rales and/or jugular venous distention)</td>
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<td></td>
<td>□ III (Pulmonary oedema)</td>
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<tr>
<td></td>
<td>□ IV (Cardiogenic shock)</td>
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<td>Cardiac arrest at admission</td>
<td>Yes</td>
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<tr>
<td>ST segment deviation</td>
<td>Yes</td>
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<tr>
<td>Elevated cardiac enzymes/markers</td>
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### B) At Discharge (to 6 months)

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<tr>
<td>Heart rate (bpm)</td>
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<td>Creatinine (mg/dL)</td>
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<td>≥ 4</td>
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<tr>
<td>Congestive HF</td>
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<td>In-hospital PCI</td>
<td>Yes</td>
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<td>In-hospital CABG</td>
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<td>Past history of MI</td>
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<td>Elevated cardiac enzymes/markers</td>
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APPENDIX V: CHA₂DS²- VASc SCORE

<table>
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<tr>
<th>Categories</th>
<th>Options</th>
<th>Points</th>
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<td>&lt; 65</td>
<td>0</td>
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<tr>
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<td>65 - 74</td>
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<td>≥ 75</td>
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</tr>
<tr>
<td></td>
<td>Female</td>
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</tr>
<tr>
<td>Congestive HF history</td>
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<td>1</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>Hypertension history</td>
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<td>Stroke/TIA history</td>
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<td>Vascular disease history</td>
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<tr>
<td></td>
<td>No</td>
<td>0</td>
</tr>
</tbody>
</table>

Score:
- CHA₂DS²- VASc score = 1: low to moderate-risk and should consider antiplatelet/ anticoagulation.
- CHA₂DS²- VASc score ≥ 2: moderate to high-risk and should be anticoagulated.
**APPENDIX VI: CALCULATION OF CrCl BY COCKCROFT-GAULT EQUATION**

Estimated GFR (eGFR) (ml/minute) = \((140 - \text{age}) \times \text{weight \over (0.814 \times S\text{Cr} \; [\mu\text{mol/L}])}\) or \(1.2 \times \left(\frac{140 - \text{age}}{\text{S\text{Cr} \; [\mu\text{mol/L}]}}\right)\)

Note: For women, multiply by 0.85

**Severity Of CKD**

<table>
<thead>
<tr>
<th>Severity of CKD</th>
<th>CrCl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal to mild</td>
<td>&gt; 60 ml/minute</td>
</tr>
<tr>
<td>Moderate</td>
<td>30-59 ml/minute</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt; 30 ml/minute</td>
</tr>
</tbody>
</table>

**APPENDIX VII: CALCULATION OF CrCl BY MDRD EQUATION**

- GFR (ml/minute/1.73m²) = \(175 \times \text{Scr}^{-1.154} \times \text{Age}^{-0.203} \times 0.742 \times 1.212\) (if African American).

**APPENDIX VIII: CALCULATION OF CrCl BY CKD-EPI EQUATION**

- GFR = \(141 \times \min(\text{Scr}/k, 1)\alpha \times \max(\text{Scr}/k, 1) - 1.209 \times 0.993\text{Age} \times 1.018\) \[if female\] X 1.159 \[if black\].

- Where Scr is serum creatinine (mg/dL), \(k\) is 0.7 for females and 0.9 for males, \(\alpha\) is \(-0.329\) for females and \(-0.411\) for males, \(\min\) indicates the minimum of Scr/k or 1, and \(\max\) indicates the maximum of Scr/k or 1.
## APPENDIX IX: PREVENTION OF CIN

<table>
<thead>
<tr>
<th>Agent</th>
<th>Concentration</th>
<th>Dose /Flow Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium Chloride (NaCl)</td>
<td>0.9% solution</td>
<td>The regime used would vary depending upon the patient’s condition and time of admission in relation to the procedure. These regimes include:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Rate of 1.0-1.5 ml/kg/hour for 3-12 hours before and 6-24 hours after the procedure ensuring a urine flow rate of 150 ml/hour.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- 0.9% NaCl @ 1 ml/kg/hour for 12 hours pre- and for 12 hours post-contrast administration. Reduce rate to 0.5 ml/kg/hour if LVEF &lt; 40%.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Isotonic NaCl @ 3 ml/kg/hour for 1-3 hours pre- and for 6 hours post-contrast administration.</td>
</tr>
<tr>
<td>Sodium bicarbonate (NaHCO3)</td>
<td>154 mEq/L in 5% dextrose in water (154 ml of 1000 mEq/l of NaHCO3 + 850 ml of 5% dextrose)</td>
<td>3 ml/kg/hour for 1 hour before the contrast followed by an infusion of 1 ml/kg/hour for 6 hours after the procedure.</td>
</tr>
<tr>
<td>N-acetylcysteine</td>
<td>Oral 1200 mg bd, one day before and one day after the contrast. IV 1200 pre-procedure and 1200 mg bd x 48 hours.</td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX X: PREVENTION OF CIN (GRADE OF RECOMMENDATION AND LEVEL OF EVIDENCE)

<table>
<thead>
<tr>
<th>ACC/ESC Classification</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I, B</td>
<td>Use minimal volume.</td>
</tr>
<tr>
<td>I, C</td>
<td>Avoid nephrotoxic agents e.g. NSAIDs</td>
</tr>
<tr>
<td>II-a, B</td>
<td>Isosmolar agent or low osmolar agents.</td>
</tr>
<tr>
<td>II-a, C</td>
<td>Use minimal volume.</td>
</tr>
<tr>
<td>II-a, B</td>
<td>Avoid nephrotoxic agents e.g. NSAIDs</td>
</tr>
<tr>
<td>II-a, C</td>
<td>Saline infusion</td>
</tr>
<tr>
<td>II-a, B</td>
<td>NaHCO3</td>
</tr>
<tr>
<td>II-a, B</td>
<td>Acetylcysteine</td>
</tr>
<tr>
<td>II-a, C</td>
<td>Pre-treatment with high dose statins</td>
</tr>
</tbody>
</table>

APPENDIX XI: CLINICAL AUDIT INDICATORS FOR STEMI

\[
\text{Number of patients with suspected ACS who had an ECG done within 10 minutes of arrival in the emergency department} = \frac{\text{Number of patients with suspected ACS who had an ECG done within 10 minutes of arrival in the emergency department}}{\text{Total number of patients with suspected ACS seen in the emergency department}} \times 100
\]

\[
\text{Number of patients with STEMI who received fibrinolytic therapy within 30 minutes (DNT)} = \frac{\text{Number of patients with STEMI who received fibrinolytic therapy within 30 minutes (DNT)}}{\text{Total number of patients with STEMI who received fibrinolytic therapy}} \times 100
\]

\[
\text{Number of patients with STEMI who received primary PCI within 90 minutes (DBT)} = \frac{\text{Number of patients with STEMI who received primary PCI within 90 minutes (DBT)}}{\text{Total number of patients with STEMI who underwent primary PCI}} \times 100
\]

\[
\text{Number of patients discharged on DAPT} = \frac{\text{Number of patients with STEMI who are discharged on DAPT}}{\text{Total number of patients with STEMI who are eligible for DAPT}} \times 100
\]
### APPENDIX XI: CLINICAL AUDIT INDICATORS FOR STEMI (continued)

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Formula</th>
</tr>
</thead>
</table>
| % of patients discharged on \(\beta\)-blockers | \[
\text{Number of patients with STEMI who are discharged on } \beta\text{-blockers} \times 100 \\
\text{Total number of patients with STEMI who are eligible for } \beta\text{-blockers}
\] |
| % of patients discharged on statins | \[
\text{Number of patients with STEMI who are discharged on statins} \times 100 \\
\text{Total number of patients with STEMI who are eligible for statins}
\] |
| % of patients discharged on ACE-I/ARB | \[
\text{Number of patients with STEMI who are discharged on ACE-I/ARB} \times 100 \\
\text{Total number of patients with STEMI who are eligible for ACE-I/ARB}
\] |
| % of patients who received an echocardiogram prior to discharge | \[
\text{Number of STEMI patients who received an echocardiogram prior to discharge} \times 100 \\
\text{Total number of patients admitted with STEMI}
\] |
| % of patients referred for cardiac rehabilitation before discharge | \[
\text{Number of STEMI patients referred for cardiac rehabilitation before discharge} \times 100 \\
\text{Total number of patients admitted with STEMI}
\] |
| % of patients receiving quit smoking advice | \[
\text{Number of STEMI patients receiving quit smoking advice} \times 100 \\
\text{Total number of patients admitted with STEMI who are smokers}
\] |
| % of patients who received ischaemic testing (e.g. sub-maximal stress test, stress echocardiogram) prior to discharge | \[
\text{Number of patients who received ischaemic testing (e.g. sub-maximal stress test, stress echocardiogram) prior to discharge} \times 100 \\
\text{Total number of patients admitted with STEMI}
\] |
APPENDIX XI: CLINICAL AUDIT INDICATORS FOR STEMI (continued)

- % of patients receiving primary PCI = \[
\frac{\text{Number of patients receiving primary PCI}}{\text{Total number of patients admitted with STEMI who are eligible for primary PCI}} \times 100
\]

- % of patients undergoing coronary angiography during index admission = \[
\frac{\text{Number of patients who underwent coronary angiography during index admission}}{\text{Total number of patients admitted with STEMI}} \times 100
\]

- % of in-hospital mortality = \[
\frac{\text{Number of STEMI deaths}}{\text{Total number of patients admitted with STEMI}} \times 100
\]

GLOSSARY

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABC</td>
<td>Airway, Breathing, Circulation</td>
</tr>
<tr>
<td>ACC</td>
<td>American College of Cardiology</td>
</tr>
<tr>
<td>ACE-I</td>
<td>Angiotensin Converting Enzyme Inhibitor</td>
</tr>
<tr>
<td>ACS</td>
<td>Acute Coronary Syndrome</td>
</tr>
<tr>
<td>ACT</td>
<td>Activated Clotting Time</td>
</tr>
<tr>
<td>ADP</td>
<td>Adenosine diphosphate</td>
</tr>
<tr>
<td>AF</td>
<td>Atrial Fibrillation</td>
</tr>
<tr>
<td>AHA</td>
<td>American Heart Association</td>
</tr>
<tr>
<td>AMI</td>
<td>Acute Myocardial Infarction</td>
</tr>
<tr>
<td>APTT</td>
<td>Activated Partial Thromboplastin Time</td>
</tr>
<tr>
<td>ARB</td>
<td>Angiotensin Receptor Blocker</td>
</tr>
<tr>
<td>AST</td>
<td>Aspartate Aminotransferase</td>
</tr>
<tr>
<td>AV</td>
<td>Atrio-ventricular</td>
</tr>
<tr>
<td>bd</td>
<td>Bis Die (twice daily)</td>
</tr>
<tr>
<td>BiPaP</td>
<td>Bi-level Positive Airway Pressure</td>
</tr>
<tr>
<td>BMS</td>
<td>Bare Metal Stents</td>
</tr>
<tr>
<td>BP</td>
<td>Blood Pressure</td>
</tr>
<tr>
<td>CABG</td>
<td>Coronary Artery Bypass Graft</td>
</tr>
<tr>
<td>CAD</td>
<td>Coronary Artery Disease</td>
</tr>
<tr>
<td>CCU</td>
<td>Cardiac Care Unit</td>
</tr>
<tr>
<td>CHD</td>
<td>Coronary Heart Disease</td>
</tr>
<tr>
<td>CIN</td>
<td>Contrast Induced Nephropathy</td>
</tr>
<tr>
<td>CK</td>
<td>Creatine Kinase</td>
</tr>
<tr>
<td>CKD</td>
<td>Chronic Kidney Disease</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
</tr>
<tr>
<td>--------------</td>
<td>-------------</td>
</tr>
<tr>
<td>CKD-EPI</td>
<td>Chronic Kidney Disease Epidemiology Collaboration</td>
</tr>
<tr>
<td>CK-MB</td>
<td>Creatine Kinase-Myocardial Band</td>
</tr>
<tr>
<td>CPG</td>
<td>Clinical Practice Guidelines</td>
</tr>
<tr>
<td>CPR</td>
<td>Cardiopulmonary Resuscitation</td>
</tr>
<tr>
<td>CrCL</td>
<td>Creatinine Clearance</td>
</tr>
<tr>
<td>CRP</td>
<td>Cardiac Rehabilitation Programme</td>
</tr>
<tr>
<td>cTn</td>
<td>Cardiac Troponins</td>
</tr>
<tr>
<td>cTnI</td>
<td>Cardiac Troponin I</td>
</tr>
<tr>
<td>cTnT</td>
<td>Cardiac Troponin T</td>
</tr>
<tr>
<td>CV</td>
<td>Cardioversion</td>
</tr>
<tr>
<td>CVD</td>
<td>Cardiovascular Disease</td>
</tr>
<tr>
<td>CPAP</td>
<td>Continuous Positive Airway Pressure</td>
</tr>
<tr>
<td>D5W</td>
<td>5% dextrose in water</td>
</tr>
<tr>
<td>DAPT</td>
<td>Dual Antiplatelet Therapy</td>
</tr>
<tr>
<td>DBT</td>
<td>Door to Balloon Time</td>
</tr>
<tr>
<td>DES</td>
<td>Drug Eluting Stents</td>
</tr>
<tr>
<td>DM</td>
<td>Diabetes Mellitus</td>
</tr>
<tr>
<td>DNT</td>
<td>Door to Needle Time</td>
</tr>
<tr>
<td>DVT</td>
<td>Deep Venous Thrombosis</td>
</tr>
<tr>
<td>ECG</td>
<td>Electrocardiogram</td>
</tr>
<tr>
<td>EF</td>
<td>Ejection Fraction</td>
</tr>
<tr>
<td>eGFR</td>
<td>Estimated Glomerular Filtration Rate</td>
</tr>
<tr>
<td>ESC</td>
<td>European Society of Cardiology</td>
</tr>
<tr>
<td>FMC</td>
<td>First Medical Contact</td>
</tr>
<tr>
<td>GFR</td>
<td>Glomerular Filtration Rate</td>
</tr>
<tr>
<td>Gp</td>
<td>Glycoprotein</td>
</tr>
<tr>
<td>GRACE</td>
<td>Global Registry of Acute Coronary Events</td>
</tr>
<tr>
<td>GTN</td>
<td>Glyceryl trinitrate</td>
</tr>
<tr>
<td>HF</td>
<td>Heart Failure</td>
</tr>
<tr>
<td>HRT</td>
<td>Hormone Replacement Therapy</td>
</tr>
<tr>
<td>HTA</td>
<td>Health Technology Assessment</td>
</tr>
<tr>
<td>IABP</td>
<td>Intra-aortic Balloon Pump</td>
</tr>
<tr>
<td>IC</td>
<td>Intracoronary</td>
</tr>
<tr>
<td>ICD</td>
<td>Implantable Cardioverter-Defibrillator</td>
</tr>
<tr>
<td>INR</td>
<td>International Normalised Ratio</td>
</tr>
<tr>
<td>IO</td>
<td>Intraosseous</td>
</tr>
<tr>
<td>IRA</td>
<td>Infarct-Related Artery</td>
</tr>
<tr>
<td>IV</td>
<td>Intravenous</td>
</tr>
<tr>
<td>LBBB</td>
<td>Left Bundle Branch Block</td>
</tr>
<tr>
<td>LDH</td>
<td>Lactate Dehydrogenase</td>
</tr>
<tr>
<td>LDL</td>
<td>Low Density Lipoprotein</td>
</tr>
<tr>
<td>LDL-C</td>
<td>Low Density Lipoprotein Cholesterol</td>
</tr>
<tr>
<td>LMWH</td>
<td>Low Molecular Weight Heparin</td>
</tr>
<tr>
<td>LV</td>
<td>Left Ventricular</td>
</tr>
<tr>
<td>LVEF</td>
<td>Left Ventricular Ejection Fraction</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
</tr>
<tr>
<td>--------------</td>
<td>-------------</td>
</tr>
<tr>
<td>LVH</td>
<td>Left Ventricular Hypertrophy</td>
</tr>
<tr>
<td>MDRD</td>
<td>Modification of Diet in Renal Disease</td>
</tr>
<tr>
<td>MI</td>
<td>Myocardial Infarction</td>
</tr>
<tr>
<td>MOH</td>
<td>Ministry of Health Malaysia</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
</tr>
<tr>
<td>MSCT</td>
<td>Multi-Slice Computed Tomography</td>
</tr>
<tr>
<td>NaCl</td>
<td>Sodium Chloride</td>
</tr>
<tr>
<td>NaHCO3</td>
<td>Sodium Bicarbonate</td>
</tr>
<tr>
<td>NCVD</td>
<td>National Cardiovascular Disease Database</td>
</tr>
<tr>
<td>NHAM</td>
<td>National Heart Association Malaysia</td>
</tr>
<tr>
<td>NSAID</td>
<td>Non-steroidal Anti-Inflammatory Drug</td>
</tr>
<tr>
<td>NSTEMI</td>
<td>Non ST Segment Elevation Myocardial Infarction</td>
</tr>
<tr>
<td>od</td>
<td>Once daily</td>
</tr>
<tr>
<td>PCI</td>
<td>Percutaneous Coronary Interventions</td>
</tr>
<tr>
<td>PCWP</td>
<td>Pulmonary Capillary Wedge Pressure</td>
</tr>
<tr>
<td>PEA</td>
<td>Pulseless Electrical Activity</td>
</tr>
<tr>
<td>ROSC</td>
<td>Return of Spontaneous Circulation</td>
</tr>
<tr>
<td>r-TPA</td>
<td>Recombinant Tissue Plasminogen Activator</td>
</tr>
<tr>
<td>RV</td>
<td>Right Ventricular</td>
</tr>
<tr>
<td>RVF</td>
<td>Right Ventricular Infarction</td>
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<tr>
<td>SBP</td>
<td>Systolic Blood Pressure</td>
</tr>
<tr>
<td>SC</td>
<td>Subcutaneous</td>
</tr>
<tr>
<td>Scr</td>
<td>Serum Creatinine</td>
</tr>
<tr>
<td>SpO2</td>
<td>Oxygen Saturation</td>
</tr>
<tr>
<td>STEMI</td>
<td>ST Segment Elevation Myocardial Infarction</td>
</tr>
<tr>
<td>tds</td>
<td>Tri die sumendus (three times per day)</td>
</tr>
<tr>
<td>TIA</td>
<td>Transient Ischaemic Attack</td>
</tr>
<tr>
<td>TIMI</td>
<td>Thrombolysis in Myocardial Infarction</td>
</tr>
<tr>
<td>TMP</td>
<td>TIMI Myocardial Perfusion Grade</td>
</tr>
<tr>
<td>TNK-tPA</td>
<td>Tenecteplase</td>
</tr>
<tr>
<td>TVR</td>
<td>Target Vessel Revascularisation</td>
</tr>
<tr>
<td>UFH</td>
<td>Unfractionated Heparin</td>
</tr>
<tr>
<td>ULRR</td>
<td>Upper Limit Reference Range</td>
</tr>
<tr>
<td>URL</td>
<td>Upper Reference Limits</td>
</tr>
<tr>
<td>VF</td>
<td>Ventricular Fibrillation</td>
</tr>
<tr>
<td>VPC</td>
<td>Ventricular Premature Contractions</td>
</tr>
<tr>
<td>VSD</td>
<td>Ventricular Septal Defect</td>
</tr>
<tr>
<td>VT</td>
<td>Ventricular Tachycardia</td>
</tr>
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</table>
REFERENCES


<table>
<thead>
<tr>
<th>Reference</th>
<th>Title and Details</th>
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301. Kumler T, Gislason GH, Kober L, Gustafsson F, Schou M, Torp-Pedersen C. Renal function at the time of a myocardial infarction maintains prognostic value for more than 10 years. BMC Cardiovasc Disord. 2011;11:37.


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