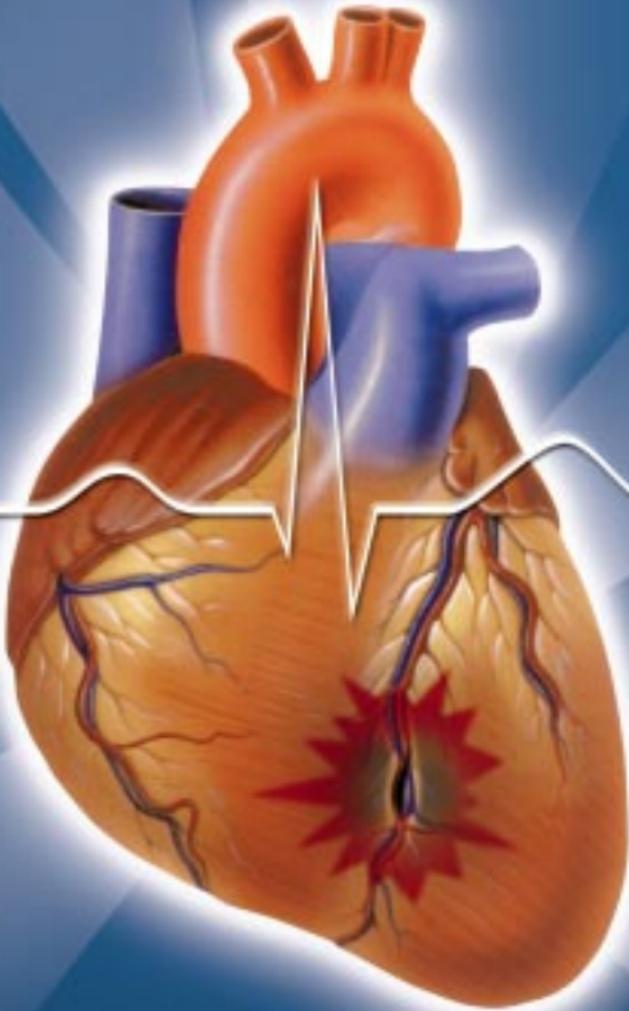


2001

**Clinical Practice Guidelines on
Acute Myocardial Infarction**



Ministry of Health



*National Heart
Association of Malaysia*



Academy of Medicine



Acute Myocardial Infarction is a major health problem, with relatively high morbidity and mortality. Although mortality has been reduced, it is still substantial. In the management of established acute myocardial infarction, time is the essence. These guidelines will help medical and health personal deal with rapid recognition and management of the patient. These guidelines are based on currently available evidence and the evidence is graded accordingly.

It is hopeful that this Clinical Practice Guidelines will indeed benefit the medical community. I am grateful to the panel of experts who put together these guidelines, those who attended the presentation of the final draft and the secretariat involved.

I would also like to thank Novartis for the grant provided to make these guidelines a reality.

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1. INTRODUCTION

Cardiovascular disease is the commonest cause of mortality in government hospitals accounting for 24.5% of all deaths for the year 1998.¹ Coronary heart disease (CHD) is the major cause of these deaths.

In more advanced countries, mortality from heart disease has decreased due to reduction in risk factors such as smoking and life style changes and better management of heart disease.²

Much progress has been made in the management of acute myocardial infarction especially in the last two decades. The majority of deaths occur soon after the onset of symptoms, the "pre-hospital phase". To reduce these deaths, the public needs to be aware of the symptoms of an infarct and to seek medical attention immediately. Healthcare personnel should be trained to deal with these patients appropriately and hospitals need to develop management strategies to maximize treatment benefits.

Guidelines in the management of acute myocardial infarction will help in addressing these issues. Treatment strategies have been graded based on levels of evidence using the system outlined below:-

GRADING OF RECOMMENDATIONS ACCORDING TO LEVELS OF EVIDENCE	
GRADE A	Based on evidence from one or more randomized clinical trials
GRADE B	Based on evidence from high quality clinical trials but no randomized clinical trial data available
GRADE C	Based on expert committee reports and/or clinical experience of respected authorities but lacking in directly applicable studies of good quality

2. DEFINITION

Acute myocardial infarction (AMI) is necrosis of heart muscle due to inadequate blood supply following an acute coronary occlusion. This occlusion is usually due to plaque rupture or fissuring with superimposed thrombosis. Rarely, this may result from coronary spasm, coronary embolism or vasculitis.³



3. PRE-HOSPITAL MANAGEMENT

Management of AMI begins in the pre-hospital phase with education of both the general public and healthcare providers.

3.1 General Public

3.1.1 Awareness and Early Recognition

Patients with CHD, persons with multiple risk factors, their relatives and the general public should receive education and counselling about:

- risk factors for CHD.
- symptoms of heart disease - chest pain, palpitations, difficulty in breathing.
- symptoms of AMI.
- the importance of early treatment.
- immediate measures to be taken in the event of the above symptoms.

Public education can be achieved via the mass media, public forums, schools, resource centres in hospitals, patient education classes, patient literature and the internet.

3.1.2 Risk Factors for CHD

Epidemiological studies have identified the following major independent risk factors for CHD.^{4,5} These include:

- Cigarette smoking, of any amount
- Elevated total and LDL cholesterol
- Elevated blood pressure
- Low HDL cholesterol
- Diabetes mellitus
- Advancing age

The predictive power of each of these major risk factors in determining an individual's global risk for CHD, is additive. An individual with more risk factors is at higher risk of developing atherosclerotic disease.

In addition, there are other risk factors that have been associated with an increased risk of CHD. These are the predisposing risk factors and the conditional risk factors.





Predisposing risk factors are those that worsen the risk associated with the independent risk factors. These are:

- Obesity (BMI > 30 kg/m²)⁶
- Abdominal obesity (waist circumference, men > 102 cm and women > 88 cm; waist-hip ratio, men > 0.9 and women > 0.8)⁷
- Physical inactivity
- Family history of premature CHD (Male sibling or parent with CHD < 55 years and/or female parent or first degree relative with CHD < 65 years)
- Ethnic origin
- Psychosocial factors

Conditional risk factors⁵ are associated with an increased risk for CHD although their causative and independent contributions to CHD have not been well documented. These include^{7,8,9,10,11,12} :

- Elevated serum triglycerides
- Elevated serum homocysteine
- Elevated serum lipoprotein (a)
- Prothrombotic factors (e.g. fibrinogen)
- Inflammatory markers (e.g. C-reactive proteins)

It is important to assess an individual's global risk for CHD. This can be done using either the American Heart Association and American College of Cardiology Multiple Risk Factor Assessment Equations⁹ or Coronary Risk Charts produced by the European Society of Cardiology.¹³ By these means it is possible to prioritize the intensity of efforts at risk reduction. Efforts to reduce the global risk in high risk individuals and in all persons with established atherosclerotic disease should be more intense than in those at low risk.

3.1.3 Symptoms of CHD

Patients with ischaemic chest pain may present with stable angina, unstable angina or AMI. Patients with the following chest pain syndromes merit early medical attention:-

- New onset chest pain (< 1 month)
- A change in a previously stable pattern of angina, such as an increase in the frequency or severity of chest pain or the occurrence of rest pain
- Chest pain that is not relieved by rest and/or nitroglycerine (GTN)

Other symptoms that warrant immediate medical attention include breathlessness, lightheadedness and faints.





3.1.4 Symptoms of AMI

Chest pain due to AMI is usually retrosternal, usually lasts at least 20 minutes, but may be shorter in duration. It may occur at rest or with activity. The pain is usually central or in the left chest and may radiate to the jaw or down the left upper limb. It may be crushing, pressing or burning in nature. The severity of the pain is variable.

Occasionally the pain may occur in the epigastric region and may be misinterpreted as indigestion and heart burn. Symptoms also include unexplained nausea and vomiting, weakness, dizziness, lightheadedness and syncope which may occur in the presence or absence of chest pain. When these symptoms occur suddenly and are severe in onset, it should raise the suspicion of an AMI.

In patients with CHD, deterioration in the usual pattern of stable angina may indicate an unstable plaque. This may progress to an infarct.

Diabetics, elderly and females may not present with typical ischaemic type chest pain. Common presenting symptoms in these patients are dyspnoea and atypical chest pains.

3.1.5 Importance of Early Treatment

Early relief of pain is important for humane reasons and to reduce the associated sympathetic activation. The latter causes tachycardia, vasoconstriction and elevated blood pressure thus increasing the work of the heart.

Early treatment of AMI is also associated with improved survival. This is because:-

- most early deaths are due to ventricular fibrillation, which is treatable. About 30% of deaths due to AMI occur within the first hour; 60% of deaths occur outside the hospital. Thus the general public and the family of patients with CHD should learn cardiopulmonary resuscitation and basic life support .
- early reperfusion therapy either by *thrombolytic therapy^{14,15,16} or primary **percutaneous transluminal coronary angioplasty (PTCA)^{17,18} reduces infarct size and improves survival.

* The newer preferred term is "fibrinolytic therapy" but for the purpose of this guideline, the older and more frequently used term "thrombolytic therapy" will be used.

** The newer preferred term is percutaneous coronary intervention (PCI) but the older and more frequently used term PTCA will be used in this guideline.



3.1.6 Immediate measures to be taken in suspected cases of AMI

For the general public: -

- Seek immediate medical attention at the nearest hospital.
- Call for an ambulance (dial 999) 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 300mg tablet of aspirin immediately.

Should patients with suspected AMI be seen by the general practitioner/ family physician, suggest:

- Ask patient to chew and swallow one 300mg tablet of aspirin.
- Give sublingual GTN.
- If available confirm the diagnosis with an ECG.
- Wherever possible, set up intravenous access.
- Pain relief with intravenous opiates (IV morphine 3-5mg slowly).
- Avoid intramuscular injections since this could result in intramuscular hematomas if thrombolytic agents are subsequently administered.
- Ask a relative or friend, or call an ambulance to send the patient quickly to the nearest hospital.
- Wherever possible, contact the doctor at the hospital so that the patient can be treated promptly on arrival.

Patients with CHD:

Patients should take one dose of sublingual GTN at the onset of ischaemic type chest discomfort and this may be repeated at 5 minute intervals for a total of 3 doses. If symptoms persist after 15 minutes, the patient should be rapidly transported to a hospital.

3.2 Health Care Personnel

Health care personnel should be trained:-

- to identify patients at high risk of developing CHD.
- to identify patients presenting with AMI.
- on the importance of early referral and treatment.
- in basic life support and cardiopulmonary resuscitation (CPR).

Patients suspected of having an AMI should be given sublingual GTN and oral aspirin immediately. An ECG should be done as soon as possible.



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

- Nature of complaint noted.
- Obtain address 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 at least two trained paramedics immediately.
- Patient should be given oxygen and aspirin (if he has not taken) and transported to hospital.
- Upon reaching the hospital, the patient should be taken directly to the Emergency Department.

4. IN-HOSPITAL MANAGEMENT

Early management of AMI is directed at:

- pain relief.
- establishing early reperfusion.
- treatment of arrhythmias.

4.1 Diagnosis

The diagnosis of AMI according to the World Health Organization (WHO) definition is based on the presence of at least two of the following three criteria¹⁹:

- A. Clinical history of ischaemic type chest discomfort.
- B. Evolutionary changes on serially obtained ECG tracings.
- C. A rise and fall in serum cardiac markers.

Recently, the European Society of Cardiology and the American College of Cardiology have proposed a new definition of AMI.²⁰ This new definition incorporates sensitive and specific serological markers and newer imaging techniques that are able to detect small infarcts that would not have been considered AMI previously.

4.1.1 History (as described earlier in 3.1.4)

4.1.2 Electrocardiographic changes

The evolutionary ECG changes of AMI include hyperacute changes of a tall peaked T-wave, ST segment elevation followed by the development of Q-wave, return of the ST segment to isoelectric and T-wave inversion. The cut off points for new or presumed new ST segment elevation at the J point are $\geq 0.2\text{mV}$ in leads V_1 , V_2 , or V_3 and $\geq 0.1\text{mV}$ in other leads. This should be present in 2 or more contiguous leads. The presence of new onset bundle branch block in a patient with typical type chest pain indicates an infarct.

The ECG criteria for clinically established AMI includes any QR wave in lead V_1 through $V_3 \geq 30$ msec (0.03 second) and/or any abnormal Q wave in leads I, II, aVL, aVF, V_4 to V_6 in any 2 contiguous leads at least 1mm in depth. These changes must be present in at least 2 consecutive ECGs.

However in some cases the ECG may be normal or show minor ST-T changes or occasionally ST segment depression (posterior and non-Q wave infarct). In patients with ongoing chest pain and in whom the clinical index of suspicion of an AMI is high, 12 lead ECG tracings repeated at close intervals of time might show evolving changes. Comparison with previous ECG's may also be helpful in such situations.

In patients with inferior infarct, one should look for posterior and RV infarct. The latter requires right sided chest leads which should be done soon after admission.

For localization of infarct based on ECG, see Table 1.

Table 1: ECG patterns of various myocardial infarction locations

Location	Leads	ECG findings
Anterior	V1-V4	ST elevation, Q wave
Anteroseptal	V1-V3	ST elevation, Q wave
Extensive anterior	V1-V6	ST elevation, Q wave
Posterior	V1-V2	ST depression, tall R wave
Lateral	I, AVL, V5-V6	ST elevation, Q wave
Inferior	II, III, AVF	ST elevation, Q wave
Right Ventricular	V4R, V5R	ST elevation, Q wave



4.1.3 Serum Cardiac Markers.

These include²¹:-

- Creatine kinase-Myocardial Band (CK-MB)
- Cardiac troponins (cTnT and cTnI)
- Creatine kinase (CK)
- Aspartate amino transferase (AST)
- Lactate dehydrogenase (LDH)
- Myoglobin

For the relative timing, rate of rise, peak value and duration of elevation of these cardiac markers following AMI, see Figure 1.

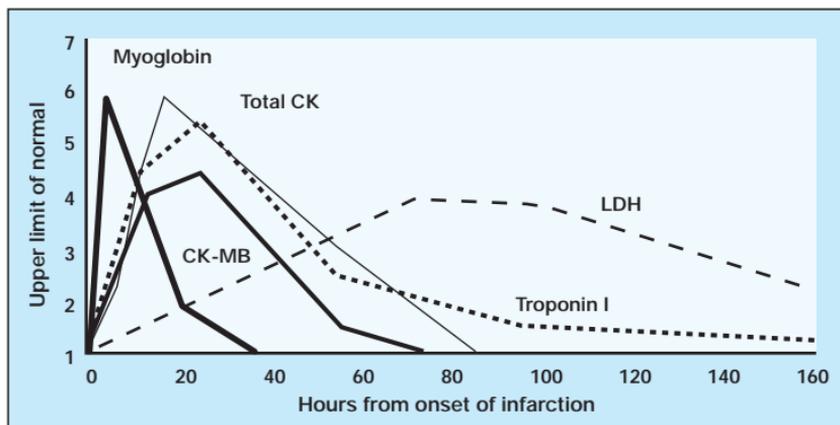


FIGURE 1. Time course of elevations of serum markers after AMI. This figure summarizes the relative timing, rate of rise, peak values and duration of elevation above the upper limit of normal of multiple serum markers following AMI.

The more specific cardiac markers are the cardiac troponins and CK-MB. Serum myoglobin rises and peaks early following AMI but is non-specific.

It is recommended that measurement of cardiac markers be done at periodic intervals. This would help to establish or exclude the diagnosis and may be useful for an estimation of infarct size. Of the cardiac markers, the most useful are cardiac troponins and CK-MB. It takes about 4-8 hours after an AMI for cardiac troponin and CK-MB levels to rise. Thus too early a measurement may result in a falsely low level of these cardiac markers. The cardiac marker LDH remains elevated up to 8 days and cardiac troponins up to 14 days after an infarct. Thus these markers are useful in patients presenting late.



For most patients, blood should be obtained for testing on hospital admission, and again at 12-24 hours. If the ECG is not diagnostic and the first blood sample is negative but the clinical index of suspicion of an AMI is high, a second sample may be taken at 4-9 hours. In most situations, elevated values for cardiac markers should be recorded on 2 consecutive blood samples to diagnose AMI.

Grade A

A single measurement of a raised Troponin T or I²² (99th percentile of the values for a reference control group) is sufficient to indicate myocardial necrosis. Values for CK-MB should rise and fall. Persistently elevated values without change are almost never due to AMI*.

Measurement of AST and LDH levels is fraught with frequent false positive elevations and should not be used to diagnose AMI. However, in situations where it is not possible to obtain more specific cardiac markers, these may be used.²³

* It is important that each individual laboratory maintains appropriate quality control in the measurement of these cardiac markers.

4.1.4 Other Diagnostic Modalities.

Imaging techniques such as echocardiography²⁴ and radionuclide techniques²⁵ are useful diagnostic tools in the patient presenting with acute chest pain. They help to:

- rule out or confirm the presence of acute infarction or ischaemia.
- identify non-ischaemic conditions causing chest pain such as valvular heart disease, pulmonary embolism, aortic dissection, etc.
- identify mechanical complications of acute infarction.
- provide prognostic information.

Echocardiography is a particularly useful bedside imaging technique in difficult diagnostic situations.



4.2 Initial Recognition and Management

When the patient with suspected AMI reaches the emergency department, evaluation and initial management should take place promptly (FAST TRACK) because the benefits of reperfusion therapy is greater the earlier it is initiated.

As soon as the patient reaches the Emergency Room, a quick history should be taken and the vital signs noted. The diagnosis should be confirmed with an ECG which should be accomplished as soon as possible preferably within 10 minutes of the patient's arrival in the emergency department. It is important to relieve pain and quickly assess the patient's suitability for reperfusion by either thrombolytic therapy or direct PTCA.

The following should be done immediately and concomitantly (see flow chart 1, page 39):-

- Assessment and stabilization of the patients' haemodynamics.
- Continuous ECG monitoring.

Grade A

- Aspirin. A dose of 150-300mg crushed or chewed to achieve rapid action.

Grade A

- Oxygen by nasal prongs / facemask.

Grade C

- Sublingual GTN (unless systolic arterial pressure < 90 mmHg).
- Venous access established and blood taken for cardiac markers, full blood count, renal profile, sugar and lipid profile. Preferably 2 intravenous lines should be set up.

Grade B

- Pain relief - morphine should be administered intravenously 2-5mg every 5-15 minutes until pain is relieved or there is evidence of toxicity - hypotension, respiratory depression or severe vomiting. Anti-emetics (IV metoclopramide 10mg or promethazine 25mg) may be given simultaneously.

- Intramuscular injections should be avoided.

Grade A

- Assessment for reperfusion. Reperfusion can be achieved either by thrombolytic therapy or by primary PTCA.

4.3 Reperfusion Strategies

Early reperfusion is crucial because TIME LOST is equivalent to MYOCARDIUM LOST.

Grade A

4.3.1 Thrombolytic Therapy

4.3.1.1 Indications and Contraindications

The history and clinical examination should focus on indications and contraindications²⁶ for thrombolytic therapy.

Indications

Before thrombolytic therapy is initiated, the following criteria should be met.

1. Symptoms consistent with AMI (see 3.1.4)
2. Electrocardiographic changes:
 - ST segment elevation more than 0.1mV in at least two contiguous leads.
 - New or presumably new left bundle branch block.
3. Time from onset of symptoms²⁷:-
 - < 6 hours: most beneficial.
 - 6-12 hours: lesser but still important benefits.
 - > 12 hours: no significant benefit except in patients with ongoing ischaemia as manifested by persistent chest pain and ST elevation on the ECG

One should not wait for the results of cardiac markers before instituting thrombolytic therapy because this will result in unnecessary delay with resultant death of myocardium. If the patient has a history suggestive of AMI and the cardiac markers are raised but the ECG does not show ST segment elevation, then the diagnosis is acute coronary syndrome (non ST segment elevation AMI) and the patient should be treated as such with aspirin, heparin and anti anginal therapy. Thrombolytic therapy should not be administered.



Absolute Contraindications²⁶

1. Previous haemorrhagic stroke at any time; other strokes, cerebrovascular events within 1 year.
2. Known intracranial neoplasm.
3. Active internal bleeding (does not include menses).
4. Suspected aortic dissection.

*** Cautions/Relative Contraindications.**

1. Severe uncontrolled hypertension on presentation (blood pressure >180/110 mmHg).
2. History of prior cerebrovascular accident or known intracerebral pathology.
3. Current use of anticoagulants in therapeutic doses (INR >2-3); known bleeding diathesis.
4. Recent trauma (within 2-4 weeks), including head trauma or traumatic or prolonged (>10 minutes) CPR or major surgery (<3 weeks).
5. Non compressible vascular punctures.
6. Recent (within 2-4 weeks) internal bleeding.
7. For streptokinase: prior exposure (especially within 5 days to 2 years) or prior allergic reaction. Circulating antibodies against streptokinase can be present up to 2 years and can impair the action of the agent.²⁸ In these situations, consider the use of tissue plasminogen activator (tPA) or direct/primary PTCA. If neither of these options are available, consider the use of higher dose (up to 3 megaunits) streptokinase.²⁹ The use of tPA is not associated with antibody formation.
8. Pregnancy.
9. Active peptic ulcer.
10. History of chronic severe hypertension.

Grade C

*These should be considered on a case-to-case basis considering the risks versus benefits. In instances such as active peptic ulcer with history of bleeding, they become absolute contraindications when weighed against a less life-threatening evolving AMI.

4.3.1.2 Types of Thrombolytic Agents

Presently, the thrombolytic agents available locally are:

- Streptokinase. This is the most widely used agent.
Regimen: 1.5 million units in 100ml saline or 5% dextrose over 1 hour.
- Tissue Plasminogen Activator (tPA). Available data indicates that this achieves better reperfusion at 90 minutes as compared to streptokinase.^{15,30} However, there is a higher reocclusion rate and heparin (unfractionated heparin or low molecular weight heparin) needs to be administered routinely following completion of the tPA infusion. There is a small increased risk of intracranial haemorrhage particularly in the elderly.³¹ The use of tPA should be considered in the following patients who:
 - have been given streptokinase within the last 2 years.²⁸
 - are allergic to streptokinase.
 - have systolic blood pressure < 90 mmHg - tPA unlike streptokinase does not cause hypotension. (See also section on cardiogenic shock - 4.5.2.2)

Regimen: 15mg IV bolus followed by 0.75mg/kg over 30 minutes and then 0.5mg/kg over 60 minutes. Dose should not exceed 100mg.³⁰

Unfractionated heparin is administered as a bolus of 5000 units followed by 1000 units/hour adjusting the dose to maintain the aPTT of 1.5 to 2 times control.

Newer thrombolytic agents, not yet available locally, can be administered as a single bolus or double bolus injection. This enables earlier administration of these agents.

Early administration of a thrombolytic agent is important, as time lost is muscle lost. One should aim for a "door-to-needle time" of less than 30 minutes.

Thrombolytic therapy should be administered preferably in the Emergency Room. During administration of thrombolytic therapy, the following should be monitored:

- Cardiac rhythm
- Blood pressure

Administration of a thrombolytic agent is generally safe and complications are uncommon and easily treated (see below 4.3.1.4). Thrombolytic therapy can also be given in the small peripheral hospitals if the patient fulfils the indications and has no contraindications. Time should not be wasted transporting the patient to a large hospital before administering the agent. Remember, **time lost is myocardium lost**. The patient can then be transported to a larger hospital upon its completion. Low risk patients with a small uncomplicated inferior AMI may be managed in the peripheral hospital.



When transporting a patient who has been given thrombolytic therapy, we suggest that:

- these patients be handled with care.
- they be treated as stretcher cases with the head end raised.
- these patients should be transported as rapidly as possible to the nearest hospital.
- that the ambulance be fitted with a defibrillator and the patient be accompanied preferably by a doctor or a paramedic trained in ACLS.
- the cardiac rhythm should be monitored continuously.
- the doctor at the receiving hospital should be contacted so that the patient can be sent directly to CCU.

Should the patient with AMI present with acute pulmonary oedema or with arrhythmias, thrombolytic therapy should not be delayed until the complication is treated. The acute pulmonary oedema or the arrhythmias should be treated at the same time as the thrombolytic therapy is being given. Even if the patient presents with bradycardia or heart block, it is often possible to treat the arrhythmia with atropine, dopamine or adrenaline and administer thrombolytics simultaneously. Following successful clot lysis and establishment of reperfusion, the left ventricular failure and the arrhythmias usually improve.

4.3.1.3 Complications of Thrombolytic Treatment

• **Bleeding**

Bleeding usually occurs at sites of venepuncture and intra-muscular injections. Severe bleeding occurs in less than 1% of cases, the most catastrophic being intra-cerebral bleeding.^{27,31} If bleeding occurs the following should be done:

- the thrombolytic agent must be discontinued.
 - fresh frozen plasma should be given (2-4 units).
 - tranexamic acid (10mg/kg) by slow IV injection repeated after 30 minutes if necessary.
- ##### • **Hypotension (systolic BP < 90 mmHg)**

Hypotension may be due to the infarct per se, or the drugs administered. If due to thrombolytic therapy, it should be treated by:

- tilting the patient head down or elevating the legs.
- discontinuing the thrombolytic agent.
- administering fluids cautiously.
- inotropic therapy (dopamine) if required.

• **Allergic reactions**

Anaphylaxis is uncommon. If it occurs, it may be treated with anti-histamines and steroids. Routine administration of these agents prior to streptokinase infusion is not recommended.



4.3.1.4 Indicators of Successful Reperfusion

There is no sensitive bedside clinical method to reliably detect successful reperfusion.³² Some useful guides are:

- resolution of chest pain (may be confounded by the usage of narcotic analgesics).
- early return of ST segment elevation to isoelectric line or a decrease in the height of the ST elevation by 50% upon completion of the thrombolytic therapy.
- early peaking of CK levels.

The occurrence of 'reperfusion arrhythmias' is not a reliable indicator of successful reperfusion. An exception to this is accelerated idioventricular rhythm which has been correlated with a patent infarct related coronary artery after thrombolytic therapy or primary angioplasty.³³

4.3.1.5 Failed Thrombolysis

Failure of thrombolysis to open up the occluded infarct related artery is manifested as continuing chest pains and persistent ST segment elevation. These patients are more likely to develop complications such as heart failure and arrhythmias.

Grade C

They should be considered for rescue PTCA. If this is not possible, then a repeat administration of the same dose of thrombolytic agent may be considered.²⁹

4.3.2 Primary/Direct PTCA

Grade A

As a reperfusion strategy, PTCA has been shown to be more effective than thrombolytic therapy in achieving and sustaining a patent-infarct related artery.^{17,34,35,36} The availability of primary PTCA is however limited to only a few centres in the country. It should be considered in the following situations:

- as an alternative reperfusion strategy in experienced centres. It must be stressed that the door-to-balloon time beyond 60-90 minutes may not justify this method.
- in patients who have definite contraindications for thrombolytic therapy.
- in patients presenting with cardiogenic shock.



4.4 Coronary Care Unit (CCU) Management

4.4.1 General Measures

A period of at least 12 hours of complete bed rest is recommended following admission to CCU. Early ambulation in the patient with an uncomplicated infarct is encouraged. Patients with haemodynamic instability will need a longer period of bed rest.

Sedation may be useful.

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

The Valsalva maneuver has been shown to produce dangerous haemodynamic and electrocardiographic changes particularly in the young and thus prevention of constipation with stool softeners is encouraged.

4.4.2 Monitoring

The general condition of the patient, vital signs, pulse oximetry and the ECG should be continuously monitored looking for complications.

4.4.3 Adjunctive Pharmacotherapy

4.4.3.1 Oxygen

Grade B

Oxygen is indicated in the presence of hypoxemia, while in uncomplicated cases its use should probably be limited to the first day or less. Low dose oxygen at 2-4 litres/min is usually adequate. One should aim to maintain the pulse oximetry above 95%.

4.4.3.2 Aspirin

Grade A

Aspirin is standard therapy and should be administered to all patients and continued indefinitely.^{14,37,38} The initial dosage of 160-325mg should be followed by a maintenance dose of 75-325mg daily. Patients who are intolerant or allergic to aspirin may be given ticlopidine 250mg bid or clopidogrel 300mg stat followed by 75mg daily. Both these agents have not been tested in the setting of AMI.



4.4.3.3 Beta-blockers

Grade A

Current recommendation are to use B-blockers orally in all patients without specific contraindications to B-blockers.^{39,40,41} This should be initiated on the first or second day after presentation. Intravenous B-blockers are particularly useful in patients who are hypertensive and tachycardic.⁴² If intravenous B-blockers are not used, then oral B-blockers should be initiated by the first or second day after presentation. In the absence of contraindications, all patients should be on B-blockers at the time of hospital discharge and should be continued for at least 2 years. In patients with residual LV dysfunction and those who are at risk of residual ischaemia, B-blockers should be continued indefinitely.

Contraindications

1. Bradycardia <60/min
2. Systolic BP <100 mmHg
3. Pulmonary congestion with crepitations beyond the bases
4. Signs of peripheral hypoperfusion
5. Atrio-ventricular (AV) block
6. Severe chronic obstructive airway disease (COAD) or asthma
7. Severe peripheral vascular disease

Table 2: Recommended dosages of B-blockers in AMI

Type	Route of administration	Usual dose
Metoprolol	Intravenous	5-15mg*
Metoprolol	Oral	25-100mg bd
Atenolol	Oral	25-100mg daily
Propranolol	Oral	20-80mg tds
Acebutolol	Oral	200mg bd

* It should be given in three 5mg doses at 2-5 minutes intervals. If the patient is stable 15 minutes after the last IV dose, oral metoprolol 50mg may be commenced.

4.4.3.4 Angiotensin Converting Enzyme Inhibitors (ACEI)

Grade A

Early use of ACEI following AMI has been shown to improve survival. In the absence of contraindications, its early use (<24 hours) is associated with a better outcome.⁴³



ACEI should be started when the blood pressure is stable and remains above 100 mmHg. The benefits of ACEI are greatest in patients with:

- heart failure⁴⁴
- anterior infarcts⁴⁵
- left ventricular (LV) dysfunction (LV ejection fraction less than 40% on echocardiography)^{46,47}

The above patients should be discharged on ACEI unless contraindicated. Ideally all patients should have the LV function reassessed at 6 weeks. ACEI should be continued indefinitely in patients who have clinical or objective evidence of LV dysfunction. In patients with normal LV function, ACEI may be stopped. However, data from one recent trial seems to suggest that ACEI may be beneficial if continued long-term even in patients with normal LV function.⁴⁸

Contraindications

1. Systolic BP <100 mmHg
2. Established contraindications to ACEI e.g. bilateral renal artery stenosis, worsening renal function with ACEI.

Table 3 : Recommended dosages of ACEI in AMI

Type	Initiation dose	Target dose
Captopril	6.25mg tds	25-50mg tds
Ramipril	2.5mg bd	5mg bd
Enalapril	2.5-5mg daily	10mg daily
Lisinopril	5mg daily	10mg daily
Perindopril	2mg daily	4mg daily

In the presence of persistent LV dysfunction, the target doses should be higher (refer to Clinical Practice Guidelines 2000 on Heart Failure Table V).

4.4.3.5 Nitrates

Grade A

The routine use of nitrates has not been shown to have a survival benefit.^{49,50}

Nitrates can be considered in patients with:

- continuing chest pain and/or ischaemia
- heart failure
- 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, heart failure and large transmural AMI.

Contraindications

1. Hypotension (systolic BP < 90 mmHg)
2. Bradycardia < 50/min
3. RV infarction
4. History of sildenafil ingestion in the preceding 24 hours.

Table 4 : Nitrate compounds for AMI therapy

Compound	Route	Dosage	Onset time
Nitroglycerine, Glyceryl Trinitrate	Intravenous	5-200µg/min*	1 min
	Sublingual	0.3-0.6mg, can repeat up to 5 times after 5 mins	2 mins
	Transdermal Patch	0.2-0.8mg over 12 hrs on, then 12 hrs off	1-2 hrs
Isosorbide dinitrate	Intravenous	1.25- 5.0mg/hr	1 min
	Sublingual	2.5-10mg	3-4 mins
Isosorbide mononitrate	Oral	20-30mg, 2-3 times daily, up to 120mg in divided doses	30-60 mins

* 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.



4.4.3.6 Calcium Channel Blockers

Grade A

There is no data to support the routine use of calcium channel blockers.⁵¹ However, they may be used as adjunctive therapy in patients with on-going ischaemia despite B-blockers and nitrates. Calcium channel blockers should be avoided in patients with LV dysfunction, pulmonary congestion, bradycardia and AV block.

4.4.3.7 Antithrombotics

Grade A

Heparin is indicated in patients:

- who received tPA (given upon completion and continued for 48 hours)
- with post infarct angina
- with atrial fibrillation
- with mural thrombus

Heparin may be given as unfractionated heparin or as low molecular weight heparin.⁵² Unfractionated heparin is administered as a bolus of 5000 units followed by 1000 units/hour adjusting the dose to maintain the aPTT of 1.5 to 2 times control.

4.4.3.8 Glycoprotein IIb/IIIa (GpIIb/IIIa) Receptor Inhibitors

These agents are being evaluated in ongoing clinical trials as adjunctive therapy to thrombolytic agents.⁵³

Grade A

In the setting of primary PTCA, there is strong evidence to support the use of GpIIb/IIIa receptor inhibitors in improving outcomes.⁵⁴

4.4.3.9 Others - Magnesium, Lignocaine

Grade A

Intravenous magnesium⁵⁰ and lignocaine⁵⁵ are not recommended for routine use in patients with AMI.



4.5 Complications of AMI

These include:

- arrhythmias
- left ventricular dysfunction
- hypotension
- others e.g. pericarditis

4.5.1 Arrhythmias

These include:

4.5.1.1 Tachyarrhythmias

Ventricular Fibrillation (VF).

- De-fibrillate 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*).

Ventricular Tachycardia (VT).

- Management strategies will depend on the patient's haemodynamic status (*refer to algorithm 2*).

Ventricular Premature Contractions (VPC).

- These are often benign and do not require treatment. Correct underlying ischaemia, hypoxia and electrolyte disturbances.

Accelerated Idioventricular Rhythm (AIVR).

- These do not require treatment.

Atrial fibrillation (AF).

- This is more commonly seen in the elderly and is associated with large and atrial infarcts. It denotes a poorer prognosis⁵⁶ and carries an increased risk of thromboembolism (*refer to algorithm 3*).

Others - Narrow complex tachycardias.

- These are uncommon in AMI.



4.5.1.2 Bradyarrhythmias

These are:

Sinus bradycardia.

This does not require treatment unless associated with symptoms and/or hypotension.

AV block.

First degree and type 1 second degree do not need treatment. Patients with type 2 second degree and complete AV block may not require treatment if haemodynamically stable. If unstable, they will usually respond to atropine or may require pacing. In anterior infarcts, these arrhythmias carry a worse prognosis and may require urgent pacing (*refer to algorithm 4*).

Asystole/Pulseless Electrical Activity

(*refer to algorithm 5*).

Temporary pacing may be achieved via the transcutaneous or transvenous routes. If thrombolytic therapy or anticoagulants have been administered, the subclavian route should be avoided for temporary transvenous pacing. Preferred sites will be femoral or brachial routes.

4.5.2 Left Ventricular Dysfunction

The clinical spectrum of left ventricular dysfunction varies from asymptomatic to cardiogenic shock. Left ventricular function can be assessed rapidly using an echocardiogram. Prognosis is related to the extent of LV dysfunction. A useful clinical classification is the Killip's Classification:

Clinical and Haemodynamic Subsets in Acute Myocardial Infarction

KILLIP CLASS	CLINICAL FEATURES	APPROXIMATE PROPORTION OF PATIENTS WITH AMI (%)	HOSPITAL MORTALITY (%)
I	No signs of LV failure	40-50	>6
II	S3 gallop, bibasal crackles	30-40	>17
III	Acute pulmonary oedema	10-15	38
IV	Cardiogenic shock	5-10	81

4.5.2.1 Heart Failure

For the management of patients presenting with acute cardiogenic pulmonary oedema, (see flow chart 1, page 39 - adapted from Clinical Practice Guidelines on Heart Failure 2000 page 10-11).

4.5.2.2 Cardiogenic Shock

Cardiogenic shock is defined as a systolic BP of < 90 mmHg associated with signs of tissue hypoperfusion such as cool, clammy skin, low urine output and mental obtundation.

In most instances this is due to extensive myocardial damage. It is important to look for mechanical causes such as acute ventricular septal defect, acute mitral regurgitation and free wall rupture. Occasionally, RV infarction may also lead to cardiogenic shock.

Patients with cardiogenic shock have a very high mortality rate and aggressive management is required. This involves intensive medical management with inotropes and intra-aortic balloon counter pulsation. It is important to establish reperfusion of the infarct related artery by either thrombolytic therapy or primary PTCA.

If the cardiogenic shock is due to a mechanical defect, then urgent surgical repair is the treatment of choice. Pre-operative coronary angiography and concomitant coronary artery bypass graft (CABG) surgery in these patients remains an issue of debate. The decision must be individualized according to the patient's condition and the imperatives of the surgeon.



4.5.2.3 Hypotension

Hypotension may be due to:

- extensive myocardial damage
- mechanical defects
- RV infarction
- arrhythmias
- drugs
- hypovolaemia

Treatment measures should be targeted to the underlying etiology. In patients presenting with acute pulmonary oedema, excessive diuresis may result in severe intravascular volume depletion and resultant hypotension. It is thus important to keep a stringent watch on intake and output charts. Clinical assessment of hydration is also helpful in these cases.

In suspected hypovolaemia or RV infarct, judicious fluid replacement with empiric IV volume challenge of 250-500ml of normal saline may be administered. Care must be taken to avoid fluid overload. If there is no improvement or there are signs of fluid retention (basal lung crepitations), therapy should be guided by invasive monitoring. Additional supportive measures such as inotropes (dopamine) and intra-aortic balloon pump may also be required.

4.5.2.4 Right Ventricular Infarct (RVI)

RVI represents a spectrum of clinical presentations, ranging from asymptomatic to cardiogenic shock.⁵⁷ Haemodynamically significant RVI complicates approximately 5-10% of all AMI. When an inferior AMI occurs with an RVI, it is associated with a significantly higher mortality.

Though RVI is clinically recognized almost exclusively in patients with inferior AMI, it can also occur in patients with extensive anterior AMI.

Clinical Diagnosis

The presence of RVI should be sought in all patients with acute inferior AMI. Elevated jugular venous pressure in the setting of inferior AMI suggests RVI. The clinical triad of hypotension, clear lung fields and elevated jugular venous pressure, though more specific, is not very sensitive.

ST elevation in the right precordial leads (V4R) is the most specific⁵⁸ finding in diagnosing RVI. However, this ECG finding may be transient, often resolving within 8-10 hours.



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.

In patients with RVI and hypotension, volume loading with normal saline (up to 1-2L) will improve the cardiac output in the majority of cases. If the blood pressure still remains low, inotropic support may be necessary. 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 intra-aortic balloon pump. Should pacing be required for symptomatic bradyarrhythmias, ideally dual chamber pacing is recommended to maintain AV synchrony.

4.5.3 Others

4.5.3.1 Chest pain post AMI

Chest pain post AMI may be due to reinfarction, ischaemia or pericarditis.

4.5.3.2 Reinfarction

Reinfarction occurs in about 3-4% of patients who have undergone thrombolytic therapy and received aspirin.⁵⁹ Reinfarction may be diagnosed by the recurrence of ischaemic type chest pain, recurrence of ST segment elevation of at least 0.1mV in at least 2 contiguous leads and re-elevation of cardiac markers. Death, severe heart failure and arrhythmias are more common in these patients. They should be considered for rescue PTCA or if this is not available, additional thrombolysis.

4.5.3.3 Post-infarct angina

Early recurrent angina, especially after successful reperfusion may occur in up to 20% of patients.⁶⁰ The ECG in these patients may show ST segment changes or pseudo-normalisation of inverted T-waves. The cardiac markers may or may not be re-elevated. These patients should be sent for early coronary angiography with view to revascularization.



4.5.3.4 Pericarditis

Pericarditis secondary to transmural AMI may produce pain as early as the first day and as late as 6 weeks after AMI.⁶¹ 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 AMI. This is immunologically mediated.⁶² It can be treated with aspirin 600mg 3-4 times a day or non-steroidal anti-inflammatory drugs (NSAIDs).

4.5.3.5 LV Thrombus and Arterial Embolism

LV mural thrombus has been identified in about 20-40% of patients with AMI. The majority of these occur following an anterior MI. Warfarin therapy for 3-6 months is advocated in these patients.⁶³

Grade A

4.5.3.6 Deep Venous Thrombosis (DVT)

The following patients are prone to DVT:

- Obese
- Diabetes
- Bed-ridden (more than 3 days)
- Severe heart failure (particularly cardiogenic shock)
- Age > 70 years
- Previous history of DVT and pulmonary embolism

Grade A

They should be considered for prophylactic anticoagulation therapy (subcutaneous heparin 5000 units bd or tds or LMWH).



4.6 Coronary Artery Bypass Graft (CABG) Surgery

4.6.1 Urgent/Emergent CABG:

Urgent/emergent CABG should be considered in the following situations:

- at the time of surgical repair of post-infarction ventricular septal defect (VSD) or mitral valve insufficiency (see section 4.5.2.2).
- patients with failed reperfusion whose coronary anatomy and clinical profile are suitable for surgery should be considered for urgent/emergent CABG if there is:
 - persistent pain or ischaemia.
 - haemodynamic instability.

These patients should be treated aggressively with intra-aortic balloon pump support. In general, surgery in these group of patients have a very high in-hospital mortality rate.

5. RISK STRATIFICATION POST-AMI

Risk stratification serves to prognosticate and identify appropriate treatment strategies. Risk stratification starts from admission and is a continuing process.

Poor prognostic indicators include:

- older person (> 65 years).
- female gender.
- previous AMI.
- anterior AMI.
- inferior AMI with RV involvement.
- diabetes mellitus.
- ECG changes in multiple leads.
- persistent or recurrent ischaemia as manifested by post-infarction angina or ST segment depression at rest.
- hypotension.
- heart failure.
- atrial fibrillation and late (after 48 hours) ventricular arrhythmias.



The above high risk patients should be considered for early coronary angiography. All other patients should be risk stratified early. This helps to identify patients who are likely to reinfarct or develop other complications such as heart failure. It also helps in the rehabilitation of patients. Low risk patients may be allowed to return to their former activities early. Risk stratification may be done by assessing:

- left ventricular function - either clinically, by chest X-ray, echocardiogram or radionuclide studies.
- residual myocardial ischaemia - either clinically (recurrent angina) or by stress testing in asymptomatic patients. Stress test may be done from 5 days post-infarct (sub-maximal stress test with a target heart rate of 70% of maximum predicted heart rate) to 3 weeks post-discharge (maximal with a target heart rate of 90% of maximum predicted heart rate or symptom limited). If the pre-discharge stress test is negative, the patient should be subjected to a maximal or symptom limited stress test in 3-6 weeks after discharge. For those who cannot exercise, the alternatives would be dobutamine stress echocardiogram or radionuclide perfusion studies.

The presence of angina, an abnormal stress test or late ventricular arrhythmias necessitates coronary angiography with a view to revascularization.

In patients with poor LV function, myocardial viability studies (dobutamine stress echocardiogram or radionuclide perfusion studies) would help to differentiate scarred from viable ischaemic myocardium. The latter patients would benefit from revascularization.

Patients with palpitations, near faints and syncope require comprehensive evaluation. This includes:

- serum electrolytes
- resting ECG
- 24 hour ambulatory ECG recording
- evaluation of LV function
- assessment for reversible myocardial ischaemia
- coronary angiography
- signal average ECG
- heart rate variability

In these patients, reversible causes such as electrolyte disturbances and ischaemia should be corrected. Patients with LV dysfunction should be treated appropriately with ACEI and B-blockers.

Patients with LV dysfunction and resuscitated sudden cardiac death should be considered for implantable cardioverter defibrillator (ICD).^{64,65} The above patients and those with sustained and non-sustained VT and syncope should be referred for electrophysiological assessment.



6. DURATION OF HOSPITALIZATION

The duration of hospital stay following an uncomplicated AMI can be limited to 3-5 days.⁶⁶ However, the exact duration needs to be individualized. Elderly patients may require a longer hospital stay.

7. SECONDARY PREVENTION

Secondary prevention includes :

- Lifestyle factors:

Cessation of smoking⁶⁷

Grade A

Patients who quit smoking can reduce the rate of deaths and infarction over the first year after smoking cessation. After three years the risk matches that of the risk of CAD patients who have never smoked.

Diet and exercise⁶⁸

Grade C

Encourage a minimum of 30-60 minutes of moderate to intensive activity, 3-4 times a week.

Aspirin

Grade A

Aspirin should be prescribed at 75-300mg daily indefinitely unless contraindicated.^{69,70}

Grade C

In patients who cannot tolerate aspirin, ticlopidine or clopidogrel is an acceptable alternative.

B-blockers

Grade A

B-blockers should be prescribed to all patients provided that they are haemodynamically stable. Their value in patients successfully reperfused who have little or no regional wall motion abnormality remains unclear. In this group, B-blockers can be tapered after 6 months. B-blockers should be continued indefinitely in those with residual dysfunction and who are at risk for residual ischaemia.

ACEI

Grade A

ACEI should be continued indefinitely in patients with LV dysfunction (EF<40%).

Grade A

In patients who cannot tolerate ACEI, it may not be unreasonable to consider usage of an angiotensin receptor blocker.



Lipid-lowering therapy

Grade A

In view of the benefits seen in long term survival, prevention of reinfarction and coronary revascularization, it is important that patients with average^{71,72} or high cholesterol⁷³ levels be given statins. Patients with normal LDL cholesterol levels but low HDL cholesterol may benefit from fibrate therapy.⁷⁴

Calcium channel blockers and nitrates

Grade A

These agents should be prescribed only for symptomatic relief of ischaemia. The former should be avoided in those with heart failure or LV dysfunction.

Anticoagulation

Grade A

Long term therapy should be considered for patients with persistent atrial fibrillation. It should be given to patients with LV thrombus for 3-6 months.

Hormone replacement therapy (HRT)

Grade A

HRT should not be initiated for secondary prevention.⁷⁵ If the patient is already on HRT, she should be continued on it.

Others

Grade A

Vitamin E is not beneficial.⁴⁸

Grade A

Garlic, lecithin, vitamin A and C are not beneficial.

8. SPECIAL GROUPS

8.1 AMI in the Elderly

Following an AMI, the elderly have a much higher in-hospital as well as one year mortality. Complications such as cardiac rupture and reinfarction are also more common. These occur despite lower total creatinine kinase levels.

Atypical presentations or silent AMI occur in about 40% of elderly patients.⁷⁶ Atypical presentations include dyspnoea (30-50% of patients), syncope, palpitations, acute confusion or acute stroke. Acute pulmonary oedema and arterial embolism are also frequent presenting symptoms.

The diagnosis of AMI in the elderly is difficult. The ECG often shows ST segment depression rather than ST segment elevation. The presence of baseline abnormalities in the resting ECG e.g. LV hypertrophy and conduction abnormalities may mask the typical changes of an AMI. The serum cardiac markers tend to be minimally elevated, often out of proportion to the haemodynamic changes observed.



Management

- Thrombolytic Therapy

Grade A

There is an increased risk of cerebral bleeding in the elderly with the use of thrombolytics particularly with tPA.³¹ Despite this, thrombolytic therapy has been shown to substantially reduce mortality in patients above the age of 65 years. The absolute benefit of thrombolytic therapy in this age group is almost double that of younger patients.²⁷

Thrombolytic therapy is often underused. This is partly due to atypical presentations, delay in seeking medical attention, atypical ECG findings and the presence of co-morbid illnesses such as hypertension or previous stroke.

- Primary PTCA

Grade A

Current evidence indicates that primary PTCA may be the preferred reperfusion strategy in view of the higher risk of haemorrhagic stroke with the use of thrombolytic agents.^{17,34,35}

- Adjunctive therapy

Aspirin

Grade A

This has been shown to be effective in the elderly. There is however a higher incidence of side effects with aspirin use.⁷⁷

B-blocker therapy

Grade A

Elderly patients derive the same benefit as younger patients with B-blockade post AMI.^{78,79}

ACEI

Grade A

The survival benefit is greater in the elderly as compared to younger patients.⁴⁶

Risk Stratification:

This has to be individualized taking into consideration the physiological age rather than the chronological age of the patient. The presence of on-going ischaemia, symptomatic malignant arrhythmias and a depressed LV function are bad prognostic indicators and would generally necessitate a more aggressive approach. Both PTCA 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 AMI in Diabetics

Diabetic patients have a higher in-hospital mortality (about 1.5 to 2 times) than non-diabetics following an AMI.⁸⁰ The prognosis is worse in women. There is a higher frequency of atypical and silent presentations in these patients.

Management

Diabetic patients should be treated in a similar manner as non-diabetics.

Insulin Glucose Potassium Infusion

Grade A

Data from one trial showed that metabolic therapy with infusion of insulin, glucose and potassium reduced mortality by 11% in 3.5 years and by 15% in those who did not have prior insulin therapy.⁸¹ There are several on-going trials addressing the dosages, timing and duration of therapy.

9. CARDIAC REHABILITATION IN AMI

Cardiac rehabilitation is aimed at optimizing the physical, psychological and social well-being of patients following the acute event. This involves an inpatient and an outpatient phase.

1. Inpatient phase

The objectives are :

- to facilitate adjustment to the coronary care unit.
- to reduce anxiety and psychological disturbances to the patient and his family.
- early ambulation.
- to advocate healthy lifestyle and modification of risk factors.

2. Outpatient phase

The objectives are:

- to gradually return patients to prior level of activities.
- to maintain a healthy lifestyle and risk factors modification.
- to ensure compliance to medications.
- to educate patient and family on CHD.



Patients who remain asymptomatic after an uncomplicated AMI and have a negative pre-discharge stress test can return to prior activities safely within 2-4 weeks.^{82,83} Patients with symptoms and an abnormal stress test require further evaluation (see section 5).

Stable patients should be encouraged to exercise daily. Minimum would be 30-60 minutes of moderate to intensive activity, 3-4 times per week (walking, cycling, swimming or other equivalent aerobic activities). They can resume driving after 1 week. Sexual activities may be resumed after 10 days.

Patients with complicated infarcts (cardiopulmonary resuscitation, hypotension, serious arrhythmias, high-degree block or congestive heart failure) should wait at least 2-3 weeks after resolution of these complications before resuming the above physical activities.

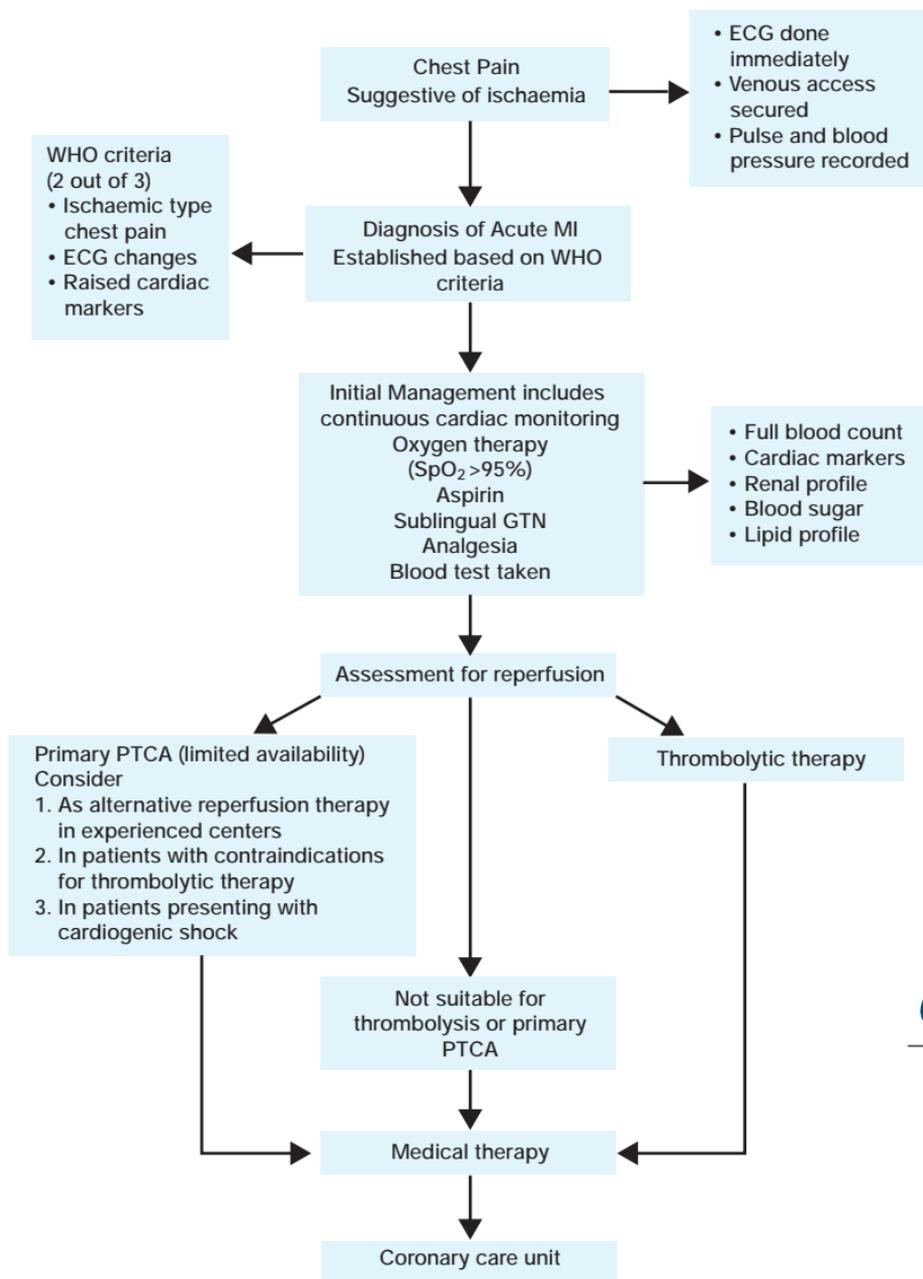
Commercial air travel should be postponed for at least 2 weeks after AMI due to the danger of pressurization and the lower oxygen tensions above 5000 feet.



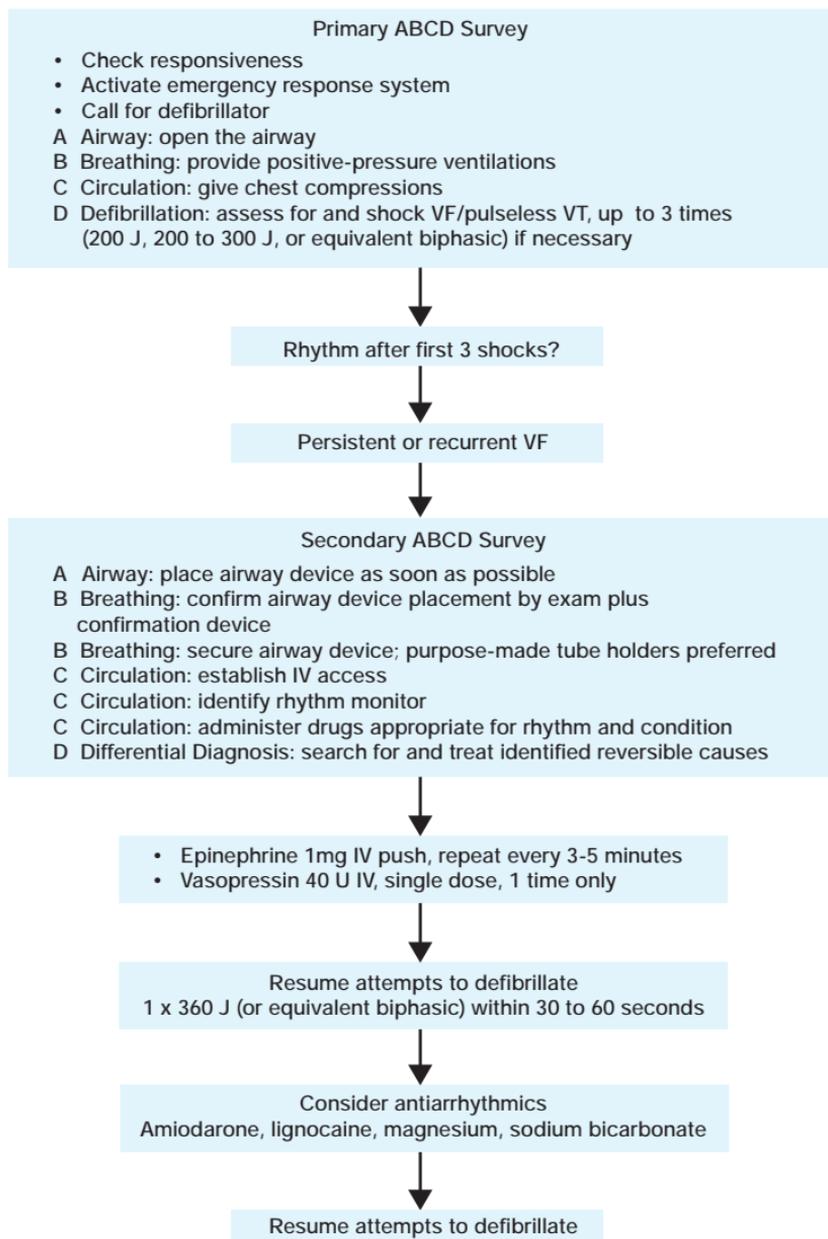
10. SUMMARY

- CHD is the most important cause of death in Malaysia.
- The public should be made aware of the early warning symptoms of an AMI and advised to seek early medical attention. Health personnel should be trained to deal with these patients appropriately.
- The diagnosis of AMI depends on the presence of 2 out of the following three criteria:
 - ischaemic type chest pain, ECG changes, rise and fall of cardiac markers.
- **TIME LOST IS MYOCARDIUM LOST**, thus early diagnosis and treatment is important.
- Early management of AMI involves pain relief, stabilization of haemodynamics and assessment for reperfusion.
- The occluded infarct-related artery should be opened as soon as possible either by thrombolytic therapy or direct PTCA.
- Adjunctive pharmacotherapy includes aspirin, B-blockers and ACEI.
- Complications of AMI include arrhythmias, LV dysfunction, hypotension, RV infarction and chest pain post AMI.
- Urgent/emergent CABG should be considered in patients with mechanical defects or failed reperfusion.
- High-risk patients should have early coronary angiography with view to revascularization. The others should be risk stratified according to the presence or absence of ischaemia and arrhythmias, and LV function.
- Secondary prevention is important and includes the use of aspirin, B-blockers, ACEI and statins.

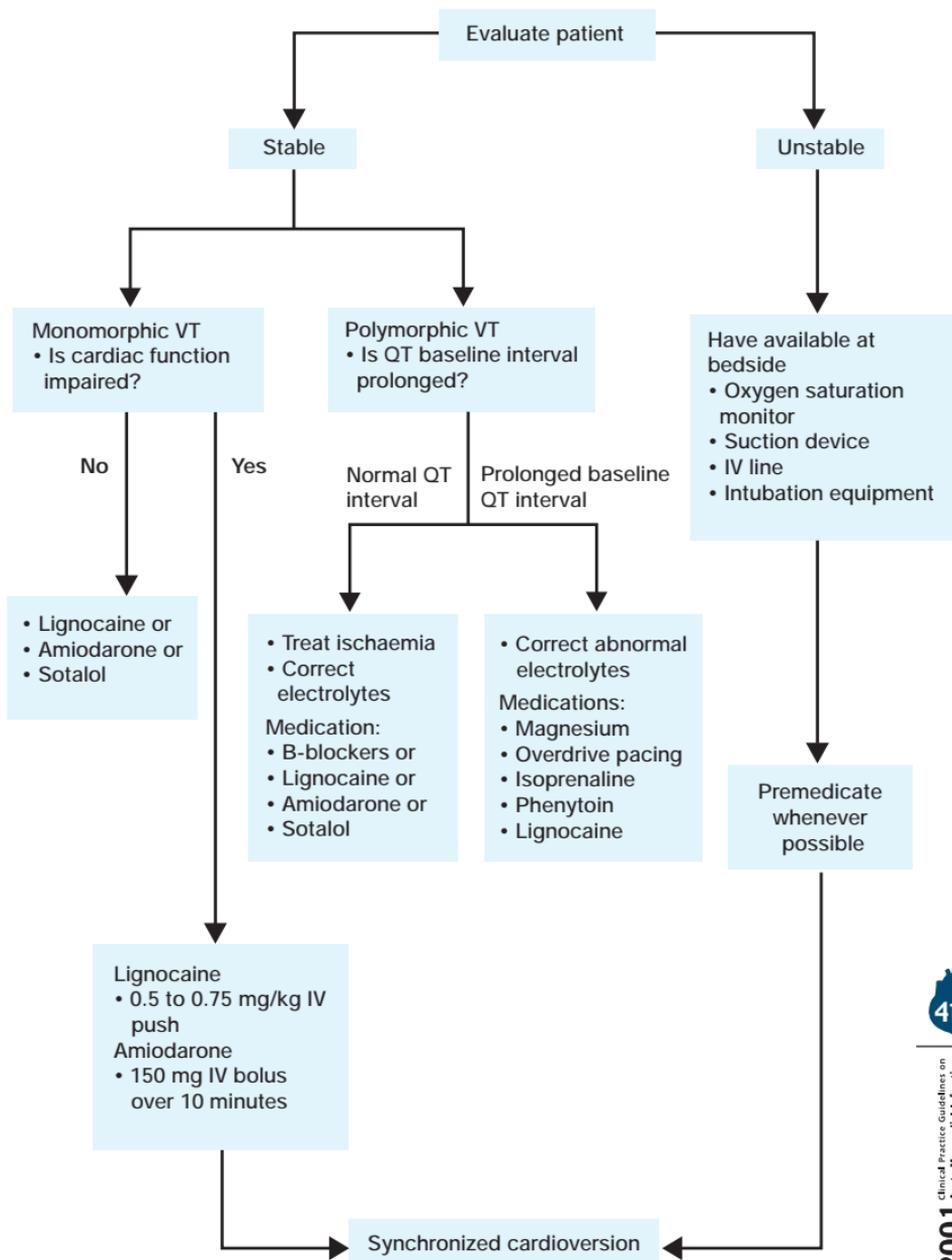
Flow Chart 1: Management of Acute Myocardial Infarction



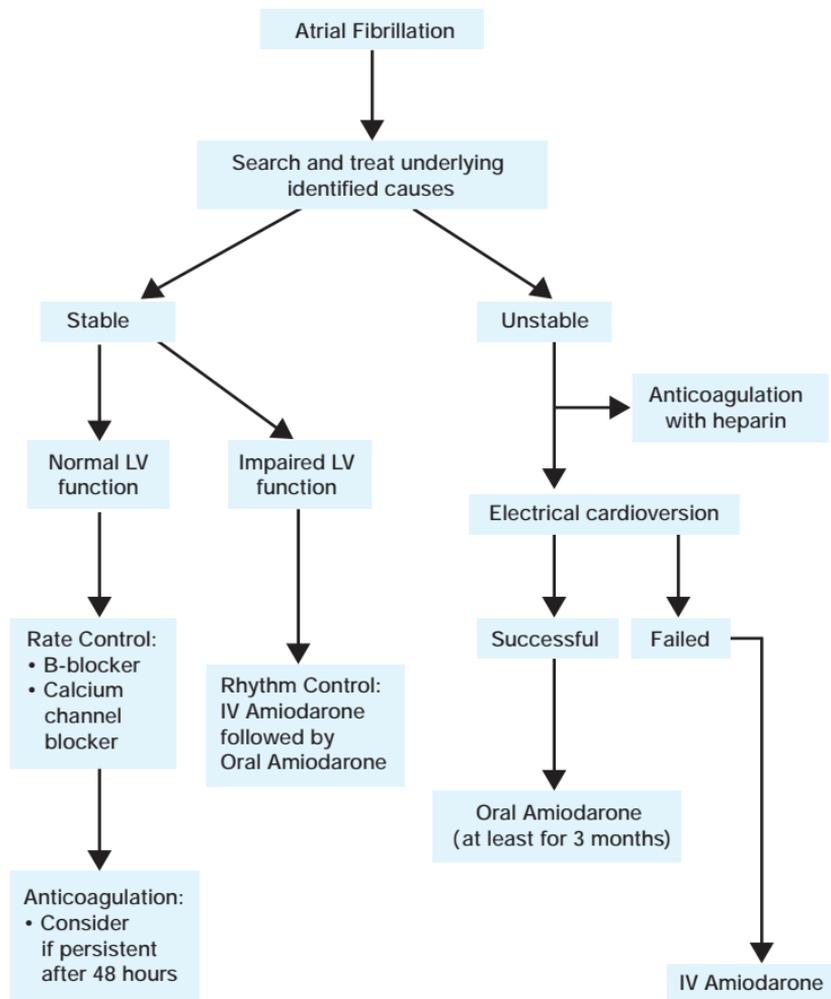
Algorithm 1: Management of Ventricular Fibrillation



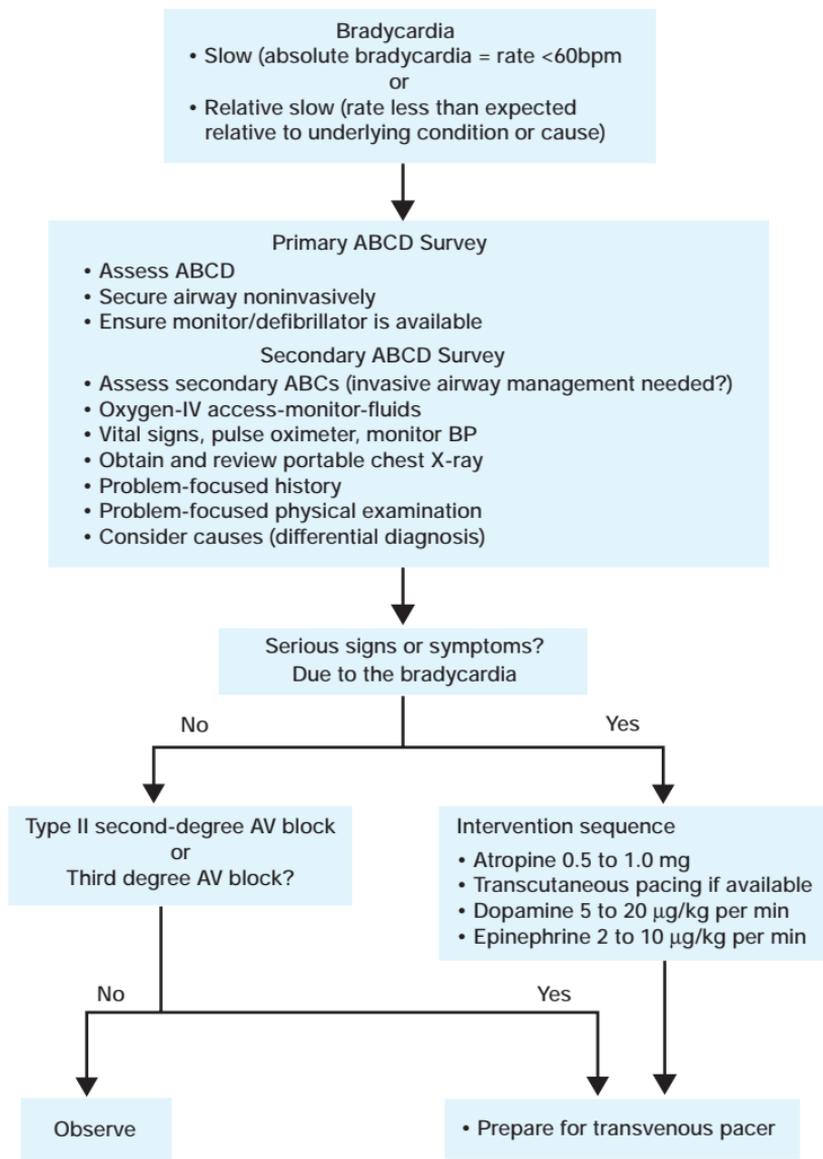
Algorithm 2: Management of Ventricular Tachycardia



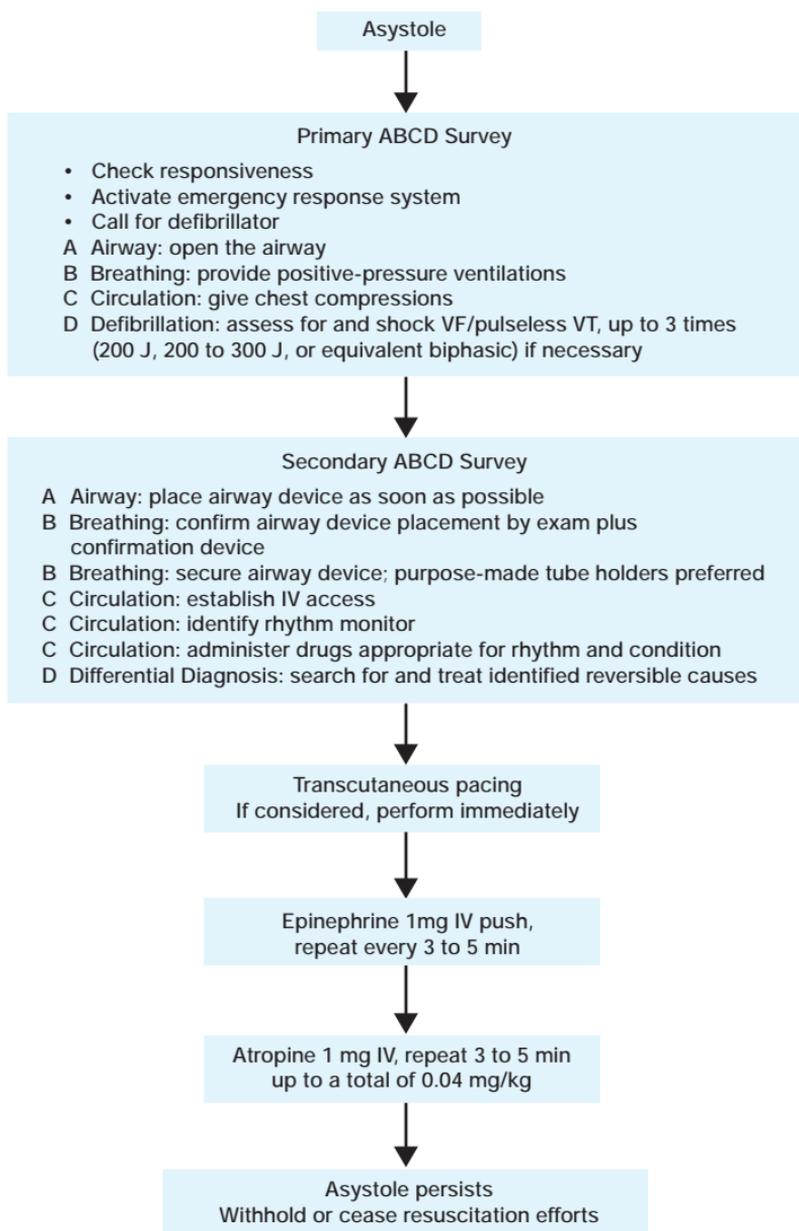
Algorithm 3: Management of Atrial Fibrillation



Algorithm 4: Management of Bradyarrhythmias



Algorithm 5: Management of Asystole





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