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**LECTURER**

**ICON**

## **ACUTE MYOCARDIAL INFARCTION**

### **1. INTRODUCTION:**

Coronary artery disease (CAD) is the leading cause of death in the United States. CAD is characterized by the accumulation of plaque within the layers of the coronary arteries. The plaques progressively enlarge, thicken, and calcify, causing a critical narrowing (> 70% occlusion) of the coronary artery lumen, resulting in a decrease in coronary blood flow and an inadequate supply of oxygen to the heart muscle.

### **2. DEFINITION:**

MI refers to a dynamic process by which one or more regions of the heart experience a severe and prolonged decrease in oxygen supply because of insufficient coronary blood flow; subsequently, necrosis or “death” to the myocardial tissue occurs. The onset of the MI process may be sudden or gradual, and the progression of the event to completion takes approximately 3 to 6 hours. MI is one manifestation of ACS.

### **3. CLASSIFICATION OF MI:**

- STEMI—whereby ST-segment elevations are seen on ECG. The area of necrosis may or may not occur through the entire wall of heart muscle.
- NSTEMI—no ST-segment elevations can be seen on ECG. ST depressions may be noted as well as positive cardiac markers, T-wave inversions, and clinical equivalents (chest pain). Area of necrosis may or may not occur through the entire myocardium.
- The region(s) of the heart muscle that becomes affected depends on which coronary artery(s) becomes obstructed

- Left ventricle is a common and dangerous location for an MI because it is the main pumping chamber of the heart.
- Right ventricular infarctions commonly occur with damage to the inferior and/or posterior wall of the left ventricle.
- The severity and location of the MI determines prognosis.

#### **4. RISK FACTORS:**

##### **4.1. Modifiable risk factors:**

Risk factors for atherosclerosis are generally risk factors for myocardial infarction:

- Diabetes (with or without insulin resistance) – the single most important risk factor for ischaemic heart disease (IHD)
- Tobacco smoking
- Air pollution
- Hypercholesterolemia (more accurately hyperlipoproteinemia, especially high low density lipoprotein and low high density lipoprotein)
- Low HDL
- high quantities of alcohol can increase the risk of heart attack High Triglycerides
- Hyperhomocysteinemia (high homocysteine, a toxic blood amino acid that is elevated when intakes of vitamins B<sub>2</sub>, B<sub>6</sub>, B<sub>12</sub> and folic acid are insufficient)
- Stress — Occupations with high stress index are known to have susceptibility for atherosclerosis
- Alcohol — Studies show that prolonged exposure to

Many of these risk factors are modifiable, so many heart attacks can be prevented by maintaining a healthier lifestyle. Physical activity, for example, is associated with a lower risk profile.

##### **4.2. Non-modifiable risk factors:**

Non-modifiable risk factors include age, sex, and family history of an early heart attack (before the age of 60), which is thought of as reflecting a genetic predisposition.

- Family history of ischaemic heart disease (IHD)
- Obesity (defined by a body mass index of more than 30 kg/m<sup>2</sup>, or alternatively by waist circumference or waist-hip ratio).
- Age — Men acquire an independent risk factor at age 45, Women acquire an independent risk factor at age 55; in addition individuals acquire another independent risk factor if they have a first-degree male relative (brother, father) who suffered a coronary vascular event at or before age 55. Another independent risk factor is acquired if one has a first-degree female relative (mother, sister) who suffered a coronary vascular event at age 65 or younger.
- Males are more at risk than females.

**Others:**

**Hyperlipidemia**

Elevated levels of total cholesterol, LDL, or triglycerides are associated with an increased risk of coronary atherosclerosis and MI. Levels of HDL less than 40 mg/dL also portend an increased risk. A full summary of the National Heart, Lung, and Blood Institute's cholesterol guidelines is available online.

**Diabetes Mellitus**

Patients with diabetes have a substantially greater risk of atherosclerotic vascular disease in the heart as well as in other vascular beds. Diabetes increases the risk of MI because it increases the rate of atherosclerotic progression and adversely affects the lipid profile. This accelerated form of atherosclerosis occurs regardless of whether a patient has insulin-dependent or non-insulin-dependent diabetes.

**Hypertension**

High blood pressure (BP) has consistently been associated with an increased risk of MI. This risk is associated with systolic and diastolic hypertension. The control of hypertension with appropriate medication has been shown to reduce the risk of MI

significantly. A full summary of the National Heart, Lung, and Blood Institute's JNC 7 guidelines published in 2003 is available online.

### **Tobacco Use**

Certain components of tobacco and tobacco combustion gases are known to damage blood vessel walls. The body's response to this type of injury elicits the formation of atherosclerosis and its progression, thereby increasing the risk of MI. A small study in a group of volunteers showed that smoking acutely increases platelet thrombus formation. This appears to target areas of high shear forces, such as stenotic vessels, independent of aspirin use. The American Lung Association maintains a website with updates on the public health initiative to reduce tobacco use and is a resource for smoking-cessation strategies for patients and health care providers.

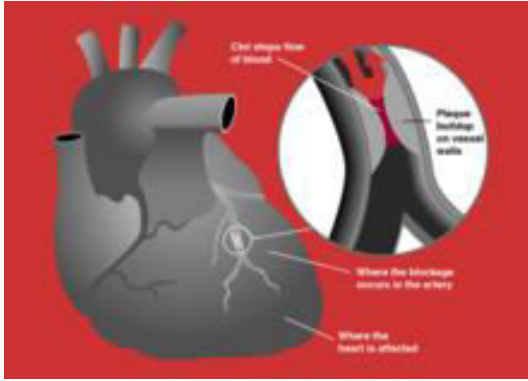
### **Male Gender**

The incidence of atherosclerotic vascular disease and MI is higher in men than women in all age groups. This gender difference in MI, however, narrows with increasing age.

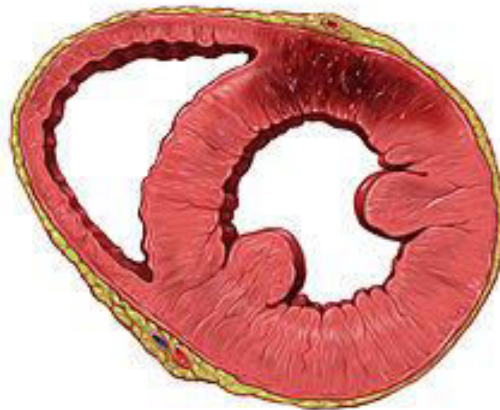
### **Family History**

A family history of premature coronary disease increases an individual's risk of atherosclerosis and MI. The cause of familial coronary events is multifactorial and includes other elements, such as genetic components and acquired general health practices (e.g. smoking, high-fat diet).

## **5. PATHOPHYSIOLOGY AND ETIOLOGY:**



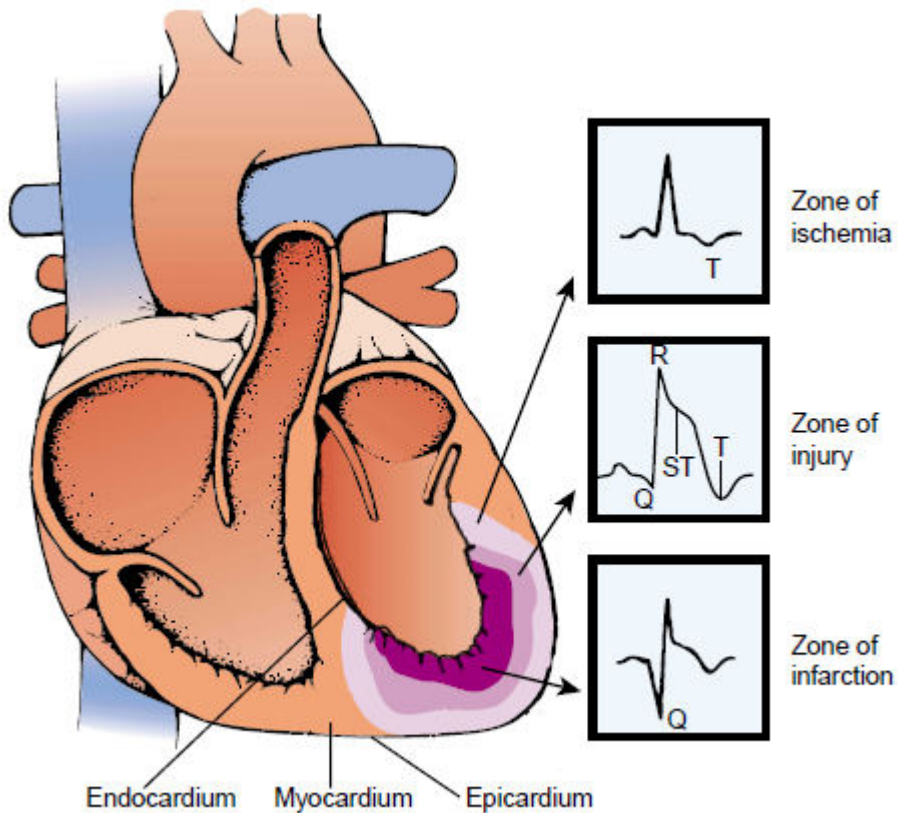
A myocardial infarction occurs when an atherosclerotic plaque slowly builds up in the inner lining of a coronary artery and then suddenly ruptures, causing catastrophic thrombus formation, totally occluding the artery and preventing blood flow downstream



Drawing of the heart showing anterior left ventricle wall infarction

- Acute coronary thrombosis (partial or total)—associated with 90% of MIs.
- Severe CAD (> 70% narrowing of the artery) precipitates thrombus formation.
- The first step in thrombus formation involves plaque rupture. Platelets adhere to the damaged area.
- Activation of the exposed platelets causes expression of glycoprotein IIb/IIIa receptors that bind fibrinogen.
- Further platelet aggregation and adhesion occurs, enlarging the thrombus and occluding the artery.
- Other etiologic factors include coronary artery spasm, coronary artery embolism, infectious diseases causing arterial inflammation, hypoxia, anemia, and severe exertion or

stress on the heart in the presence of significant CAD (ie, surgical procedures or shoveling snow).



**FIGURE 28-4** Effects of ischemia, injury, and infarction on ECG recording. Ischemia causes inversion of T wave because of altered repolarization. Cardiac muscle injury causes elevation of the ST segment and tall, symmetrical T waves. With Q-wave infarction, Q or QS waves develop because of the absence of depolarization current from the necrotic tissue and opposing currents from other parts of the heart.

- Different degrees of damage occur to the heart muscle
- **Zone of necrosis**—death to the heart muscle caused by extensive and complete oxygen deprivation; irreversible damage
- **Zone of injury**—region of the heart muscle surrounding the area of necrosis; inflamed and injured, but still viable if adequate oxygenation can be restored

- **Zone of ischemia**—region of the heart muscle surrounding the area of injury, which is ischemic and viable; not endangered unless extension of the infarction occurs

## **6. CLINICAL MANIFESTATIONS:**

### **Signs and Symptoms of an Acute Myocardial Infarction (MI) or Acute Coronary Syndrome (ACS)**

#### **Cardiovascular**

Chest pain or discomfort, palpitations, Heart sounds may include S3, S4, and new onset of a murmur. Increased jugular venous distention may be seen if the MI has caused heart failure. Blood pressure may be elevated because of sympathetic stimulation or decreased because of decreased contractility, impending cardiogenic shock, or medications. Pulse deficit may indicate atrial fibrillation. In addition to ST-segment and T-wave changes, ECG may show tachycardia, bradycardia, and dysrhythmias.

#### **Respiratory**

Shortness of breath, dyspnea, tachypnea, and crackles if MI has caused pulmonary congestion. Pulmonary edema may be present.

#### **Gastrointestinal**

Nausea and vomiting

#### **Genitourinary**

Decreased urinary output may indicate cardiogenic shock.

#### **Skin**

Cool, clammy, diaphoretic, and pale appearance due to sympathetic stimulation from loss of contractility may indicate cardiogenic shock. Dependent edema may also be present due to poor contractility.

#### **Neurologic**

Anxiety, restlessness, light-headedness may indicate increased sympathetic stimulation or a decrease in contractility and cerebral oxygenation.

The same symptoms may also herald cardiogenic shock.

Headache, visual disturbances, altered speech, altered motor function, and further changes in level of consciousness may indicate cerebral bleeding if patient is receiving thrombolytics.

## Psychological

Fear with feeling of impending doom, or patient may deny that anything is wrong.

- Chest pain
  - Severe, diffuse, steady substernal pain; may be described as crushing, squeezing, or dull
  - Not relieved by rest or sublingual vasodilator therapy, but requires opioids
  - May radiate to the arms (usually the left), shoulders, neck, back, and/or jaw
  - Continues for more than 15 minutes
  - May produce anxiety and fear, resulting in an increase in heart rate, BP, and respiratory rate
  - Some patients exhibit no complaints of pain.

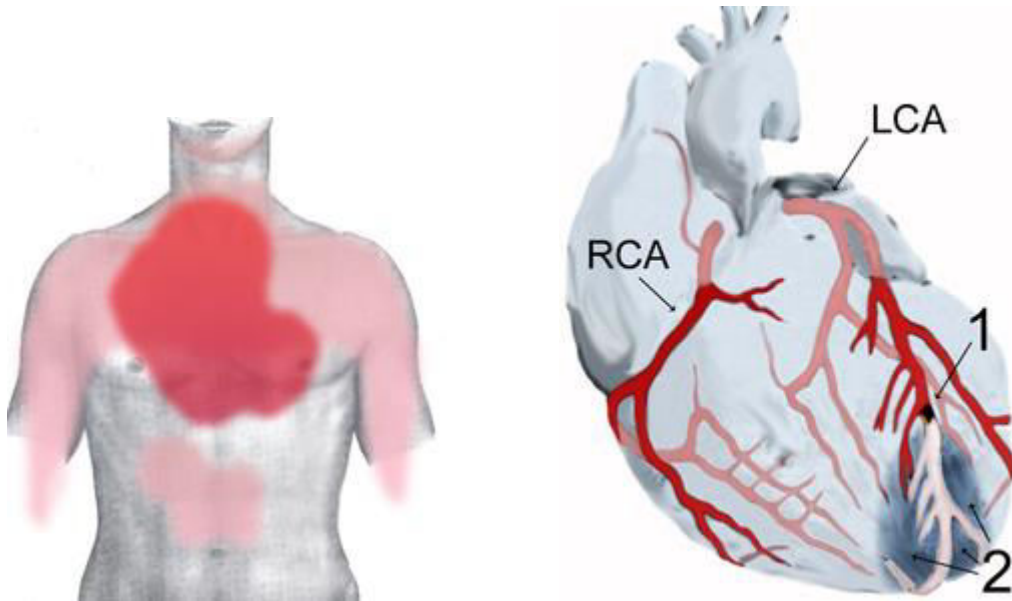


Diagram of a **myocardial infarction** (2) of the tip of the anterior wall of the heart (an apical infarct) after occlusion (1) of a branch of the left coronary artery (LCA), right coronary artery = RCA.





- Diaphoresis, cool clammy skin, facial pallor
- Hypertension or hypotension
- Bradycardia or tachycardia
- Premature ventricular and/or atrial beats
- Palpitations, severe anxiety, dyspnea
- Disorientation, confusion, restlessness
- Fainting, marked weakness
- Nausea, vomiting, hiccups

Atypical symptoms: epigastric or abdominal distress, dull aching or tingling sensations, shortness of breath, extreme fatigue

## **7. DIAGNOSTIC EVALUATION:**

### **PATIENT HISTORY**

The patient history has two parts: the description of the presenting symptom (eg, pain) and the history of previous illnesses and family health history, particularly of heart disease. Previous history should also include information about the patient's risk factors for heart disease.

### **LABORATORY TESTS**

Historically, laboratory tests used to diagnose an MI included **creatin kinase (CK)**, with evaluation of isoenzymes and lactic dehydrogenase (LDH) levels. Newer laboratory tests with faster results, resulting in earlier diagnosis, include myoglobin and

troponin analysis. These tests are based on the release of cellular contents into the circulation when myocardial cells die. An LDH test is now infrequently ordered because it is not useful in identifying cardiac events.

**Creatine Kinase and Its Isoenzymes.** There are three CK isoenzymes: CK-MM (skeletal muscle), CK-MB (heart muscle), and CK-BB (brain tissue). CK-MB is the cardiac-specific isoenzyme; CK-MB is found mainly in cardiac cells and therefore rises only when there has been damage to these cells. CK-MB assessed by mass assay is the most specific index for the diagnosis of acute MI. The level starts to increase within a few hours and peaks within 24 hours of an MI. If the area is reperfused (eg, due to thrombolytic therapy or PTCA), it peaks earlier.

**Myoglobin.** Myoglobin is a heme protein that helps to transport oxygen. Like CK-MB enzyme, myoglobin is found in cardiac and skeletal muscle. The myoglobin level starts to increase within 1 to 3 hours and peaks within 12 hours after the onset of symptoms. The test takes only a few minutes to run. An increase in myoglobin is not very specific in indicating an acute cardiac event; however, negative results are an excellent parameter for ruling out an acute MI. If the first myoglobin test results are negative, the test may be repeated 3 hours later. Another negative test result confirms that the patient did not have an MI.

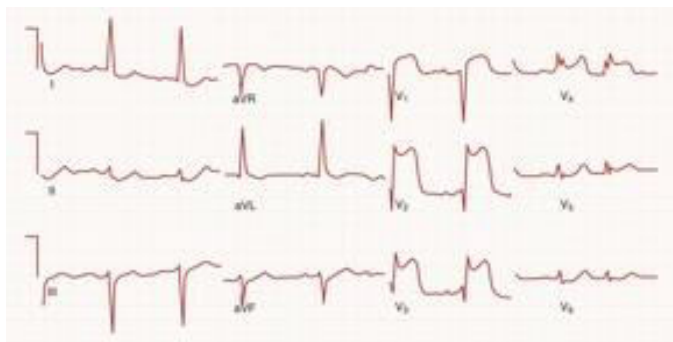
**Troponin.** **Troponin**, a protein found in the myocardium, regulates the myocardial contractile process. There are three isomers of troponin (C, I, and T). Because of the smaller size of this protein and the increased specificity of the troponins I and T for cardiac muscle, these tests are used more frequently to identify myocardial injury (unstable angina or acute MI). The increase in the level of troponin in the serum starts and peaks at approximately the same time as CK-MB. However, it remains elevated for a longer period, often up to 3 weeks, and it therefore cannot be used to identify subsequent extension or expansion of an MI.

**Table 28-5 • Serum Markers of Acute Myocardial Infarction**

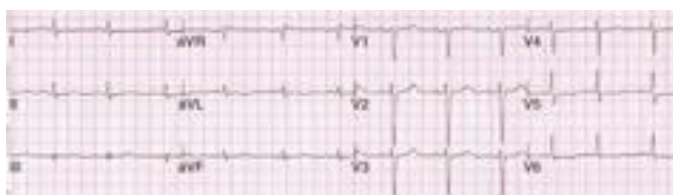
SERUM TEST	EARLIEST INCREASE (HR)	TEST RUNNING TIME (MIN)	PEAK (HR)	RETURN TO NORMAL
Total CK	3-6	30-60	24-36	3 days
CK-MB: isoenzyme	4-8	30-60	12-24	3-4 days
mass assay	2-3	30-60	10-18	3-4 days
Myoglobin	1-3	30-60	4-12	12 hr
Troponin T or I	3-4	30-60	4-24	1-3 wk

**ECG Changes**

- Generally occur within 2 to 12 hours, but may take 72 to 96 hours.
- Necrotic, injured, and ischemic tissue alters ventricular depolarization and repolarization.
  - ST-segment depression and T-wave inversion indicate a pattern of ischemia.
  - ST elevation indicates an injury pattern.
  - Q waves indicate tissue necrosis and are permanent. A pathologic Q wave is one that is greater than 3 mm in depth or greater than one-third the height of the R wave.



ST elevation MI



Non ST elevation MI

### **Echocardiogram**

The echocardiogram is used to evaluate ventricular function. It may be used to assist in diagnosing an MI, especially when the ECG is nondiagnostic. The echocardiogram can detect hypokinetic and akinetic wall motion and can determine the ejection fraction. Location of the infarction (anterior wall, anteroseptal) is determined by the leads in which the ischemic changes are seen.

### **Other Findings**

- Elevated CRP and lipoprotein (a) due to inflammation in the coronary arteries.
- Abnormal coagulation studies (prothrombin time [PT], partial thromboplastin time [PTT]).
- Elevated white blood cell (WBC) count and sedimentation rate due to the inflammatory process involved in heart muscle cell damage.
- Radionuclide imaging allows recognition of areas of decreased perfusion.
- PET determines the presence of reversible heart muscle injury and irreversible or necrotic tissue; extent to which the injured heart muscle has responded to treatment can also be determined.
- Cardiac muscle dysfunction noted on echocardiography or cardiac magnetic resonance imaging (MRI).

### **8. MANAGEMENT:**

Therapy is aimed at reversing ischemia to preserve cardiac muscle function, reduce the infarct size, and prevent death. Innovative modalities provide early restoration of coronary blood flow. The use of pharmacologic agents improves oxygen supply and demand, reduces and prevents dysrhythmias, and inhibits the progression of CAD.

The pharmacologic therapy for treatment of MI is standard

MONA—acronym that outlines the immediate pharmacologic interventions used to treat MI.

- Morphine's analgesic effects decrease the pain, relieve anxiety, and improve cardiac output. Morphine—given I.V. Used to treat chest pain. Endogenous catecholamine release during pain imposes an increase in the workload on the

heart, thus causing an increase in oxygen demand. by reducing preload and afterload.

- O (Oxygen)—given via nasal cannula or face mask. Increases oxygenation to ischemic heart muscle.
- N (Nitrates)—given sublingually, spray, or I.V. Vasodilator therapy reduces preload by decreasing blood return to the heart and decreasing oxygen demand.
- A (Aspirin)—immediate dosing by mouth is recommended to halt platelet aggregation.

#### Other Medications

- Thrombolytic agents, such as tissue plasma activator (Activase), streptokinase (Streptase), and reteplase (Retavase), reestablish blood flow in coronary vessels by dissolving thrombus.
  - No effect on the underlying stenosis that precipitated the thrombus to form.
  - Administered I.V. or I.C.
- Anti-arrhythmics, such as amiodarone, decrease the ventricular irritability that occurs after MI.
  - Given I.V. via bolus, then infusion over 24 hours.
- **Antiplatelet drug therapy** such as aspirin and/or clopidogrel should be continued to reduce the risk of plaque rupture and recurrent myocardial infarction. Aspirin is first-line, owing to its low cost and comparable efficacy, with clopidogrel reserved for patients intolerant of aspirin. The combination of clopidogrel and aspirin may further reduce risk of cardiovascular events, however the risk of hemorrhage is increased.

#### Antiplatelet Medications

Treatment Modality	Aspirin	Clopidogrel
Medical	75-162 mg/day indefinitely	Optional: 75 mg/day × 1

management		month
Bare Metal stent	162-325 mg/day × 1 month, then 75- 162 mg/day indefinitely	300 mg loading dose, * then 75 mg/day × 1 month
Sirolimus eluting stent (Cypher)	162-325 mg/day × 3 months, then 75- 162 mg/day indefinitely	300 mg loading dose, * then 75 mg/day × 1 year
Paclitaxel eluting stent (Taxus)	162-325 mg/day × 6 months, then 75- 162 mg/day indefinitely	300 mg loading dose, * then 75 mg/day × 1 year

- **Beta blocker therapy** such as metoprolol or carvedilol should be commenced. These have been particularly beneficial in high-risk patients such as those with left ventricular dysfunction and/or continuing cardiac ischaemia.  $\beta$ -Blockers decrease mortality and morbidity. They also improve symptoms of cardiac ischemia in NSTEMI.

### Beta Blocker Therapy

Agent	Dosing	Original Trial
Metoprolol	15 mg IV × 1 then 200 mg/day PO in divided doses	MIAMI <sup>19</sup>
Atenolol	5-10 mg IV × 1, then 100 mg/day PO	ISIS-1 <sup>20</sup>
Carvedilol	6.25 mg bid titrated to 25 mg BID	CAPRICORN <sup>21</sup>

- **ACE inhibitor therapy** should be commenced 24–48 hours post-MI in hemodynamically-stable patients, particularly in patients with a history of MI, diabetes mellitus, hypertension, anterior location of infarct (as assessed by ECG), and/or evidence of left ventricular dysfunction. ACE inhibitors reduce mortality, the development of heart failure, and decrease ventricular remodelling post-MI.

## ACE Inhibitors

Agent	Dosing (PO)	Original Trial
Captopril	6.25 mg tid titrated to 50 mg tid	SAVE: 3-16 days post-MI in asymptomatic patients with EF <40% <sup>22</sup>
Ramipril	1.25 mg bid titrated to 5 mg bid	AIRE: 3-10 days post-MI with symptoms of heart failure <sup>23</sup>
Captopril	6.25 mg bid titrated to 50 mg bid	ISIS-4: started within 24 hr of MI <sup>24</sup>
Lisinopril	5 mg/day titrated to 10 mg/day	GISSI-3: started within 24 hr of MI <sup>25</sup>

- **Statin therapy** has been shown to reduce mortality and morbidity post-MI. The effects of statins may be more than their LDL lowering effects. The general consensus is that statins have plaque stabilization and multiple other ("pleiotropic") effects that may prevent myocardial infarction in addition to their effects on blood lipids.
- The aldosterone antagonist agent eplerenone has been shown to further reduce risk of cardiovascular death post-MI in patients with heart failure and left ventricular dysfunction, when used in conjunction with standard therapies above. Spironolactone is another option that is sometimes preferable to eplerenone due to cost.

### Unfractionated Heparin Dosing

#### Loading Dose

- 60 U/kg IV bolus
- Max 5000 U if >65 kg or 4000 U if <65 kg

#### Maintenance Dose

- 12 U/kg/hr IV

- Max 1000 U/hr if >65 kg or 800 U/hr if <65 kg

**Titration Goal**

- PTT 50-70 sec

**Low-Molecular-Weight Heparin**

Generic name	t <sub>1/2</sub> (after dosing)	SC Dosing in ACS	FDA Approved Indications
Dalteparin	3-5 hr	120 U/kg SC bid	Prevention of ischemic complications in UA and NSTEMI
Enoxaparin	4.5 hr	100 U/kg (1 mg/kg) SC q12h	Prophylaxis of ischemic complications of UA and NSTEMI when administered with aspirin

**9. PERCUTANEOUS CORONARY INTERVENTIONS:**

- Mechanical opening of the coronary vessel can be performed during an evolving infarction.
- Percutaneous coronary interventions (PCIs), including percutaneous transluminal coronary angioplasty, coronary stenting, and atherectomy, can be used instead of, or as an adjunct to, thrombolytic therapy
- Should be performed within 30 minutes of initial diagnosis of MI.

**Surgical Revascularization**

- Emergency CABG surgery can be performed within 6 hours of evolving infarction.



- Benefits of this therapy include definitive treatment of the stenosis and less scar formation on the heart.

#### **10. COMPLICATIONS:**

- Dysrhythmias
- Sudden cardiac death due to ventricular arrhythmias
- Infarct expansion (thinning and dilation of the necrotic zone)
- Infarct extension (additional heart muscle necrosis occurring after 24 hours of acute infarction)
- Heart failure (with 20% to 35% left ventricle damage)
- Cardiogenic shock
- Reinfarction
- Ischemic cardiomyopathy
- Cardiac rupture
- Papillary muscle rupture
- Ventricular mural thrombus
- Thromboemboli
- Ventricular aneurysm
- Cardiac tamponade
- Pericarditis (2 to 3 days after MI)
- Dissection of coronary arteries during angioplasty
- Psychiatric problems—depression, personality changes

#### **11. NURSING ASSESSMENT:**

- Ask patient to describe anginal attacks.
- Obtain a baseline 12-lead ECG.
- Assess patient's and family's knowledge of disease.
- Identify patient's and family's level of anxiety and use of appropriate coping mechanisms.

- Gather information about the patient's cardiac risk factors. Use the patient's age, total cholesterol level, LDL and HDL levels, systolic BP, and smoking status to determine the patient's 10-year risk for development of CHD according to the Framingham Risk Scoring
- Evaluate patient's medical history for such conditions as diabetes, heart failure, previous myocardial infarction (MI), or obstructive lung disease that may influence choice of drug therapy.
- Identify factors that may contribute to noncompliance with prescribed drug therapy.
- Review renal and hepatic studies and complete blood count (CBC).
- Discuss with patient current activity levels. (Effectiveness of antianginal drug therapy is evaluated by patient's ability to attain higher activity levels.)
- Discuss patient's beliefs about modification of risk factors and willingness to change.

#### **Nursing Diagnoses:**

- Acute Pain related to an imbalance in oxygen supply and demand
- Decreased Cardiac Output related to reduced preload, afterload, contractility, and heart rate secondary to hemodynamic effects of drug therapy
- Anxiety related to chest pain, uncertain prognosis, and threatening environment

#### **Nursing Interventions:**

##### Relieving Pain

- Determine intensity of patient's angina.
  - Ask patient to compare the pain with other pain experienced in the past and, on a scale of 0 (no pain) to 10 (worst pain), rate current pain.
  - Observe for other signs and symptoms, including diaphoresis, shortness of breath, protective body posture, dusky facial color, and/or changes in level of consciousness (LOC).
- Position patient for comfort; Fowler's position promotes ventilation.

- Administer oxygen if prescribed.
- Obtain BP, apical heart rate, and respiratory rate.
- Obtain a 12-lead ECG as directed.
- Administer antianginal drug(s) as prescribed.
- Report findings to health care providers.
- Monitor for relief of pain, and note duration of anginal episode.
- Take vital signs every 5 to 10 minutes until angina pain subsides.
- Monitor for progression of stable angina to unstable angina: increase in frequency and intensity of pain, pain occurring at rest or at low levels of exertion, pain lasting longer than 5 minutes.
- Determine level of activity that precipitated anginal episode.
- Identify specific activities patient may engage in that are below the level at which anginal pain occurs.
- Reinforce the importance of notifying nursing staff when angina pain is experienced.

### **Maintaining Cardiac Output**

- Carefully monitor the patient's response to drug therapy.
  - Take BP and heart rate in a sitting and a lying position on initiation of long-term therapy (provides baseline data to evaluate for orthostatic hypotension that may occur with drug therapy).
  - Recheck vital signs as indicated by onset of action of drug and at time of drug's peak effect.
  - Note changes in BP of more than 10 mm Hg and changes in heart rate of more than 10 beats/minute.
  - Note patient complaints of headache (especially with use of nitrates) and dizziness (more common with ACE inhibitors).
    - Administer or teach self-administration of analgesics as directed for headache.

- Encourage supine position to relieve dizziness (usually associated with a decrease in BP; preload is enhanced by this mechanism, thereby increasing BP).
- Institute continuous ECG monitoring or obtain 12-lead ECG as directed. Interpret rhythm strip every 4 hours for patients on continuous monitoring (beta-adrenergic blockers and calcium channel blockers can cause significant bradycardia and varying degrees of heart block).
- Evaluate for development of heart failure (beta-adrenergic blockers and some calcium channel blockers decrease contractility, thus increasing the likelihood of heart failure).
  - Obtain daily weight and intake and output.
  - Auscultate lung fields for crackles.
  - Monitor for the presence of edema.
  - Monitor central venous pressure (CVP) if applicable.
  - Assess jugular vein distention.
- Monitor laboratory tests as indicated (cardiac markers).
- Be sure to remove previous nitrate patch or paste before applying new paste or patch (prevents hypotension) and to reapply on different body site. To decrease nitrate tolerance, transdermal nitroglycerin may be worn only in the daytime hours and taken off at night when physical exertion is decreased.
- Be alert to adverse reaction related to abrupt discontinuation of beta-adrenergic blocker and calcium channel blocker therapy. These drugs must be tapered to prevent a “rebound phenomenon”: tachycardia, increase in chest pain, hypertension.
- Discuss use of chromotherapeutic therapy with health care provider (tailoring of anti-anginal drug therapy to the timing of circadian events).
- Report adverse drug effects to health care provider.

### **Decreasing Anxiety**

- Assess patient for signs of hypoperfusion, auscultate heart and lung sounds, obtain a rhythm strip, and administer oxygen as prescribed. Notify the health care provider immediately.
- Document all assessment findings, health care provider notification and response, and interventions and response.
- Explain to patient and family reasons for hospitalization, diagnostic tests, and therapies administered.
- Encourage patient to verbalize fears and concerns about illness through frequent conversations—conveys to patient a willingness to listen.
- Administer medications to relieve patient's anxiety as directed. Sedatives and tranquilizers may be used to prevent attacks precipitated by aggravation, excitement, or tension.
- Explain to patient the importance of anxiety reduction to assist in control of angina. (Anxiety and fear put an increased stress on the heart, requiring the heart to use more oxygen.) Teach relaxation techniques.
- Discuss measures to be taken when an anginal episode occurs. (Preparing patient decreases anxiety and allows patient to accurately describe angina.)
  - Review the questions that will be asked during anginal episodes.
  - Review the interventions that will be employed to relieve anginal attacks.

### **Patient Education and Health Maintenance**

#### **Instruct Patient and Family about CAD**

- Review the chambers of the heart and the coronary artery system, using a diagram of the heart.
- Show patient a diagram of a clogged artery; explain how the blockage occurs; point out on the diagram the location of patient's lesions.
- Explain what angina is (a warning sign from the heart that there is not enough blood and oxygen because of the blocked artery or spasm).
- Review specific risk factors that affect CAD development and progression; highlight those risk factors that can be modified and controlled to reduce risk.

- Discuss the signs and symptoms of angina, precipitating factors, and treatment for attacks. Stress to patient the importance of treating angina symptoms at once.
- Distinguish for patient the different signs and symptoms associated with stable angina versus preinfarction angina.
- Give patient and family handouts to review and encourage questions for a later teaching session.

## **12. PREVENTION:**

According to the AHA/American College of Cardiology (ACC) Guidelines for Secondary Prevention for Patients with Coronary and Other Atherosclerotic Vascular Disease (2006):

- Cessation of smoking
- Control of high blood pressure (below 130/85 mm Hg in those with renal insufficiency or heart failure; below 130/80 mm Hg in those with diabetes; below 140/90 mm Hg in all others)
- 3. Diet low in saturated fat (< 10% of calories), cholesterol (<300 mg/day), trans-fatty acids, sodium (>6 g/day), alcohol (2 or fewer drinks/day in men, 1 or fewer in women)
- Low-dose aspirin daily for those at high risk
- Physical exercise (at least 30 minutes of moderate intensity exercise most days)
- Weight control (ideal body mass index 18.5 to 24.9 kg/m<sup>2</sup>); waist circumference less than 40 inches for men, less than 35 inches for women
- 7. Control of diabetes mellitus (fasting glucose <110 mg/dL and HbA<sub>1C</sub> < 7%)
- Control of blood lipids with low-density lipoprotein (LDL) goal less than 100.

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