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Textbook of
Cardiothoracic Nursing



B Venkatesan

JAYPEE

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CHAPTER
3

Coronary Artery Disease

INTRODUCTION

Coronary artery disease (CAD), or more specifically, coronary atherosclerotic heart disease, is the primary cause of death in the United States for both men and women. Most CAD results from the deposition of atheromatous plaque in the large and medium-sized arteries of the heart. A less frequent and usually idiopathic form of CAD is due to coronary spasm. Coronary artery atherosclerosis starts insidiously and is usually distributed irregularly in various blood vessels. Arteriosclerosis is hardening of the arteries. Atherosclerosis is a subset of arteriosclerosis, and is the formation of *atheromas* (fibrous fatty intimal plaques) in arterial walls.

Atherosclerotic blood vessels have reduced expansion with systole and abnormal wave propagation. This can reduce or obstruct blood flow to areas of the myocardium, sometimes with seeming abruptness, resulting in myocardial ischemia. Some plaque formations are unstable and may undergo spontaneous rupture. These formations are more closely associated with acute ischemic events. The four most common and serious complications of CAD are angina pectoris, unstable angina, myocardial infarction, and sudden cardiac death as the result of arrhythmias. Most evidences suggest that increased oxygen demand is the most frequent mechanism of provocation for angina pectoris. By contrast, the acute coronary syndromes of unstable angina and myocardial infarction are caused by disruption of plaque, platelet plugging, and coronary thrombosis. The peak incidence of clinical pathology for men is between the ages of 50 and 60 years, and for women, between the ages of 60 and 70 years.

ETIOLOGY

The exact pathogenesis of CAD is not clear, and no single theory adequately explains the atherosclerotic process. Two main explanations have been proposed: The lipid hypothesis and the chronic endothelial injury hypothesis. These explanations are probably interrelated and are certainly not mutually exclusive.

The lipid hypothesis speculates that elevation in lipid plasma levels promotes lipid penetration of arterial walls. In general, it is considered that this process is instigated by abnormal lipid metabolism or excessive dietary intake of cholesterol and saturated fats, particularly when coupled with a genetic predisposition. Low-density lipoproteins (LDLs) are the primary atherogenic lipid, whereas high-density lipoproteins (HDLs) have a protective effect and probably help mobilize LDLs.

When LDLs undergo oxidation in the body, they become harder to mobilize and locally cytotoxic. This is where the lipid

hypothesis and endothelial injury hypothesis potentially overlap. According to the latter theory, the initial step in the formation of atherosclerosis is a weakening of the arterial glycosaminoglycans (GAGs) layer. GAGs protect the internal lining of the artery (the endothelium) and promote its repair. The exposed endothelial cells of the artery are subject to free-radical damage, and damage to the endothelial lining makes those sites more permeable to plasma constituents, particularly lipoproteins. Several factors, besides oxidized LDLs, can damage endothelial cells and induce plaque formation. These factors are immunologic, inflammatory, viral, chemical, mechanical, and physical in nature. In response to cell injury, macrophages migrate from circulation into the cells that line the inside of the artery known as the subendothelial layer of the intima. Ultimately as this process continues, smooth muscle cells of the intima join connective tissue and intracellular and extracellular lipids to form fibrous plaque. Adhesion and aggregation of platelets and release of various growth factors further augment the fibrotic process, resulting in occlusion of the blood vessel lumen and potential for plaque disruption and thrombus formation.

RISK FACTORS

Non-modifiable Risk Factors

- Age
  - A dominant influence
  - Atherosclerosis begins in the young, but does not precipitate organ injury until later in life
- Gender
  - Men more prone than women, but by age 60–70 both have about equal frequency
- Family history
  - Familial cluster of risk factors—Genetic differences.

Modifiable Risk Factors (Potentially Controllable)

- Dyslipidemias; particularly high low density cholesterol (LDL-C) and low high density cholesterol (HDL-C). (ATP III classification of LDL, total and HDL cholesterol; LDL: optimal <100 mg/dL, near or above optimal 100–139 mg/dL, borderline high 140-159 mg/dL, high >160 mg/dL. Total cholesterol: Desirable <200 mg/dL, borderline high 200-239 mg/dL, high >240 mg/dL. HDL: Low <40 mg/dL.
- Hypertension (50 million in the US, 1/3 undiagnosed, 3/4 under treated). (Classification of HTN; Normal BP: systolic <120 mmHg and diastolic <80 mmHg, prehypertension: Systolic 120-139 or diastolic 80-89 mmHg, hypertension: systolic >140 or diastolic >90 mmHg.
- Diabetes mellitus (8% of US population). (Classification: Normal fasting glucose <110 mg/dL, impaired 110 ≤126 mg/dL, diabetes >126 mg/dL).
- Smoking (most important modifiable risk factor), CAD accounts for 35%–40% of all smoking related deaths.
- Family history of premature coronary artery disease (CAD); First degree male relatives <55 years or females <65 years.
- Obesity (18% of US population) and lack of exercise.
- Alcohol abuse.
- Others (20% of CAD occurs in individuals without any of the classical risk factors); homocysteinemia, high sensitivity C-reactive protein (hs-CRP), fibrinogen, lipoprotein a (Lpa), infection (*Chlamydia pneumoniae*).

Initial inflammation → endothelial dysfunction → monocyte recruitment/ differentiation to intima → growth factors stimulate smooth muscle proliferation → platelet activation/aggregation → atheroma with thin fibrous cap, no more protecting vessels

LDL cholesterol can deposit in tissues. Even worse, radicals may oxidize LDL cholesterol. Oxidized LDL is very toxic, and directly kills endothelial cells and activates macrophages.

Toxins such as nicotine and homocysteine have a similar effect. Hypertension can cause formation of the radicals that oxidize LDL.

PATHOPHYSIOLOGY OF ATHEROSCLEROSIS (FIGS 3.1 AND 3.2)

Atherosclerosis is the main cause of coronary artery disease. The process begins as disruption of endothelial function due to the accumulation of lipoprotein droplets in the intima of the coronary vessels. Water insoluble lipids are carried in the bloodstream attached to water soluble apolipoproteins (lipoproteins). High concentrations of low density lipoprotein (LDL) can permeate an already disrupted or dysfunctional endothelium where it undergoes oxidation and, in diabetics, glycation. Modified LDL attracts leukocytes into the intima and can be scavenged by macrophages leading to the formation of foam cells. These cells replicate giving rise to one of the earliest pathological lesions; the fatty streak. The fatty streak is the earliest visualized lesion of atherosclerosis.

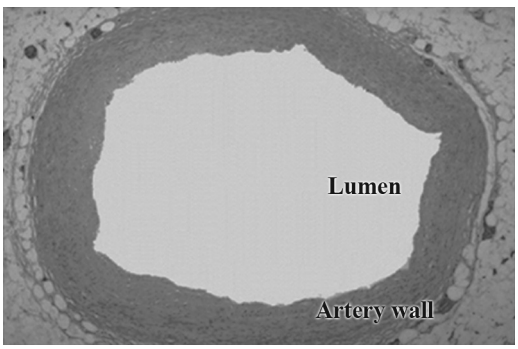


Figure 3.1: Microscopic view of normal coronary artery

Smooth muscle cells are then recruited and they migrate to the site of the foamy cells. Smooth muscle cells proliferate and manufacture extracellular matrix. A large volume of the plaque is occupied by extracellular matrix (collagen and proteoglycan) secreted by the smooth muscle cells. The fatty streak is now transformed into the fibrous plaque. At this point, the lesion begins to encroach on the lumen of the vessel. Small blood vessels form in these plaques (angiogenesis) and these plaques can subsequently calcify. Inflammation plays an important role in promoting smooth muscle cell migration and proliferation. The final lesion, the advanced complicated lesion, consists of a fibrous cap overlying a lipid rich core which also contains necrotic material, this core is highly thrombogenic.

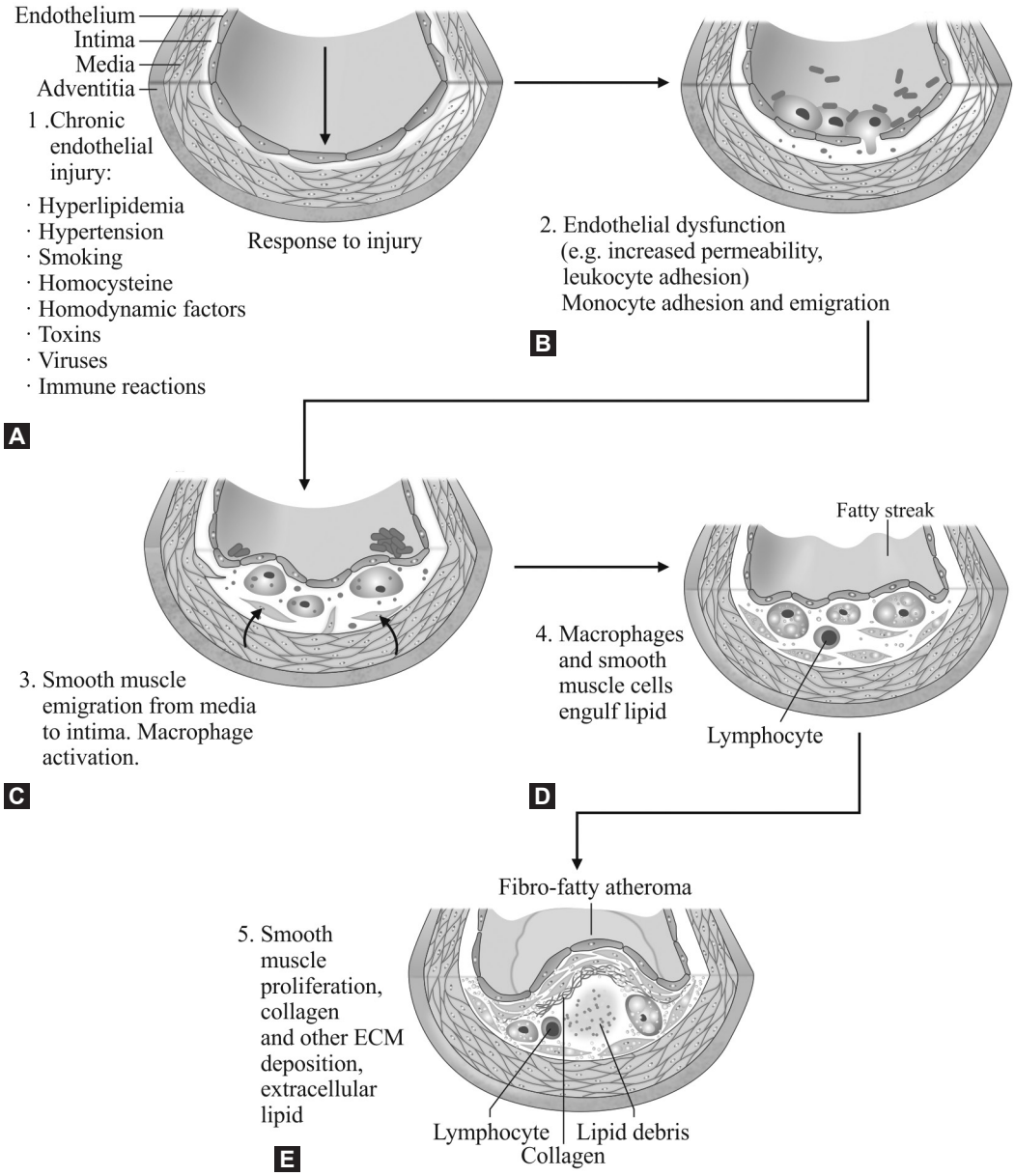
THEORIES OF PATHOGENESIS

The various theories of CAD are summarized in Table 3.1.

SIGNS AND SYMPTOMS

The following signs and symptoms may occur:

- Angina, the classic sign of CAD, results from a reduced supply of oxygen to the myocardium (Table 3.2). It may be described as
  - Burning, squeezing, or tightness in the chest that may radiate to the left arm, neck, jaw, or shoulder blade
- Nausea and vomiting as a result of reflex stimulation of the vomiting centers by pain
- Cool extremities and pallor caused by sympathetic stimulation



Figures 3.2A to E: (A) Response to injury; (B) Endothelial dysfunction; (C) Initiation of fatty streak; (D) Fatty streak; (E) Fibro-fatty atheroma

- Diaphoresis due to sympathetic stimulation
- Xanthelasma (fat deposits on the eyelids) occurs secondary to hyperlipidemia and atherosclerosis
- CAD may be asymptomatic in the older adult because of a decrease in sympathetic response

Table 3.1: Theories for coronary artery disease

Hypothesis	*Mechanism of atherogenesis*
Response to injury	Arterial endothelium is injured by altered hemodynamic force (hypertension) bacterial infection, hyperlipidemia, endothelial membrane is disturbed, permanently is altered, and platelets, lipids, smooth muscles cells aggregate on the intimal layers
Senescence hypothesis	Suggests that the development of atherosclerosis occurs with age, smooth muscle cell proliferation results from an age dependence in control over replication of intimal smooth muscle cells
Thrombogenic	The aggregation of platelets at the injury site converts into connective, results from the migration of smooth muscle cells and fibroblasts from arterial wall into the thrombus
Lipid-irritation	Holds that the deposit of lipids in the arterial wall from the blood acts as an irritant that induces proliferation of smooth muscle cells
Hemodynamic	Attempts to explain plaque formation, results from pressure, stress that all acts as irritation of endothelial wall

Table 3.2: Types of angina

Types	*Description*
Stable angina	Pain is predictable in frequency and duration and is relieved by rest and nitroglycerin
Unstable angina	Pain increases in frequency and duration and is more easily induced; it indicates a worsening of coronary artery disease that may progress to myocardial infarction
Prinzmetal's or variant angina	Pain is caused by spasm of the coronary arteries; it may occur spontaneously and may not be related to physical exercise or emotional stress
Microvascular angina	Impairment of vasodilator reserve causes angina-like chest pain in a person with normal coronary arteries
Post-infarction angina	Pain occurs after MI, when residual ischemia may cause episodes of angina
Intractable angina	It is severe chest pain
Nocturnal angina and angina decubitus	Nocturnal angina occurs only at night but not necessarily when the person is in the recumbent position or during sleep. Angina decubitus is chest pain that occurs only while the person is lying down and is usually relieved by standing or sitting

- Dyspnea and fatigue are two key signals of ischemia in an active, older adult.

DIAGNOSIS

The following tests help diagnose coronary artery disease:

- Electrocardiography may be normal between anginal episodes. During angina, it may show ischemic changes, such as T-wave inversion, ST-segment depression and, possibly, arrhythmias. ST-segment elevation suggests Prinzmetal's angina
- Exercise testing may be performed to detect ST-segment changes during exercise, indicating ischemia, and to determine a safe exercise prescription
- Coronary angiography reveals location and degree of coronary artery stenosis or obstruction, collateral circulation, and the condition of the artery beyond the narrowing

- Myocardial perfusion imaging with thallium-201 may be performed during treadmill exercise to detect ischemic areas of the myocardium; they appear as "cold spots"
- Stress echocardiography may show abnormal wall motion.

TREATMENT

Treatment of CAD may involve:

- Nitrates, such as nitroglycerin (given sublingually, orally, transdermally, or topically in ointment form) or isosorbide dinitrate (given sublingually or orally) to reduce myocardial oxygen consumption
- Beta-adrenergic blockers to reduce the workload and oxygen demands of the heart by reducing heart rate and peripheral resistance to blood flow
- Calcium channel blockers to prevent coronary artery spasm
- Antiplatelet drugs to minimize platelet aggregation and the risk of coronary occlusion
- Antilipemic drugs to reduce serum cholesterol or triglyceride levels
- Antihypertensive drugs to control hypertension
- Oestrogen replacement therapy to reduce the risk for CAD in postmenopausal women.

SURGICAL MANAGEMENT

- Coronary Artery Bypass Graft (CABG) surgery to restore blood flow by bypassing an occluded artery using another
- Percutaneous transluminal coronary angioplasty: It is a surgical technique in which a balloon-tipped catheter is inserted into blocked coronary artery. The balloon is inflated to compress the plaque and open the artery, this procedure is called balloon angioplasty.
- Atherectomy is a procedure that uses a device consisting of a balloon and motorized blockage and mechanically positioned against blockage and mechanically debris of the plaque removed from the artery.
- Laser are currently being used with balloon angioplasty to vaporize atherosclerotic plaque.

MYOCARDIAL INFARCTION

In myocardial infarction (MI)—also known as a heart attack—reduced blood flow through one of the coronary arteries results in myocardial ischemia and necrosis. Cardiovascular disease is the leading cause of death in the United States and Western Europe, death usually results from cardiac damage or complications of MI. Each year, approximately 900,000 people in the United States experience MI. Mortality is high when treatment is delayed, and almost half of sudden deaths due to MI occur before hospitalization, within 1 hour of the onset of symptoms. The prognosis improves if vigorous treatment begins immediately.

Causes

Predisposing risk factors include:

- Hereditary
- Gender (men and postmenopausal women are more susceptible to MI than premenopausal women, although the incidence is rising among women, especially those who smoke and take oral contraceptives)
- Hypertension (>140/90 mmHg risk for MI, younger age)
- Smoking
- Elevated serum triglyceride, total cholesterol, and low-density lipoprotein levels

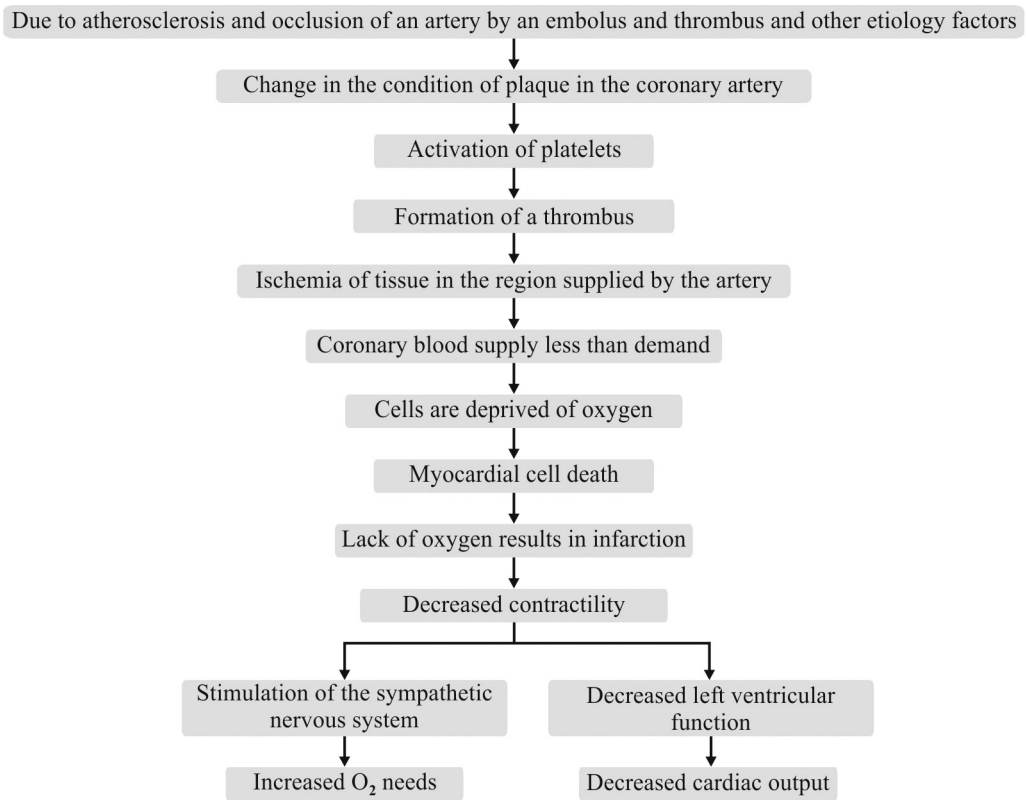
- Obesity
- Excessive intake of saturated fats
- Sedentary lifestyle
- Aging (35 to 40 years male are more common)
- Stress or type A personality
- Drug use, especially cocaine and amphetamines.

Pathophysiology (Flow chart 3.1)

MI results from occlusion of one or more of the coronary arteries. Occlusion can stem from atherosclerosis, thrombosis, platelet aggregation, or coronary artery stenosis or spasm. If coronary occlusion causes prolonged ischemia, lasting longer than 30 to 45 minutes, irreversible myocardial cell damage and muscle death occur. All MIs have a central area of necrosis or infarction surrounded by an area of potentially viable hypoxic injury. This zone may be salvaged if circulation is restored, or it may progress to necrosis. The zone of injury, in turn, is surrounded by an area of viable ischemic tissue.

Although ischemia begins immediately, the size of the infarct can be limited if circulation is restored within 6 hours. Several changes occur after MI. Cardiac enzymes and proteins are released by the infarcted myocardial cells, which are used in the diagnosis of an MI. Within 24 hours, the infarcted muscle becomes

Flow chart 3.1: Pathophysiology of MI



edematous and cyanotic. During the next several days, leukocytes infiltrate the necrotic area and begin to remove necrotic cells and thinning the ventricular wall. Scar formation begins by the third week after MI, and by the sixth week, scar tissue is well-established.

Clinical Manifestation

Cardiovascular System

- Chest pain or discomfort, palpitation, new onset of murmur
- Increased jugular venous distention may be seen in the MI caused by heart failure
- Blood pressure may be elevated because of sympathetic stimulation
- ST–segment, T wave changes
- ECG shows tachycardia, bradycardia, dysrhythmias.

Respiratory System

- Shortness of breath
- Dyspnea
- Tachycardia
- Crackles MI has caused pulmonary congestion
- Pulmonary edema.

GI System

- Nausea and vomiting.

Genitourinary System

- 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.

Neurologic

- Anxiety, restlessness, headache, visual disturbance and altered speech.

Psychological

- Fear with feeling of impending doom or patient may think that something is wrong with him.

Diagnostic Evaluation (Table 3.3)

- Serial 12-lead electrocardiography (ECG) may reveal characteristic changes, such as serial ST-segment depression in non-Q-wave MI (subendocardial MI that affects the innermost myocardial layer) and ST-segment elevation in Q-wave MI (transmural MI with damage extending through all myocardial layers). An ECG can also identify the location of MI, arrhythmias, hypertrophy, and pericarditis.
- Serial cardiac enzymes and proteins may show a characteristic rise and fall of cardiac enzymes, specifically CK-MB, and the proteins troponin T and I, and myoglobin to confirm the diagnosis of MI.
- Laboratory testing may reveal elevated white blood cell count and erythrocyte sedimentation rate due to inflammation, and increased glucose levels following the release of catecholamines.
- Echocardiography may show ventricular wall motion abnormalities and may detect septal or papillary muscle rupture.
- Chest X-rays may show left-sided heart failure or cardiomegaly.

Table 3.3: Types of MI and ECG leads placement

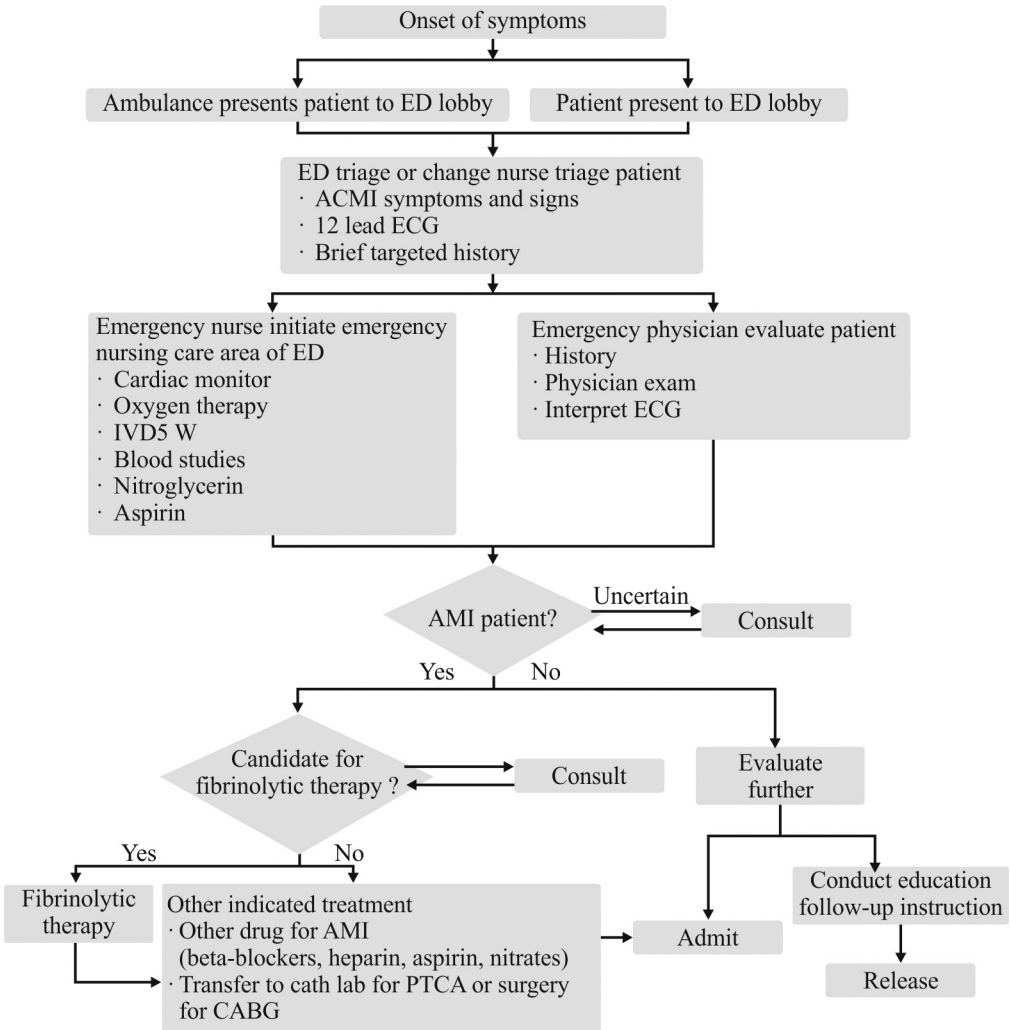
Types of myocardial infarction	*Leads*
Inferior	II, III, aVF
Anterior	V3, V4
Septal	V1, V2
Lateral	I, aVL, V5, V6
Anterolateral	I, aVL, V3-V6
Posterior	V1 or V2
Right ventricular	II, III, aVF, V1R–V4R

- Nuclear imaging scanning using thallium-201 and technetium 99m can be used to identify areas of infarction and areas of viable muscle cells.
- Cardiac catheterization may be used to identify the involved coronary artery as well as to provide information on ventricular function and pressures and volumes within the heart.

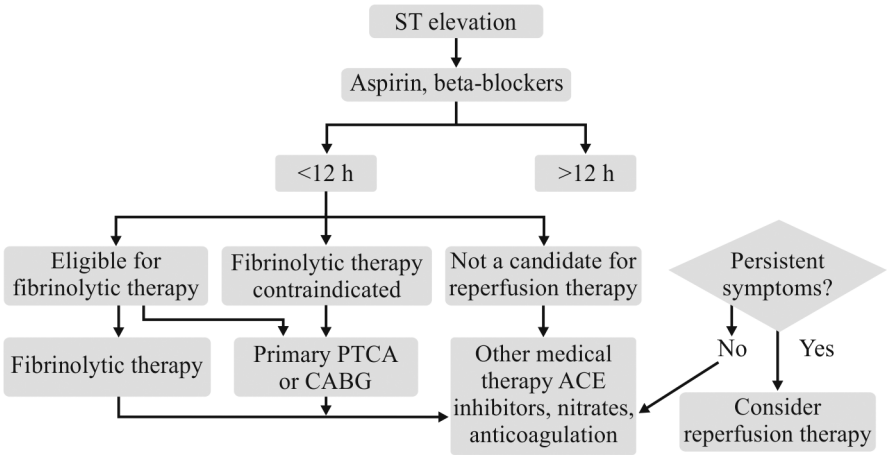
Medical Management (Flow charts 3.2 to 3.4)

- Assessment of patients with chest pain in the emergency department within 10 minutes of an MI because at least 50% of deaths take place within 1 hour of the onset of symptoms. Moreover, thrombolytic therapy is most effective when started

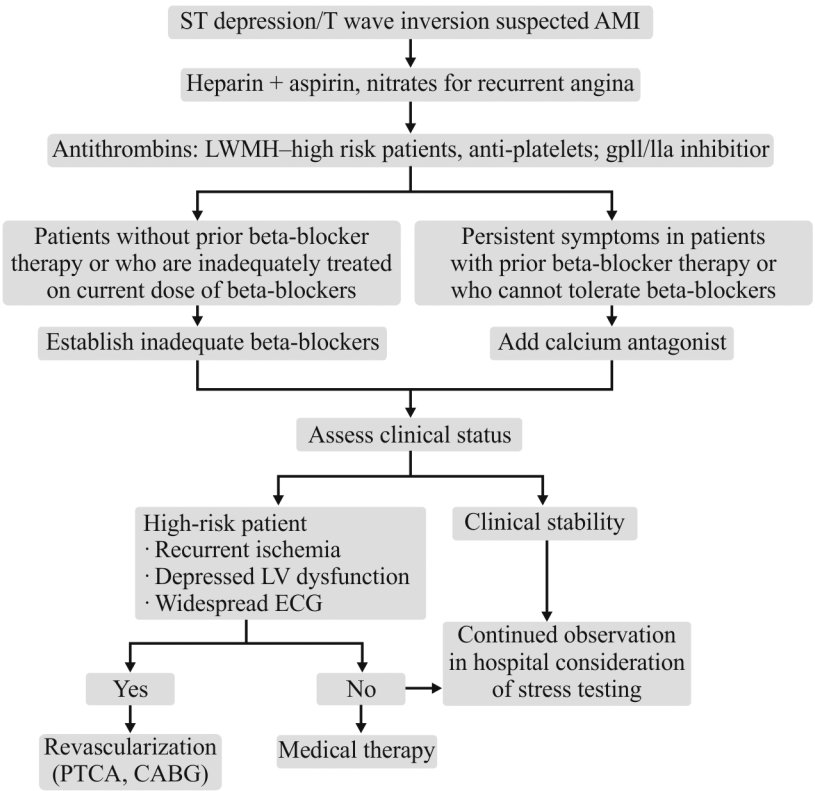
Flow chart 3.2: Emergency department algorithm—protocol for patients with symptoms or signs of MI



Flow chart 3.3: Recommended treatment algorithm for the management of patients with ST-elevation MI



Flow chart 3.4: Recommended treatment algorithm for the management of patients with non ST-elevation MI



within the first 6 hours after the onset of symptoms
- Oxygen by nasal cannula for 2 to 3 hours to increase oxygenation of the blood
- Nitroglycerin sublingually to relieve chest pain, unless systolic blood pressure is less than 90 mmHg or heart rate is less than 50 or greater than 100 beats per minute, morphine or meperidine (Demerol) for analgesia because pain stimulates the sympathetic nervous system, leading to an increase in heart rate and vasoconstriction.

Aspirin

Give stat 320 mg on day of Acute Myocardial Infarction (AMI). Then 160 mg daily -to inhibit platelet aggregation.

Nitroglycerin

Indications

For first 24–48 hours in patients with AMI and CHF, large anterior infarction, persistent ischemia or hypertension. Continued use beyond 48 hours in patients with recurrent angina or persistent pulmonary congestion.

Contraindications

- Initial systolic BP <90 mmHg
- Marked bradycardia (<50 bpm) or tachycardia
- Suspected RV infarction.

Thrombolysis—Heparin

Indications

- IV unfractionated heparin (UH) for patients post- thrombolysis with rt-PA
- IV UH for patients undergoing primary percutaneous coronary intervention (PCI)
- LMWH SC for patients with NSTEMI (enoxaparine 1 mg/kg bd) not given thrombolysis and with no contraindications to heparin.

Action: To dissolve and lyse the thrombus in a coronary artery, allowing blood to flow through the coronary artery again (reperfusion) minimizing the size of the infarction and preserving ventricular function.

Beta-blockers

Indications

- Start within 12 hours post-AMI if no contraindication irrespective of concomitant, thrombolysis or primary PCI
- Continuing or recurrent ischemic pain
- Tachyarrhythmias, e.g. rapid AF NSTEMI.

Action: It is used to decrease myocardial oxygen demand by reducing HR, and shown to reduce the size of the infarction.

ACEI

Indications

Within first 24 hours of a suspected AMI with clinical heart failure in absence of hypotension (SBP>100 mmHg). AMI with LV ejection fraction <40% or patients with clinical heart failure during and after convalescence from AMI.

Action: Prevent the conversion of angiotension from I to II. The abscence of angiotension II, the blood pressure decreases oxygen demand of heart.

Anti-dysrhythmias Drugs

- Class I—sodium channel blocks (phenytoin)
- Class II—beta-adrenergic blocks (atenolol)
- Class III—potassium channel blocks (cordarone)
- Class IV—calcium channel blocks (verapamil).

Cholesterol Lowering Drugs

Examples are niaan, ezetimble.

Stool Softeners

Patient may be precrubed to constipation as a result of bedrest and opoid administration. For example, colace.

Intra-aortic Balloon Counterpulsation

Indications for Placement

- Cardiogenic shock not quickly reversed with pharmacological therapy as a stabilizing measure for angiography and prompt revascularization
- Acute mitral regurgitation or VSD complication gMI as a stabilizing therapy for angiography and repair/revascularizton
- Recurrent intractable ventricular arrhythmias with hemodynamic instability
- Refractory post-MI angina as a bridge to angiography and revascularization.

Surgical Management

- Percutaneous transluminal coronary angioplasty (PTCA) may be an alternative to thrombolytic therapy if it can be performed in a timely manner in an institution with personnel skilled in the procedure.
- Coronary artery bypass grafting surgery: It consists of the construction of new conduits (vessels to transport blood) between the aorta, or other major arteries, and the myocardium distal to the obstructed coronary artery. The procedure involves one or more grafts using internal mammary artery, saphenous vein and radial artery.
- Minimally invasive direct coronary artery bypass (MIDCAB): It is a technique that offers the patient with single vessels disease. the technique requires several small invasions between the ribs, off pump coronary artery bypass procedure uses full or partial sternotomy to enable access to all coronary vessels.
- Transmyocardial laser revascularization: It is an indirect revascularization procedure used for patients with advanced CAD who are not candidates for traditional procedures. The procedures involve the use of a high energy laser that triggered electrocardiographically to create channel between the left ventricular cavity and the coronary microcirculation.

Complications

Complications of MI include:

- Arrhythmias
- Carcinogenic shock
- Heart failure causing pulmonary edema
- Pericarditis
- Rupture of the atrial or ventricular septum, ventricular wall, or valves
- Mural thrombi causing cerebral or pulmonary emboli
- Ventricular aneurysms
- Myocardial rupture
- Extensions of the original infarction.

Nursing Management

Teaching about Angina

- When a patient experiences angina, the nurse should direct the patient to stop all activates and sit or rest in the bed in a semi-fowler's position to reduce the oxygen requirement of the ischemic myocardium
- Administer nitroglycerin sublingually.

Reducing Anxiety

- Various stress reduction methods should be explored with the patient, e.g. music therapy
- Addressing the spiritual needs of the patients and family may also assist in alleging anxiety and tears.

Reducing Pain

- Balance activity and rest are important aspects of the educational plan for the patient and family in improving respiratory function
- Regular and careful assessment of respiratory function can help detect clearly signs of pulmonary complications

- Fluid volume status prevents overloading the heart and lungs.

Promoting Adequate Tissue Perfusion

- Checking skin temperature and peripheral pulses frequently is important to ensure adequate tissue perfusion
- Administer the oxygen adequately.

Self Administration of Nitroglycerin

Teaching About Sublingual Nitroglycerin

- Instruct the patient to make sure that the mouth is moist, the tongue is still, and salvia is not swallowed until the nitroglycerin tablet dissolves; if the pain is severe, the patient can rush the tablets between the teeth to hasten sublingual absorption
- Educate the patient to carry the medications at all times as treatment precaution. However NTG is very unstable, it should be carried securely in its original container (cupped dark glass bottle), tablet should never be removed and stored in metal or plastic pillbox
- Explain the patient that NTG is volatile and inactivated by heat, moist, air, light and time, renew the supply every 6 months
- Tablet should be taken before angina producing activity (exercise, sexual intercourse) teaching about side effects (flushing, throbbing, headache, hypotension, tachycardia).

Topical NTG

- Availablas lanolin-petrolatum paste or patch
- Avoid applying on skin irritation
- Should not be applied on skin with excessive hair or scar tissue
- Most physicians prescribe application of topical nitroglycerin paste 3 times or 4 times daily every 6 hours (excluding mid-night dose)
- Physicians prescribe application of topical nitroglycerin patch every morning and removal at 10 pm.

Nursing Diagnoses Care of Patient with MI

- Acute pain (chest) related to imbalance myocardial oxygen supply and demand
- Ineffective tissue perfusion related to myocardial injury as evidence in dyspnea, dysrhythimas and peripheral edema
- Decreased cardiac output related to electrical factors, decreased myocardial contractility
- Anxiety related to perceived or actual threat of death, pain, possible lifestyle changes
- Activity intolerance related to generalized weakness secondary to decreased cardiac output
- Potential impaired gas exchange related to fluid overload left ventricular dysfunction.

BIBLIOGRAPHY

1. Ashley EA, Niebauer J. Cardiology. 1st edition. London. Published by Remedica; 2004.
2. Block JM. Medical Surgical Nursing. 6th edition. New York. WB Saundars Publication; 2002.
3. Davidson's Principles and Practice of Medicine. 50th edition. Philadelphia. Churchill Livingstone and Elsevier Science Limited; 2002.
4. Ghosh AK. Mayo Clinic Internal Medicine Review. 8th edition. Canada. Mayo Clinic Scientific Press; 2008.
5. Harrison's Principles of Internal Medicine. 16th edition. New York. McGraw-Hill Medical Publishing Division; 2005.
6. Lewis. Medical Surgical Nursing. 7th edition. New Delhi. Mosby Publication; 2007.

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