Management of Patients With Coronary Vascular Disorders
Coronary Artery Disease

Insufficient blood flow to the heart muscle from narrowing of coronary artery may cause chest pain.

Plaque in coronary artery
CORONARY ATHEROSCLEROSIS

- Is an abnormal accumulation of lipid, or fatty substances and fibrous tissue in the vessel wall

- These substances create blockages or narrow the vessel in a way that reduces blood flow to the myocardium

- Atherosclerosis is a progressive disease that can be curtailed and, in some cases, reversed
Artery with Atherosclerosis

Disease Progression

Normal | Fatty Streaks | Plaque | Stenosis

Arterial Wall
Lumen
Blood Cells
Cholesterol
Pathophysiology

- Atherosclerosis begins as fatty streaks, lipids that are deposited in the intima of the arterial wall

- \( T \) lymphocytes and monocytes (that become macrophages) infiltrate the area to ingest the lipids and then die; this causes smooth muscle cells within the vessel to proliferate and form a fibrous cap over the dead fatty core

- These deposits, called atheromas or plaques, protrude into the lumen of the vessel, narrowing it and obstructing blood flow
If the fibrous cap of the plaque is *thick* &

the lipid pool remains relatively *stable*,

it can resist the stress from blood flow and vessel movement.

If the cap is *thin*,

- The lipid core may *grow*;
- Causing it to *rupture* and *hemorrhage* into the plaque;
- Allowing a *thrombus* to develop.

The thrombus may obstruct blood flow ➔ leading to sudden cardiac death or an acute myocardial infarction (MI), which is the death of heart tissue.
Atherosclerosis is a condition where fatty deposits build up inside the walls of the arteries. This process begins with the formation of early atheroma, characterized by the deposition of lipid-rich material on the arterial intima. As the atheroma progresses, it can develop into a 'stabilized' plaque, which is characterized by a small lipid pool, a thick fibrous cap, and a preserved lumen.

Further progression can lead to a 'vulnerable' plaque, characterized by a thin fibrous cap, a large lipid pool, and numerous inflammatory cells. When a vulnerable plaque develops, it can lead to thrombosis of a ruptured plaque, which forms a fibrous cap. Over time, this can result in a healed ruptured plaque with a narrow lumen and fibrous intima.

Left coronary arteries and acute myocardial infarction are also illustrated, showing the potential consequences of atherosclerosis in the heart.
**Pathophysiology**

- Angles of the coronary arteries

- The many angles and curves of the coronary arteries contribute to the vessels’ susceptibility to atheromatous plaques

- Arteries shown as dashed lines supply the posterior wall of the heart
Causes of heart disease

- Is most often caused by atherosclerosis of the coronary arteries
- Decrease blood flow to the heart

1. vasospasm (sudden constriction or narrowing) of a coronary artery
2. myocardial trauma from internal or external forces, structural disease
3. Congenital anomalies
4. Decreased oxygen supply
   (eg, from acute blood loss, anemia, or low blood pressure)
5. Increased demand for oxygen
   (eg, from rapid heart rate, thyrotoxicosis, or ingestion of cocaine)
Clinical Manifestations

Symptoms and complications according to:

1. Location and degree of narrowing of the arterial lumen

2. Thrombus formation

3. Obstruction of blood flow to the myocardium
**Clinical Manifestations**

- **Angina pectoris refers to**
  - Chest pain that is brought about by myocardial ischemia

- **If the ischemia is great enough, of long duration, or both**
  - Irreversible damage and death of myocardial cells, or MI

- **Over time, irreversibly damaged myocardium undergoes degeneration and is replaced by scar tissue, causing various degrees of myocardial dysfunction**
Significant myocardial damage may cause:

- Inadequate cardiac output
- The heart cannot support the body’s needs for blood, which is called heart failure (HF)

A decrease in blood supply from CAD may even cause the heart to stop abruptly, called sudden cardiac death.
Clinical Manifestations of CAD

- Acute onset of chest pain
- May be asymptomatic
  
  \(\text{(in those older, women, have diabetes, a history of heart failure)}\)

Typical symptoms:

- shortness of breath
- Nausea
- unusual fatigue
- changes on the (ECG)
- high levels of cardiac enzymes
- Dysrhythmias
- sudden death
Risk Factors

- Use of tobacco
- Hypertension
- Elevated blood lipid levels
- Family history of premature cardiovascular disease
  - (first-degree relative with cardiovascular disease at age 55 or younger for men and at age 65 or younger for women)
- Age
  - >45 years for men
  - >55 years for women
Nonmodifiable Risk Factors for Coronary Artery Disease

- Family history of coronary heart disease
- Increasing age
- Gender
  
  (heart disease occurs three times more often in men than in premenopausal women)

- Race
  
  (higher incidence of heart disease in African Americans than in Caucasians)
Modifiable Risk Factors for Coronary Artery Disease

- High blood cholesterol level
- Cigarette smoking, tobacco use
- Hypertension
- Diabetes mellitus
- Lack of estrogen in women
- Physical inactivity
- Obesity
Coronary Artery Disease Risk Equivalents

- Individuals at highest risk for a cardiac event within 10 years are those with existing (CAD) & those with any of the following diseases, which are called CAD risk equivalents:
  
  - Diabetes
  - Peripheral arterial disease
  - Abdominal aortic aneurysm
  - Carotid artery disease
CONTROLLING CHOLESTEROL ABNORMALITIES:

- The desired goal is to have low LDL values and high HDL values

The desired level of LDL depends on the patient:

- Patients with one or no risk factors
  Less than 160 mg/dL

- Patients with two or more risk factors
  Less than 130 mg/dL

- Patients with CAD or a CAD risk equivalent
  Less than 100 mg/dL
Control of LDL

- Serum cholesterol and LDL levels can usually be controlled by diet and physical activity.
- Medication therapy.
CONTROLLING CHOLESTEROL ABNORMALITIES

- The level of HDL should exceed $40 \text{ mg/dL}$ and should ideally be more than $60 \text{ mg/dL}$.

- A high HDL level is a strong negative risk factor (is protective) for heart disease.
Controlling TG Abnormalities

- Triglyceride is another fatty substance, made up of fatty acids, that is transported through the blood by a lipoprotein.

- Although an elevated triglyceride level (>200 mg/dL) may be due to:
  1. Genetic in origin
  2. Obesity
  3. Physical inactivity
  4. Excessive alcohol intake
  5. High-carbohydrate diets
  6. Diabetes mellitus
  7. Kidney disease
  8. Medications (birth control pills, corticosteroids, and beta-adrenergic blockers when given in higher doses)
Management of elevated triglyceride

- Weight reduction
- Increased physical activity
- Medications
Dietary Measures

- Soluble fibers, which are found in fresh fruit, cereal grains, vegetables, and legumes, enhance the excretion of metabolized cholesterol

**Table 28-1 • Nutrient Content of the Therapeutic Lifestyle Changes (TLC) Diet**

<table>
<thead>
<tr>
<th>NUTRIENT</th>
<th>RECOMMENDED INTAKE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total calories*</td>
<td>Balance intake and expenditure to maintain desirable weight</td>
</tr>
<tr>
<td>Total Fat</td>
<td>25%–35% of total calories</td>
</tr>
<tr>
<td>Saturated fat†</td>
<td>&lt;7% of total calories</td>
</tr>
<tr>
<td>Polyunsaturated Fat</td>
<td>Up to 10% of total calories</td>
</tr>
<tr>
<td>Monounsaturated Fat</td>
<td>Up to 20% of total calories</td>
</tr>
<tr>
<td>Carbohydrate‡</td>
<td>50%–60% of total calories</td>
</tr>
<tr>
<td>Fiber</td>
<td>20–30 g/day</td>
</tr>
<tr>
<td>Protein</td>
<td>Approximately 15% of total calories</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>&lt;200 mg/day</td>
</tr>
</tbody>
</table>
Physical Activity

- Regular, moderate physical activity increases HDL levels and reduces triglyceride levels

- The goal for the average person is a total of 30 minutes of exercise, three to four times per week
Patients should begin with a *5-minute warm-up* period to stretch and prepare the body for the exercise.

The nurse should inform patients to stop any activity if they develop:
- Chest pain
- Unusual shortness of breath
- Dizziness
- Lightheadedness
- Nausea
Effects of smoking:

- The inhalation of smoke
- Increases the blood *carbon monoxide* level
  - Hemoglobin to *combine with CO than with O₂*
  - Decreased amount of *available oxygen*
- Decrease the *heart’s ability to pump*
Effects of smoking:

- The nicotinic acid in tobacco
  - Triggers the release of catecholamines
  - Raise the heart rate and blood pressure
  - Coronary arteries to constrict
  - May be a factor in the increased incidence of sudden cardiac death
Effects of smoking:

- **Use of tobacco**
  - Increases platelet adhesion
  - Higher probability of thrombus formation

People who stop smoking reduce their risk of heart disease by 30% to 50% within the first year, and the risk continues to decline as long as they refrain from smoking.
Managing Hypertension

Hypertension is defined

- Blood pressure measurements that repeatedly exceed 140/90 mm Hg

Long-standing elevated blood pressure may

- Increased stiffness of the vessel walls
- Leading to vessel injury
- Resulting inflammatory response within the intima
Hypertension can also

- Increase the work of the left ventricle
- Pump harder to eject blood into the arteries

Over time

- Heart to enlarge and thicken (HYPERTROPHY)
  condition that may eventually lead to cardiac failure
Hypertrophic stimuli

G-protein-coupled receptors

Induction of hypertrophic programs

NAB-1

Egr-1

R1

RV

LV

Left ventricular hypertrophy

Heart failure
Which of them are more compliant?
DEPARTAMENTO DE ANATOMIA Y ANATOMIA PATOLOGICA COMPARADAS
FACULTAD DE VETERINARIA UNIVERSIDAD DE CORDOBA
CONTROLLING DIABETES MELLITUS

2/3 to 3/4 of diabetic patient death related to Cardiovascular Disease

Hyperglycemia

✓ Fosters dyslipidemia
✓ Increased platelet aggregation
✓ Altered red blood cell function

All of above factor can lead to thrombus formation
Gender and Estrogen Level

- Women tend not to recognize the symptoms as early as men and to *wait longer to report their symptoms and seek medical assistance*!!!

- In women *younger than age 55*, the incidence of CAD is *significantly lower than in men*

- After *age 55*, the incidence in women is approximately equal to that in men
Hormone replacement therapy (HRT) has decreased *postmenopausal symptoms* and the *risk for osteoporosis related bone fractures*.

*but*

HRT also has been associated with:

- *CAD*
- *BC*
- *DVT*
- *CVA*
- *PE*
ANGINA PECTORIS
Angina Pectoris

- Angina pectoris is a clinical syndrome usually characterized by episodes or paroxysms of pain or pressure in the anterior chest.

- The cause is usually insufficient coronary blood flow.
Factors associated with typical anginal pain:

- Physical exertion
  - Increasing myocardial oxygen demand

- Exposure to cold
  - Vasoconstriction ➔ Elevated blood pressure ➔ Increased oxygen demand

- Eating a heavy meal
  - Increases the blood flow to the mesenteric area for digestion
  - Reducing the blood supply available to the heart muscle

- Stress or any emotion-provoking situation
  - Release of adrenaline
  - Increasing BP ➔ Accelerate the HR ➔ Increase the myocardial workload
Types of Angina

Stable angina:
- predictable and consistent pain that **occurs on exertion** and is **relieved by rest**

Unstable angina: (also called pre infarction angina or crescendo angina)
- symptoms occur more frequently and last longer than stable angina
- The **threshold** for pain is **lower**, and pain may occur at **rest**

Intractable or refractory angina:
- severe **incapacitating** chest pain
Types of Angina (Cont)

Variant angina (also called Prinzmetal’s angina):
- Pain at rest with reversible ST-segment elevation
- Thought to be caused by coronary artery vasospasm

Silent ischemia:
- Objective evidence of ischemia
  (such as ECG changes with a stress test)
- Patient reports no symptoms
Clinical Manifestations

- A feeling of indigestion to a choking
- Heavy sensation in the upper chest
- Pain accompanied by severe apprehension
- Feeling of impending death.
- Retrosternal pain, tightness
- The pain or discomfort is poorly localized and
- May radiate to the neck, jaw, shoulders, and inner aspects of the upper arms, usually the left arm
Clinical Manifestations

- A feeling of weakness or numbness
  Arms, wrists, and hands may accompany the pain
- Shortness of breath,
- Pallor
- Diaphoresis
- Dizziness or lightheadedness
- Nausea and vomiting
Assessment and Diagnostic Findings

- Clinical manifestations of ischemia
- Patient’s history
- A 12-lead ECG
- Blood laboratory values
- Echocardiogram
- C-reactive protein (CRP):

  High blood levels of CRP have been associated with increased coronary artery calcification and risk of an acute cardiovascular event (eg, MI) in seemingly healthy individuals.
Medical Management

The objectives of the medical management of angina:

- Decrease the oxygen demand of the myocardium
- Increase the oxygen supply

Medically, these objectives are met through:

- Pharmacologic therapy
- Control of risk factors
Revascularization procedures

Percutaneous coronary interventional (PCI) procedures:

- Percutaneous transluminal coronary angioplasty (PTCA)
- Intracoronary stents
- Atherectomy
- CABG

Percutaneous transluminal myocardial revascularization (PTMR)

PHARMACOLOGIC THERAPY

**Nitroglycerin:**

Actions:

1. Reduce myocardial oxygen consumption
   - Decreases ischemia and relieves pain
2. Dilates primarily the veins
3. Dilates the arteries *(High Dose)*
4. Increase coronary blood flow by
   - Preventing vasospasm
   - Increasing perfusion through the collateral vessels
Nitroglycerin

Dilation of the veins

⇒ *venous pooling of blood* throughout the body
⇒ decrease *venous returns*
⇒ Decreasing *filling pressure* (preload) is reduced

If the patient is hypovolemic, the decrease in filling pressure can cause a significant decrease in cardiac output and blood pressure.
**Route of administration**

- **Sublingual tablet or spray:**
  - Alleviates the pain of ischemia within 3 minutes

- **Topical agent:**
  - Fast acting and is a convenient way to administer the medication

- **Intravenous administration:**
  - According patient symptom
Precautions on IV administration

The amount of nitroglycerin administered is based on the patient’s symptoms.

Observe BP for hypotension.

It usually is not given if the systolic blood pressure is 90 mm Hg or less.

After the patient is symptom free, the nitroglycerin may be switched to a topical preparation within 24 hours.
Beta-Adrenergic Blocking Agents

- Propranolol (Inderal)
- Atenolol (Tenormin)
**Actions**

- Reduce myocardial oxygen consumption
  - Blocking the beta-adrenergic sympathetic stimulation to the heart
  - The result is a reduction in HR
  - Slowed conduction of an impulse through the heart
  - Decreased BP
  - Reduced myocardial contractility (force of contraction)
  - Establishes a more favorable balance between (demands) and available (supply)

- Reduce the incidence of recurrent angina, infarction, and cardiac mortality
Cardiac side effects and possible contraindication

- Hypotension
- Bradycardia
- Advanced atrioventricular block
- Decompensated heart failure
Precautions on administration:

- If a beta-blocker is given intravenously, Monitor
  - ECG
  - Blood pressure
  - Heart rate

- Contraindicated in Pt. with bronchial asthma
- Should not be stopped abruptly

- Patients with diabetes who take beta-blockers are instructed to assess their blood glucose levels more often and to observe for signs and symptoms of hypoglycemia
Precautions on administration

Beta Blocker Agents:

Hypoglycemic S&S

- Sweating
- Nervousness
- Tremor
- Tachycardia
- Hunger
- Neurologic symptoms ranging from bizarre behavior and confusion to seizures and coma.
Calcium Channel Blocking Agents: (diltiazem)

Decrease:
- SA node automaticity
- and
- AV node conduction

Resulting in:
1. Slower HR
   (negative Chronotrop effect)
2. Decrease in the strength of the heart muscle contraction
   (negative inotropic effect)
3. Decrease the workload of the heart
Calcium Channel Blocking Agents: (diltiazem)

Relax the blood vessels ➔
- Decrease in BP
- Increase in coronary artery perfusion

Dilating the smooth muscle wall of the coronary arterioles ➔
- Increase myocardial oxygen supply

Reducing systemic arterial pressure and the workload of the LV
- Decrease myocardial oxygen demand
Antiplatelet

- **Aspirin:**
  - It prevents platelet activation
  - Reduces the incidence of MI
  - Reduce Death in patients with CAD

- **Dose:**
  - 160- to 325-mg should be given to the patient with angina *(as soon as the diagnosis is made)*
  - Continued with 81 to 325 mg daily
Antiplatelet

- **Clopidogrel (Plavix):**
  - Given to patients who are allergic to aspirin
  - Given in addition to aspirin in patients at high risk for MI
  - Take a few days to achieve their antiplatelet effect

- They also cause gastrointestinal upset, including:
  - ✗ Nausea
  - ✗ Vomiting
  - ✗ Diarrhea
Other Clopidogrel Side effects:

- Bloody or black, tarry stools
- Nosebleeds
- Pinpoint red or purple spots on your skin or in mouth
- Problems with vision, speech, or walking
- Red or dark brown urine
- Seizures
- Severe stomach pain
- Shortness of breath, tiredness, uneven heartbeat
- Yellowing of skin or the whites of eyes
- Unusual bleeding, bruising, or weakness
- Vomiting of blood or vomit that looks like coffee grounds
Anticoagulant Medications

Heparin:

Unfractionated heparin prevents the formation of new blood clots

The amount of heparin administered is based on

- The results of (aPTT)

Heparin therapy is usually considered therapeutic when

- The aPTT is 1.5 to 2 times the normal aPTT value
Signs and symptoms indicate a significant risk for a cardiac event:

Patient is hospitalized
May be given an intravenous bolus of heparin and
Started on a continuous infusion or
Given an intravenous bolus every 4 to 6 hours

5000u Bolus ➔ 25000/50 cc ➔ 1cc 500 u ➔ 1h 2cc
low-molecular-weight heparin (LMWH): may be used instead of intravenous unfractionated heparin to treat patients with unstable angina or non-ST-segment elevation MIs
Patient is monitored for signs and symptoms of external and internal bleeding, such as:

- Low BP
- Increased HR
- Decrease in serum Hgb and HCT values
Oxygen Administration

Usually initiated at the onset of chest pain

- Increase oxygen delivered to the myocardium
- Decrease pain

The therapeutic effectiveness of oxygen is determined by observing the rate and rhythm of respirations

Blood oxygen saturation is monitored by pulse oximetry, the normal oxygen saturation (SpO2) level is greater than 93%
<table>
<thead>
<tr>
<th>ACRONYM</th>
<th>FACTORS ABOUT PAIN THAT NEED TO BE ASSESSED</th>
<th>ASSESSMENT QUESTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>P</td>
<td>Position/Location</td>
<td>“Where is the pain? Can you point to it?”</td>
</tr>
<tr>
<td></td>
<td>Provocation</td>
<td>“What were you doing when the pain began?”</td>
</tr>
<tr>
<td>Q</td>
<td>Quality</td>
<td>“How would you describe the pain?”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>“Is it like the pain you had before?”</td>
</tr>
<tr>
<td></td>
<td>Quantity</td>
<td>“Has the pain been constant?”</td>
</tr>
<tr>
<td>R</td>
<td>Radiation</td>
<td>“Can you feel the pain anywhere else?”</td>
</tr>
<tr>
<td></td>
<td>Relief</td>
<td>“Did anything make the pain better?”</td>
</tr>
<tr>
<td>S</td>
<td>Severity</td>
<td>“How would you rate the pain on a 0–10 scale with 0 being no pain and 10 being the most amount of pain?” (or use visual analog scale or adjective rating scale)</td>
</tr>
<tr>
<td></td>
<td>Symptoms</td>
<td>“Did you notice any other symptoms with the pain?”</td>
</tr>
<tr>
<td>T</td>
<td>Timing</td>
<td>“How long ago did the pain start?”</td>
</tr>
</tbody>
</table>
Myocardial infarction
Definition

It's the death of apportion of heart muscle in an area where there is sudden loss of blood supply.
Clinical Manifestations

- Chest pain
  - Occurs suddenly and continues despite rest and medication is the presenting symptom in most patients with an MI

- Shortness of breath
- Anxiety and restless
- Cool, pale, and moist skin
- Tachypnea bradycardia dyshyhmia
- May be a symptomatic
- Increase B.P or decrease B.P
- Decrease U.O
- Nausea and vomiting
- Anxiety, fear, headache, visual disturbances, altered speech
Diagnoses of M.I

- ECG (electrocardiogram)
- Laboratory test: cardiac enzyme
  - Creatinine kinase (ck) isoenzymes
  - CK-MB (heart muscle)
  - Troponin: regulate the myocardial contractility
  - LDH
ELECTROCARDIOGRAM

- It should be obtained within **10 minutes** from the time a patient reports pain or arrives in the emergency department.

- The classic ECG changes are:
  - T-wave inversion
  - ST-segment elevation
  - Development of an abnormal Q wave

- The injured myocardial cells depolarize normally.
- But repolarize more rapidly than normal cells.
- ST segment to rise at least 1 mm above the isoelectric line.
Figure 8.1 Sequence of changes seen during evolution of myocardial infarction.
Electrocardiographic Criterion

The most frequently used electrocardiographic criterion for identifying acute myocardial infarction is ➔

*ST segment elevation (two or more anatomically contiguous leads)*
Electrocardiographic Criterion

Early stages of AMI the ECG ➔ May be normal or near normal !!!

Less than half Of Patients With AMI Have clear diagnostic Changes On Their First trace

About 10% of patients with a proved AMI (on the basis of clinical history and enzymatic markers) Fail to Develop ST segment Elevation or depression

In Most cases, however, Serial Electrocardiograms Show Evolving Changes that tend To Follow Well Recognized patterns
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.
Creatine Kinase and Its Isoenzymes.

There are 3 CK isoenzymes:

- CK-MM (skeletal muscle)
- CK-MB (heart muscle): CK-MB is the cardiac-specific isoenzyme found mainly in cardiac cells and rises only when there has been damage to these cells
- CK-BB (brain tissue)
Laboratory Test (Myoglobin)

- Myoglobin is a heme protein that helps to transport oxygen

- It is found in cardiac and skeletal muscle

- The myoglobin level
  - Starts to increase within 1 to 3 hours
  - Peaks within 12 hours after the onset of symptoms

- 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
Laboratory Test (Troponin)

- Protein found in the myocardium, regulates the myocardial contractile process

- Troponins I and T for cardiac muscle, used 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

→ Cannot be used to identify subsequent extension or expansion of an MI
<table>
<thead>
<tr>
<th>SERUM TEST</th>
<th>EARLIEST INCREASE (HR)</th>
<th>TEST RUNNING TIME (MIN)</th>
<th>PEAK (HR)</th>
<th>RETURN TO NORMAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total CK</td>
<td>3–6</td>
<td>30–60</td>
<td>24–36</td>
<td>3 days</td>
</tr>
<tr>
<td>CK-MB: isoenzyme</td>
<td>4–8</td>
<td>30–60</td>
<td>12–24</td>
<td>3–4 days</td>
</tr>
<tr>
<td>mass assay</td>
<td>2–3</td>
<td>30–60</td>
<td>10–18</td>
<td>3–4 days</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>1–3</td>
<td>30–60</td>
<td>4–12</td>
<td>12 hr</td>
</tr>
<tr>
<td>Troponin T or I</td>
<td>3–4</td>
<td>30–60</td>
<td>4–24</td>
<td>1–3 wk</td>
</tr>
</tbody>
</table>
Medical management for MI

M.I can treat by open the blocked artery & restore blood flow to the affected area of the heart muscle by:

Reperfusion:

✓ Established in the first 4-6 hrs of heart attack by using (percutaneous transluminal coronary angioplasty (PTCA))

✓ Thrombolytic medication
Pharmacological Treatment

Thrombolytic agents:

- The purpose of thrombolytics
  - Dissolve and lyse the thrombus in a coronary artery (thrombolysis)
  - Allowing blood to flow through the coronary artery again (reperfusion)
  - Minimizing the size of the infarction
  - Preserving ventricular function
Antiplatelets

Aspirin to reduce tendency of platelets in the blood to clump & clot

Nitroglycerine:

Vasodilator

Analgesics:

The analgesic of choice for acute MI is morphine sulfate
Potential Complications

- Acute pulmonary edema
- Heart failure
- Cardiogenic shock
- Dysrhythmias and cardiac arrest
- Pericardial effusion and cardiac tamponade
- Myocardial rupture
A systemic assessment include careful history, particularly as it related to symptom

Each symptom must be evaluated with regard to

- **Time**
- **Duration**
- **Factors that precipitate the symptom**
- **Factor that elieve it**
NURSING DIAGNOSES

1. Ineffective cardiopulmonary perfusion related to reduced coronary blood flow

2. Potential impaired gas exchange related to fluid overload from L.V

3. Potential altered peripheral tissue perfusion related to decreased cardiac output

4. Anxiety related to fear of death
Planning and Goals

The major goals of the Patient include:

1. Relief the pain
2. Prevention of further myocardial damage
3. Absence of respiratory dysfunction
4. Reducing anxiety
5. Absence of or recognition of complications
6. Maintenance or attainment of adequate tissue Perfusion by decreasing the heart’s workload
Nursing Interventions

RELIEVING PAIN AND OTHER SIGNS AND SYMPTOMS OF ISCHEMIA:

Administration of oxygen
Elevate head of the bed: is beneficial for the following reasons:

- Tidal volume improves because of reduced pressure from abdominal contents on the diaphragm and better lung expansion and gas exchange

- Drainage of the upper lung lobes improves

- Venous return to the heart (preload) decreases, which reduces the work of the heart.
Improve Respiratory Function:

- Changing position
- Deep breathing

Promoting Adequate Tissue Perfusion

- Checking skin temperature and peripheral pulses frequently is important to ensure adequate tissue perfusion
- Oxygen may be administered to enrich the supply of circulating oxygen
Reducing Anxiety

✔ Developing a trusting and caring relationship with the patient

✔ Providing information to the patient and family

✔ Ensuring a quiet environment, preventing interruptions that disturb sleep

MONITORING AND MANAGING POTENTIAL COMPLICATIONS
Invasive Coronary Artery Procedures

Percutaneous Transluminal Coronary Angioplasty (PTCA): uses:

In patient who do not experience angina but are at high risk for a cardiac event as identified by noninvasive testing

In patient with recurrent chest pain that is unresponsive to medical therapy

In patient with a significant amount of myocardium at risk but are poor surgical candidates

In patient with an acute MI (as an alternate to thrombolysis and after thrombolysis)