Angina (Angina Pectoris) WHAT WENT WRONG?

Angina Pectoris

- A narrowing of blood vessels to the coronary artery, secondary to arteriosclerosis
  - Results in inadequate blood flow through blood vessels of the heart muscle, causing chest pain.
- Precipitated by physical activity, excitement, or emotional stress.
- Atherosclerotic occurs when there is a buildup of plaque within the coronary arteries.
- Angina is often the first symptom that heart disease exists.
- When the demand for oxygen by the heart muscle exceeds the available supply, chest pain occurs.

Three categories of angina

- *Stable angina*—pain is relieved by rest or nitrates and symptoms are consistent
- *Unstable angina*—pain occurs at rest; is of new onset; is of increasing intensity, force, or duration; isn't relieved by rest; and is slow to subside in response to nitroglycerin.
- *Prinzmetal's or vasospastic angina*—usually occurs at rest or with minimal formal exercise or exertion; often occurs at night.

HALLMARK SIGNS AND SYMPTOMS

- Chest pain lasting 3 to 5 minutes—not all patients get substernal pain; it may be described as pressure, heaviness, squeezing, or tightness. Use the patient's words.
- Can occur at rest or after exertion, excitement, or exposure to cold—due to increased oxygen demands or vasospasm.
- Usually relieved by rest—a chance to re-establish oxygen needs

HALLMARK SIGNS AND SYMPTOMS..

- Pain may radiate to other parts of the body such as the jaw, back, or arms— angina pain is not always felt in the chest. Ask if the patient has had similar pain in the past.
- Sweating (diaphoresis)—increased work of body to meet basic physiologic needs; anxiety.
- Tachycardia—heart pumping faster trying to meet oxygen needs as anxiety increases.
- Difficulty breathing, shortness of breath (dyspnea)—increased heart rate increases respiratory rate and increases oxygenation.
- Anxiety—not getting enough oxygen to heart muscle, the patient becomes nervous.

INTERPRETING TEST RESULTS

- Electrocardiogram during episode:
- T-wave inverted with initial ischemia, which is reduced blood flow due to an obstructed vessel, usually first sign.
- ST-segment changes occur with injury to the myocardium (heart muscle).
- Abnormal Q-waves due to infarction of myocardium.
- Labs: troponins, CK-MB, which is an enzyme released by damaged cardiac tissue 2 to 6 hours following an infarction, electrolytes.
- Chest x-ray to determine signs of heart failure.

INTERPRETING TEST RESULTS…

- Coronary arteriography to determine plaque build-up in coronary arteries.
- Cardiac PET (positron emission tomography) to determine plaque build-up in coronary arteries.
- Stress testing to determine symptoms when at exercise or under pharmacologic stress.
- Echocardiogram or stress-echo to determine any abnormality of wall motion due to ischemia.

- Cardiology consult.
- Nonemergent labs: Complete Blood Count (CBC) used to determine the general health status of the patient, chemistry (provides information about the status of eletrolytes, kidneys, acid/base balance, blood sugar and calcium levels), Prothrombin Time (PT/INR), Activated Partial Throboplastin Time(PTT) (helps to detect and diagnose bleeding disorders and the effectiveness of anticoagulants
- Cholesterol panel to evaluate risk

TREATMENT

- The goal of treatment is to deliver sufficient oxygen to the heart muscle to meet its need.
- When suspecting chest pain, always give oxygen as the first line of defense.
- Medications are used initially to treat symptoms and increase blood flow to the heart muscle.
- Medications are used for symptom control and cholesterol management in the long term.
- Cardiovascular interventions are used to maintain adequate blood flow through the coronary arteries.
- Administer beta-adrenergic blocker—this class has a cardioprotective effect, decreasing cardiac workload and likelihood of arrhythmia.
- Drugs like propranolol, nadolol, atenolol, metoprolol.

- Aspirin for antiplatelet effect.
- Analgesic—typically morphine intravenously during acute pain. The medicine is very fast-acting when given this way and will decrease myocardial oxygen demand as well as decrease pain.
- Administer nitrates—aids in getting oxygenated blood to heart muscle

NURSING INTERVENTION

- Monitor vital signs—look for change in BP, P, R; irregular pulse; pulse deficit; when a discrepancy is found between an atrial rate and a radial rate, when measured simultaneously; pulse oximetry.
- Notify physician if systolic blood pressure is less than 90 mmHg.
- Nitrates dilate arteries to the heart and increase blood flow.
- You may have an order to hold nitrates if SBP <90 mmHg to reduce risk of patient passing out from lack of blood flow to brain.

NURSING INTERVENTION…

- Notify physician if heart rate is less than 60 beats per minute.
- Beta-adrenergic blockers slow conduction through the AV node and reduce the heart rate and contractility.
- You may have an order to hold beta blockers if heart rate goes below 60; you should continuously monitor the patient's pulse rate.
- Assess chest pain each time the patient reports it.
- Remember PQRST (an acronym for a method of pain assessment) as follows
- Determine the ***p****lace,* ***q****uality (describe the pain—stabbing, squeezing, etc.),* ***r****adiation (does the pain travel anywhere else?),* ***s****everity (on a scale of 1 to 10), and* ***t****iming (when it started and how long it lasts and what preceded the pain).*

Myocardial Infarction (MI)

- **WHAT WENT WRONG?**

Myocardial Infarction (MI)

- Blood supply to the myocardium is interrupted for a prolonged time due to the blockage of coronary arteries.
- This results in insufficient oxygen reaching cardiac muscle, causing cardiac muscles to die (necrosis).
- MI is commonly known as a heart attack.
- The area of infarction is often due to build-up of plaque over time (atherosclerosis).
- It may also be due to a clot that develops in association with the atherosclerosis within the vessel.
- Patients are typically (not always) symptomatic, but some patients will not be aware of the event; they will have what is called a silent MI.

HALLMARK SIGNS AND SYMPTOMS

- Chest pain that is unrelieved by rest or nitroglycerin, unlike angina
- Pain that radiates to arms, jaw, back and/or neck
- Shortness of breath, especially in the elderly or women
- Nausea or vomiting possible
- May be asymptomatic, known as a *silent MI*, which is more common in diabetic patients
- Heart rate >100 (tachycardia) because of sympathetic stimulation, pain, or low cardiac output
- Variable blood pressure
- Anxiety
- Restlessness
- Feeling of impending doom
- Pale, cool, clammy skin; sweating (diaphoresis)
- Sudden death due to arrhythmia usually occurs within first hour

INTERPRETING TEST RESULTS…

EKG.

- T-wave inversion—sign of ischemia.
- ST-segment elevated or depressed—sign of injury.
- Significant Q-waves—sign of infarction.
- Decreased pulse pressure because of diminished cardiac output.
- Increased white blood count (WBC) due to inflammatory response to injury.
- Blood chemistry:
- Elevated creatine kinase MB (CK-MB)—usually done serially, the numbers will rise along a predetermined curve to signify myocardial damage and resolution.
- Elevated troponin I- and troponin T-proteins elevated within one hour of myocardial damage.
- Less than 25 ml/hr of urine output due to lack of renal blood flow.

TREATMENT

- Treatment is focused on reversing and preventing further damage to the myocardium.
- Thrombolytic therapy is instrumental in reducing mortality.
- A three-hour time window is ideal for maximizing benefit.
- Medications are used to enhance blood flow to the heart muscle while reducing the workload of the heart.
- Supplemental oxygen is used to help meet myocardial oxygen demand.
- Data from coronary angioplasty and percutaneous coronary intervention (stenting) of an occluded artery have been impressive.
- Following the acute management, the patient will have to make lifestyle changes—altering diet and exercise, stopping smoking, and so on.

- Administer oxygen, aspirin.
- Administer antiarrhythmics because arrhythmias are common as are conduction disturbances.
  - Amiodarone.
  - Lidocaine.
  - Procainamide.
- Electrical cardioversion for unstable ventricular tachycardia.
- In cardioversion, an initial shock is administered to the heart to re-establish sinus rhythm

- Administer antihypertensive to keep blood pressure low.
- Hydralazine.
- Percutaneous revascularization.
- Administer thrombolytic therapy within 3 to 12 hours of onset because it can re-establish blood flow in an occluded artery, reduce mortality, and halt the size of the infarction

- Administer analgesics to relieve pain, reduce pulmonary congestion, and decrease myocardial oxygen consumption.
- Morphine.
- Administer nitrates to reduce ischemic pain by dilation of blood vessels; helps to lower BP.
- Nitroglycerin.
- Place patient on bed rest
- Low-fat, low-caloric, low-cholesterol diet.

NURSING INTERVENTION

Monitor:

- Cardiovascular—look for changes or instability in pulse, heart sounds, murmur.
- Respiration—look for changes, fluid in lung fields, shortness of breath.
- EKG continuous monitoring for arrhythmias.
- Vital signs—check for changes in BP, pulse quality, peripheral pulses.
- Pulse-oximetry monitoring.
- Explain to the patient:
- Change to a low-fat, low-cholesterol, low-sodium diet.
- The difference between angina pain and myocardial infarction pain.
- When to take nitroglycerin.
- Medication.

PROGNOSIS

- The outcome depends on the coronary artery that is affected.
- The earlier the person enters the healthcare system, the better the prognosis is, because emergency measures will be available for otherwise fatal arrhythmias

- ***Coronary Artery Disease (CAD)***

WHAT WENT WRONG?

- Cholesterol, calcium and other elements carried by the blood are deposited on the wall of the coronary artery resulting in the narrowing of the artery and the reduction of blood flow through the vessel.
- This impedes blood supply to the heart muscle.
- These deposits start out as fatty streaks and eventually develop into plaque that inhibits blood flow through the artery.
- Elevated cholesterol levels and fat intake can contribute to this plaque build-up, as can hypertension, diabetes, and smoking.
- When the plaque builds up within the artery, the heart muscle is deprived of oxygen and nutrients ultimately damaging the heart muscle.

HALLMARK SIGNS AND SYMPTOMS

- Asymptomatic.
- Chest pain (angina) because of decreased blood flow to heart muscle and/or increase in myocardial oxygen demand resulting from stress.
- Pain may radiate to the arms, back, and jaw.
- Chest pain occurs after exertion, excitement, or when the patient is exposed to cold temperatures because there is an increase in blood flow throughout the body, raising the rate.
- Chest pain lasts between 3 to 5 minutes.
- Chest pain can occur when the patient is resting

INTERPRETING TEST RESULTS

Blood chemistry:

- Increased total cholesterol.
- Decreased high-density lipoproteins (HDL)—helps with reverse transport of cholesterol.
- Increased low-density lipoproteins (LDL).
- Electrocardiogram during chest pain:
- T-wave inversion—sign of ischemia.
- ST-segment depressed—sign of injury to muscle.
- The waves are depressed because of tissue injury.

TREATMENT

- Treatment consists of risk factor modification, life style changes, medications, and revascularization

NURSING INTERVENTION

- Monitor vital signs—signs of hypertension, irregular heart rate
- Monitor electrocardiogram—look for end organ damage, signs of heart disease
- Monitor labs—periodic lipid panel, liver function for patients on statins
- Monitor for myalgias (muscle aches)
- Explain to the patient:
- Stop smoking
- Reduce alcohol consumption
- Change to a lower-fat, lower-cholesterol diet, as well as increased dietary fiber intake
- Increase daily activity
- Weight reduction

- ***Cardiac Tamponade***

WHAT WENT WRONG?

- A large amount of liquid accumulates in the sack around the heart (pericardium), creating pressure on the heart that reduces the filling of ventricles with blood.
- This results in a low volume of blood being pumped with each contraction.
- The accumulating pressure within the pericardium may be due to fluid, pus, or blood.
- The end result is decreased stroke volume and cardiac output.
- The cause of tamponade may be trauma, postoperative, post-MI, uremia, or cancer.
- The fluid may develop rapidly or over time, depending on cause.
- Tamponade is a life-threatening condition. The seriousness is related to the amount of pressure within the heart and the resulting decrease in ventricular filling.

HALLMARK SIGNS AND SYMPTOMS

- Neck vein distention—accumulation of fluid within the pericardium causes pressure on the heart, which prevents the venous return from the jugular veins. This causes distention, more pronounced on inspiration.
- Restlessness due to decreased oxygen to the brain.
- Muffled (dull) heart sounds on auscultation because it’s harder to hear through fluid.
- Pulsus paradoxus—decrease of 10 mmHg or more in SBP during inspiration— change in pressure within the chest during inspiration, resulting in decreased ventricular filling, decreased output, fall in SBP.
- Sweating (diaphoresis).
- Difficulty breathing (dyspnea).
- Tachycardia.
- Hypotension.
- Fatigue.

INTERPRETING TEST RESULTS

- Echocardiograph: Ultrasound image of the heart to assess the heart’s position, structure, and motion.
- Ventricle and atria are compressed. Fluid found within pericardial sac.
- Cardiac catheterization.
- Chest x-ray shows an enlarged heart if large effusion present.
- Electrocardiogram used to rule out other cardiac

TREATMENT

- Treatment is directed at reducing the pressure on the heart from the accumulating fluids in the pericardial sac.
- The following may be necessary to support and stabilize the patient.
- *Pericardiocentesis*: Aneedle is inserted into the pericardium and fluid is aspirated or drained.
- • Administer adrenergic agent—increases heart rate and blood pressure.