

Care of the Pt with Coronary Artery Disease

Epidemiology

CAD affects 13.2 million people in the US and causes nearly 700K deaths each year. Native Americans have the highest prevalence, and people with educational levels below a high school diploma have the highest rate of CAD. Regionally, the rate is highest in the South and lowest in the awesome West. It is THE leading cause of death for all U.S. ethnic groups, EXCEPT for Asian females. The highest incidence is white males age 45+.

Pathophysiology

The most common cause of CAD is atherosclerosis. Recall that this is when a plaque forms on the artery wall (the plaque is called an atheroma). It is precipitated by lipoproteins and fibrous tissue...also inflammation and injury to the endothelial cells lining the artery make it prone to atheromas. Inflammation and injury are caused by excessive pressure (HTN), toxins in cigarette smoke, infections and hyperlipidemia. Note that low-dose aspirin can decrease inflammation along the endothelial lining, so that explains why so many pts are on aspirin! The lesions or plaques can ulcerate or rupture leading to clots that expand the size of the blockage...this is an "acute thrombosis". The atheromas tend to build up in bifurcated arteries (there are areas of high pressure at the bifurcation)...main arteries involved are carotid, coronary and renal.

Myocardial Ischemia occurs when oxygen supply is inadequate to meet metabolic demands.

There are three determinants for this:

- 1) myocardial workload: things that increase myocardial workload are increased afterload (HTN), increased O₂ demands, pain, ischemia, tachycardia (decreased filling times), increased preload (CHF and low EF), increased contractility (digitalis or increased metabolism), increased metabolic demands (fever, infection, hyperthyroidism)
- 2) coronary perfusion: things that affect coronary perfusion would be a thrombosis, atheroma, constriction of artery, atherosclerosis, vasospasm (r/t nicotine, caffeine, meth, cocaine), and poor perfusion pressure (low preload, hypotension, dehydration, hypertrophy).
- 3) blood oxygen content: things that cause this are reduced atmospheric oxygen content (CO₂ poisoning, or high altitude), impaired gas exchange (CHF, pneumonia, PE), or low RBC and Hgb content (anemia, blood loss). Note that if you have an elderly pt with blood loss, the risk for MI ↑↑↑↑.

Recall that the Coronary Arteries perfuse during diastole!

Categories of CAD

- Chronic ischemic heart disease
 - stable angina (predictable, often happens with activity or between 4-6 am and around 10am.
 - vasospastic angina (very short duration)
 - silent myocardial ischemia. Women tend to get the silent version more than men. A woman may not feel pain with a silent ischemia, but will say "I just don't feel right", "I'm tired", "My arms are heavy", etc...
- Acute coronary syndrome
 - unstable angina (unexpected, happens in a low-stress environment. This would be angina that lasts more than 20 minutes and does not respond to nitro or rest. It will lead to an MI.
 - myocardial infarction

Risk factors for CAD

These are non-modifiable and modifiable. The non-modifiable ones are age (men > 45 and women > 55), gender (men > women...until women hit menopause, then I guess it's equal?), genetic factors (male first degree relative younger than 55 or female first degree relative younger than 65.) Modifiable risk factors are hyperlipidemia, hypertension, diabetes, smoking, obesity, sedentary lifestyle and atherogenic diet.

Smoking

The MOST modifiable of this is to STOP SMOKING! Cigarette smoking is the LEADING independent risk factor for CAD. It is responsible for more CAD-related deaths than lung cancer or COPD. The effects of cigarette smoking are dose dependent, so it is important to know how much your pt smokes. Smoking contributes to CAD by damaging the endothelium leading to atheromas, stimulating catecholamine release, constricting the arteries and limiting tissue perfusion, reducing HDL levels. Quitting smoking reduces your risk of developing CAD by 50%!!!

Hyperlipidemia

Low Density Lipoproteins are the primary carriers of cholesterol. Just remember that LDL = lethal! The presence of LDLs promotes atherosclerosis, so you want to have your levels below 100 mg/dL. High Density Lipoproteins help clear cholesterol from the arteries and thus have a protective effect. You want the HDLs to be above 35 mg/dL and you can increase them by exercising :-). Hormone therapy can also increase HDLs, but it has its own mess of consequences.

Diabetes Mellitus

DM is associated with higher lipid levels, a higher incidence of HTN, and is often associated with obesity. DM affects the endothelium which contributes to the process of atherosclerosis...and this is why diabetics are always on low-dose aspirin!

Hypertension

HTN = a consistent BP > 140 systolic or 90 diastolic. It affects more than 1/3 of people over the age of 50 in the U.S. HTN is more common in African Americans, Hispanics and Whites (in that order). It also damages endothelial cells and stimulates atherosclerotic plaque formation.

Diagnosing CAD

- Cholesterol levels. Desirable is a total cholesterol < 200, with a 1:3 ratio of HDL to total cholesterol.
- Risk factor assessment
- C-reactive protein (will be ↑ in inflammation)
- ECG/exercise ECG (stress test)
- Myocardial perfusion imaging (angiography?)
- Cardiac catheterization

Assessing the pt with CAD

- The complaint will be pain in the chest along with accompanying symptoms (N/V, dizziness, palpitations). When assessing pain go through PQRST (precipitate, quality, radiate, site, timing/duration).
- Focused history. Ask about onset, duration, precipitating event, intensity. Also ask about previous events...has this ever happened before? You will also want to identify any risk factors...are they diabetic, do they have high cholesterol, HTN, etc...
- Physical assessment. For the most part the physical assessment will look pretty normal: breath sounds, work of breathing, heart tones, pulses, edema, skin signs.

RED ALERT! Stable vs. Unstable Angina

Stable occurs with a predictable amount of activity or stress.

Unstable occurs with increasing frequency and severity, and it may occur at times unrelated to activity or stress.

Manifestations of Angina

- chest pain
- quality (ask if it is burning, sharp, occurs with respiration, does it hurt to press on chest, does it rise and fall in a crescendo pattern? If it occurs with respiration, I'm pretty sure this means not MI)
- associated manifestations (N/V, sweating, palpitations, dizzy)
- atypical manifestations (pt may complain of heartburn)
- precipitating factors (what else is going on? been sick lately?)
- relieving factors (what makes it better?)

Diagnostics for Angina

- 12-lead EKG (about 80% accurate for MI detection)
 - Resting EKG may be totally normal; may show "non-specific changes in ST segment" or "non-specific changes in T-wave"...this is very vague!; may show acute changes of ischemia (ST elevation or T-wave inversion); may show previous acute MI (Q-wave); may show acute evolving MI...oh boy!
- Echocardiogram: This is a non-invasive test that evaluates cardiac structures and fxn. It may be done at rest or during exercise. A transesophageal echocardiography (TEE) identifies abnormal blood flow patterns as well as structures...it is a more accurate ECHO with less artifact.
- Angiogram...the GOLD STANDARD! Can see blockages! This is an invasive procedure where a catheter guided by fluoroscopy is introduced into the femoral artery and threaded to the coronary artery (sounds awful!). Dye is injected

into the coronary artery which allows for visualization of obstruction or stenosis. A narrowing of a vessel by more than 50% is significant.

Medications for CAD

- Nitrates: long-acting and short-acting; used to treat anginal attacks; usual dose is sublingual nitroglycerin 0.4 mg q 5 minutes x3; common SE are headache; you need an IV line in case of ↓↓↓ BP!
- Beta Blockers: atenolol and metoprolol; block cardiac stimulating effects of epinephrine; note that most BB are cardio-selective, but some can cause bronchospasm.
- Calcium Channel Blockers: reduce myocardial oxygen demand, increase myocardial blood and oxygen supply, potent coronary vasodilators; verapamil and diltiazem

Acute Coronary Syndrome

Acute coronary syndrome is defined as unstable cardiac ischemia, unstable angina (not responding to nitro and also is unpredictable), and acute myocardial ischemia. It is a common cause of admission into the hospital. It is characterized by injury to myocardial cells....it is the MOST COMMON cause of sudden cardiac death!

Pathophysiology of ACS

Coronary blood flow is acutely reduced, the artery is not fully occluded (but is > 70% occluded), and myocardial cells are injured or ischemic (but not infarcted/dead). It is precipitated by the following:

- rupture or erosion of atherosclerotic plaque
- formulation of a blood clot that partially occludes a vessel (platelet aggregation)
- coronary vasospasm (stress, smoking, drugs)
- progressive vessel obstruction by atherosclerotic plaque
- increased myocardial oxygen demand or decreased supply
- plaque can rupture d/t change in hemodynamics (ex: increased BP)

When a Plaque Ruptures

- The exposed lipid core of the plaque attracts platelets and stimulates platelet aggregation
- Extrinsic clotting pathway is stimulated
- Thrombin is generated
- Fibrin is deposited
- Formation of a clot severely impairs or obstructs blood flow...the clot can grow quickly!!!
- Myocardial cells become ischemic leading to changes on the EKG (ST elevation and inverted T waves)
 - Recall that ST elevation = injury and T wave inversion = ischemia
- IF THE LEFT MAIN CORONARY ARTERY IS blocked...this is a "TIME BOMB"...the whole left ventricle will not get perfused and your patient will probably die. Not good news!
- The ECG on the right represents changes in the reading related to ACS

Manifestations of ACS

- chest pain
- dyspnea
- diaphoresis
- pallor
- cool, moist skin
- tachycardia
- hypotension b/c losing contractile force. (around 90-100 SBP)

Diagnosis of ACS

- 12-lead EKG: may be normal or may show non-specific changes, or may show ischemic patterns.
- Cardiac markers may be normal or slightly elevated
- Stat portable CXR
- **Note that a younger pt will not have collateral circulation built up → higher risk for death!

Treatment for ACS: MONA!

- M = morphine (do this **THIRD** if pt still having pain)
 - reduces myocardial ischemia
- O = oxygen (do this **FIRST!**)
 - reduces myocardial ischemia
- N = nitro (do nitro and aspirin **SECOND**)
 - restores blood flow to ischemic myocardium; can also use beta-blockers
- A = aspirin
 - reduces risk of blood clotting; can also use heparin and antiplatelet drugs aspirin, Integrelin

More therapies include revascularization procedures:

- PTCA: a balloon tipped catheter is positioned across the area of narrowing and inflated, compressing the plaque against the wall of the vessel.
- PCI or PCR: a broad term meaning to thread a catheter (12 Fr) into the plaque and then do something to open the vessel like stenting or angioplasty.
- Stenting: inserting a metal scaffold used to maintain an open lumen. Reduce the rate of restenosis (stent has meds on it?...I think that's what she said)
- CABG: coronary artery bypass grafting...much bigger procedure than the others

GOALS FOR ACS Tx
 Reduce risk of blood clotting
 Reduce myocardial ischemia
 Restore blood flow to ischemic myocardium

Nursing care after PCR

- Prevent complications. These are things like hematoma (keep HOB < 30 or even as low as < 15 to prevent constriction on the femoral artery), pseudoaneurism, embolism, hypersensitivity to contrast dye (can lead to contrast induced nephropathy so you need your pt to drink fluids to flush the dye out), dysrhythmias, bleeding at the site (pressure dressing), vessel perforation, restenosis or re-occlusion of vessel (there's a fine line with heparin therapy in regards to controlling clotting but not promoting bleeding).
- Head to toe assessment
- Monitor vitals and cardiac rhythm continuously
- Maintain NTG infusion
- Administer anticoagulants and antiplatelet meds
- Monitor, treat and report chest pain
- Maintain bedrest with HOB at 30 degrees or less
- Monitor distal pulses
- Monitor I/O...want to be sure the kidneys are working!

Coronary Artery Bypass Grafting (CABG)

This procedure uses a section of vein or artery to create a connection between the aorta and the coronary artery beyond the obstruction. Commonly used veins are the internal mammary artery and the saphenous vein. In 90% of cases angina is relieved or reduced, and the procedure is recommended for people with multiple vessel disease, impaired LV function and diabetics.

Nursing Care of CABG Pt

- Pre-op patient education should include: cardiac recovery unit, tubes, drains, general appearance, monitoring equipment to be used, respiratory support, endotracheal tube, suctioning and communication, incisions, dressings, pain management.
- Decreased cardiac output
- Hypothermia
- Acute pain
- Ineffective airway clearance
- Impaired gas exchange
- Risk for infection

Complications of CABG

- Cardiac tamponade...this is when blood gets into the pericardial sac. S/S are: tachycardia, hypotension, decreased urine output, increased CVP (normal range is 4-10 mm Hg), JVD, muffled heart tones
- Perioperative MI. S/S are: anginal or cardiac pain, tachycardia, hypotension
- Fluid/electrolyte balance
- Malnutrition
- Stroke
- Renal failure

Chest tube out in 1-2 days
Out of bed next morning
Home in 3-4 days

Acute Myocardial Infarction

Acute MI involves the necrosis or death of myocardial cells. You will see this as Q-waves on the EKG. 700,000 people experience an AMI in the U.S. each year and 500,000 of those go on to experience another MI. People rarely survive their third MI, and 60% of deaths occur within 1 hour after symptoms begin. It is caused by unstable or complicated lesions...these are plaque-like lesions that are prone to rupture and thrombus formation. A stable lesion is likely to cause angina.

Acute MI Pathophysiology

The MI occurs when blood flow to a portion of the cardiac muscle is completely blocked → prolonged tissue ischemia → irreversible cell damage. Ischemia lasting more than 20 - 45 minutes → cellular death and tissue necrosis. Cellular acidosis, electrolyte imbalances and hormones released in response to cellular ischemia affect impulse conduction and myocardial contractility. The risk for dysrhythmias increases and myocardial contractility decreases → ↓ stroke volume, ↓ CO, and ↓ BP and ↓ tissue perfusion. The subendocardium suffers the initial damage (within 20 mins) b/c this area is most susceptible to changes in coronary blood flow. If blood flow is restored at this time, the infarction is limited to this subendocardial tissue. If not, damage progresses to the epicardium within 1-6 hours. When all layers of the myocardium are affected, it is known as a transmural infarction. This is why there is a 30-minute door to needle timeframe....you must act fast!

Collateral Vessels

When a large artery is compromised, collateral vessels connecting smaller arteries in the coronary system dilate to maintain blood flow in the cardiac muscle. The degree of collateral circulation helps determine the extent of the myocardial damage from ischemia. Acute occlusion of a coronary artery without any collateral flow results in massive tissue damage and possible death.

Coronary Arteries

The coronary artery that is occluded determines the area of damage. Myocardial infarction usually affects the left ventricle because it is the major "workhorse" of the heart...its muscle mass is greater as are its oxygen demands.

- Occlusion of the LAD artery affects blood flow to the anterior wall of the ventricle, thus one has an anterior wall MI.
- Occlusion of the left circumflex artery causes a lateral MI.
- Right ventricular inferior and posterior infarcts involve occlusion of the right coronary artery and posterior descending artery.
- Occlusion of the left main coronary artery is the most devastating, causing ischemia of the entire left ventricle and a grave prognosis.

Cocaine Intoxication and MI

Cocaine intoxication causes increased SNS activity by increasing the release of catecholamines from central peripheral stores AND interfering with the reuptake of catecholamines. The increased concentration of catecholamines causes increases in the heart rate and contractility. It also increases automaticity of cardiac tissues and the risk of dysrhythmias. Additionally, there is vasoconstriction and hypertension. This client may present with ALOC, confusion, restlessness, seizure activity, tachycardia, hypotension, increased RR and respiratory crackles. Methamphetamine can cause the same issue.

Manifestations of Acute MI

- PAIN...the classic manifestation: more severe than angina, continuous (no crescendo affect), sudden onset and usually not associated with activity, described as crushing/pressure/heavy/squeezing, located substernal and radiating, lasts more than 20 minutes and is not relieved by nitro or rest.
 - Women and older adults often experience atypical chest pain presenting with complaints of indigestion, heartburn, N/V.
 - Up to 25% of pt with acute MI deny chest discomfort...this is a "silent MI". Sometimes you find these years later on an EKG.
- Compensatory mechanisms cause OTHER symptoms
 - anxiety
 - tachycardia
 - vasoconstriction
 - cool, clammy, mottled skin
 - diaphoresis
 - tachypnea (28-30 bpm)
 - sense of impending doom
 - increased WBC and temp

Complications of MI

- Dysrhythmias
 - Dysrhythmias or irregularities of heart rhythm are the most frequent complication of MI. Ischemic, injured and infarcted tissue is arrhythmogenic. It affects the generation and conduction of electrical impulses. The risk of ventricular fibrillation is greatest the first hour after AMI. It is a frequent cause of sudden cardiac death. If the infarct affects a conduction pathway, electrical conduction may be affected. Any degree of atrioventricular AV block may occur following MI, especially when the anterior wall is infarcted.
- Pump failure
 - MI reduces myocardial contractility, ventricular wall motion, and compliance. Impaired contractility and filling may produce heart failure. The greatest risk of heart failure occurs when large portions of the left ventricle are infarcted. May occur more often with an anterior MI. Loss of 20 – 30% of the left ventricular muscle mass may cause manifestations of left sided heart failure (dyspnea, fatigue, weakness, respiratory crackles)
- Cardiogenic shock
 - Occurs as a result of impaired tissue perfusion due to pump failure. Functioning myocardial muscle mass decreases by more than 40%. The heart is unable to pump enough blood to meet the needs of the body and maintain organ function. Low cardiac output due to cardiogenic shock also impairs perfusion of the coronary arteries and myocardium, further increasing tissue damage. Mortality from cardiogenic shock is greater than 70%.
- Infarct extension
 - approximately 10% of clients experience extension or reinfarction in the area of the original infarction during the first 10 to 14 days after an MI.
- Structural defects
 - Necrotic muscle is replaced by scar tissue that is thinner than the ventricular muscle mass. This can lead to such complications as ventricular aneurysm, rupture of the interventricular septum or papillary muscle and myocardial rupture.
- Pericarditis/Dressler's syndrome
 - Tissue necrosis prompts an inflammatory response. Inflammation of the pericardial tissue surrounding the heart, may complicate AMI, usually within 2 – 3 days. Pericarditis causes chest pain that may be aching or sharp and stabbing, aggravated by movement or deep breathing. A pericardial friction rub may be heard on auscultation of heart sounds
 - Dressler's syndrome is thought to be a hypersensitivity response to necrotic tissue or an autoimmune disorder, may develop days to weeks after AMI. It is a symptom complex characterized by fever, chest pain, and dyspnea. Dressler's syndrome may spontaneously resolve or recur over several months causing significant discomfort and distress.

Nursing Care for Acute MI

- Relieve chest pain. This is vital. If you fix the pain you fix the problem. Pain stimulates the SNS which increases HR and BP → increased cardiac workload. Sublingual nitro may be given up to 3 doses at 5 minute intervals. IV nitro may be continued for the first 24-48 hours to reduce myocardial work. Nitro decreases myocardial oxygen demand and may increase the supply of oxygen to the myocardium. It dilates coronary arteries and collateral channels in the heart increasing blood flow to save myocardial tissue at risk. Close monitoring is necessary to assess for hypotension and tachycardia. Morphine is also a drug of choice for chest pain unrelieved by nitro and also for sedation. NOTE that many pts will wait up to 4 hours after chest pain starts to seek care!!!
 - Nitroglycerine 0.4 mg q 5 mins x 3
 - Nitroglycerine drip 10 mcg/hour
 - Morphine sulfate 4-8 mg IV (usually give 2 mg at a time and titrate)
- Reduce the extent of myocardial damage. Time is muscle...there is a 1-hour time frame!
 - ASA as a platelet inhibitor
 - Fibrinolytic agents dissolve or break up clots, activate the fibrinolytic system, restore blood flow to the obstructed artery. These are given within 6 hours of MI onset and there are many complications (bleeding related). So, people with known bleeding disorder, cerebrovascular disease, uncontrolled HTN, pregnancy or recent trauma/surgery are NOT candidates for this therapy.
- Maintain CV stability
 - Monitor for dysrhythmias
 - Lidocaine given for ectopy, v-tach/v-gib (toxicity is an issue). 1.5 mg/kg IVP
 - Amiodarone for v-fib/v-tach...the drug of choice for dysrhythmias! 300 mg IVP, followed by 150 mg IVP
 - Atropine for bradycardia and AV blocks...it speeds things up!
 - Epinephrine 1:10,000 solution; 1 mg in 10 mls IVP for v-fib/asystole/PEA
- Decrease cardiac workload
 - Monitor BP and MAP
 - Beta blockers decrease heart rate, reduce cardiac work and reduce myocardial oxygen demands
 - ACE inhibitors reduce myocardial remodeling (occurs with chronic HTN) and reduces incidence of heart failure
 - Anticoagulants maintain patency of coronary arteries
- Prevent complications
- Nursing Interventions
 - Monitor continuously
 - Maintain patency of peripheral IVs for emergency meds
 - Oxygen administration
 - Bed rest x 12 hours
 - Quiet/calm environment
 - Liquid diet for 4-12 hours, then low fat, reduced sodium diet
 - No caffeine, avoid very hot and cold foods
 - Monitor for signs of reperfusion...decreased chest pain, return of ST segment to baseline, reperfusion syndrome
 - Therapeutic time, teaching
- Manage Complications
 - Pump failure (leads to CHF).
 - Intra-aortic balloon pump provides mechanical circulatory support. It is positioned in the descending aorta and it inflates on diastole, deflates on systole. It augments cardiac output by 10-15%.
 - Ventricular assist devices take complete control of cardiac function. They are used in cardiogenic shock and as a bridge to heart transplant. These devices have a high risk of infection!

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