

Inflammatory Heart Disease

All inflammatory heart disorders have the propensity to lead to heart failure, so this is a very serious condition!

Definition: Any layer of cardiac tissue can become inflamed (Endocardium = Endocarditis, Myocardium = Myocarditis, Pericardium = Pericarditis). The damage can occur to the valves, muscle or pericardial lining. It can be mild or it can be life threatening!

What causes carditis? **Rheumatic Heart Disease**...aka Rheumatic Fever!

Rheumatic Heart Disease is a systemic inflammatory disease, that manifests as a heart problem. It is d/t an abnormal immune response to pharyngeal infections. It is caused by group A beta hemolytic strep (GABHS, or "strep throat"), and damages to the heart valves. The good news is, it is becoming rare in industrialized nations (probably b/c we have antibiotics), and the peak incidence is 5-15 years of age. 3% of people who have untreated GABHS develop rheumatic fever. Of those about 50% develop carditis.

Strep Throat

Looks like the flu, but there is NO COUGH. Voice may be muffled d/t sore throat!

Risk factors for Rheumatic Heart Disease

- Crowded living conditions
- Malnutrition
- Immunodeficiency
- Poor access to health care
- Possibility of a genetic factor in susceptibility

Pathophysiology of Rheumatic Heart Disease

A type of proteins on the GABHS bind to cells in the heart, muscles and brain → immune response leads to inflammation of the tissues (when the joints get involved you have Rheumatoid Arthritis) → Lesions develop in connective tissues at the heart, joints and skin. As far as the heart goes, the valve structures are affected and have small vegetative lesions on the valve leaflets. To make things even worse, this usually involves all three layers of the heart. As the inflammatory process resolves, fibrous scarring occurs, causing deformity of the heart. Bad news!

This disease is slowly progressive over years and years. The valvular deformity may follow acute or recurrent attacks of rheumatic fever. Eventually the valve leaflets become rigid and deformed, and the chordae tendoneae become fibrous and shorten. → stenosis and regurgitation (valve diseases)

Stenosis = valve gets stiff very very small with just a tiny opening for the blood to flow through. Sometimes the valves are fused together. This causes an obstruction to forward blood flow.

Regurgitation = occurs when the valve fails to close properly (becomes incompetent and "floppy") and allows blood to flow backward through the valve. The valves on the left side of the heart are usually the most affected (mitral valve and the aortic valve.)

Manifestations of Rheumatic Fever

2-3 weeks after the strep infection you'll have...

- a new fever,
- migratory joint pain initially (red, warm, swollen, tenderness at more than 1 joint...usually large joints of extremities)
- a rash (erythema marginatum...it is red, blotchy, slightly elevated and does NOT itch...located at inner aspect of upper arms, trunk and thighs)
- you could have neuro symptoms but those are rare (irritability, inability to concentrate, clumsiness, muscle spasms)
- cardiac symptoms are chest pain, tachycardia, pericardial friction rub, and evidence of heart failure.

Diagnosis for Rheumatic Fever

- History and physical assessment
- CBC with ESR (ESR will be ↑ d/t inflammation)
- WBC is elevated with > 10% bands (bandemia)

- RBCs are decreased
- C-Reactive protein is positive
- ASO titer tests for strep antibodies
- Throat culture may be positive for strep

Treatment for Rheumatic Fever

- Immediate antibiotics for strep!!! Penicillin, Erythromycin. May continue low doses for 5-7 years. YES, YEARS!!!
- NSAIDs for joint pain
- Corticosteroids for inflammation in joints and heart lining

Nursing Care for Rheumatic Fever

- The first thing you want to do is ID strep pharyngitis ASAP!
 - sore throat, with tonsillar exudate, throat will be cherry red
 - lymphadenopathy (enlarged lymph nodes)
 - fever
 - headache
 - NO cough
- Complete abx...lots of teaching needed here!
- Communicability teaching (strep is contagious!)
- Close monitoring and follow-up
- Accurate assessment

Nursing Dx for Rheumatic Fever

- Acute pain (NSAIDs, and anti-inflammatory drugs...give with food or antacids)
- Activity intolerance b/c pt may develop heart failure
- Risk for fluid/electrolyte imbalance (GABHS can cause glomerular nephritis which leads to kidney failure)
- Risk for altered nutrition
- Risk for altered tissue perfusion

Things you can do!

- warm moist compress for local pain relief (joints)
- report signs of aspirin toxicity (tinnitus, vomiting, GI bleed)
- monitor pt for signs of fatigue, weakness, dyspnea on exertion b/c pt may develop heart failure. Explain to pt the importance of activity limitations and gradually increasing activity.
- look for glomerular nephritis (cola colored urine, fatigue, HTN). If present, restrict sodium.
- Increase calories for healing and to support immune system. High CHO, high protein recommended.
- listen for pericardial friction rub r/t altered tissue perfusion.
- listen for crackles in lungs r/t altered tissue perfusion
- monitor urine output r/t altered tissue perfusion
- monitor BP (high BP is sign of glomerular nephritis)

Types of Carditis (Endocarditis, Myocarditis, Pericarditis)

Infective Endocarditis

This is an inflammation of the endocardium (inner lining of the heart). The valves are usually affected and it is usually infectious (two types...subacute and acute). *Subacute bacterial endocarditis* accounts for 90% of cases. It develops slowly and occurs in pts with previous heart valve damage. *Acute bacterial endocarditis* is rare...it has an abrupt onset and occurs in pts with no previous heart problems.

Incidence/Risk Factors for Infective Endocarditis

- It is relatively uncommon...that's the good news!
- The greatest risk factors are:
 - previous heart damage (often congenital)
 - valve prosthesis

- IV drug abuse is BIG risk factor
- central venous catheters
- indwelling urinary catheters → urosepsis (catheter infection rate is 75% for UTI)
- dental procedures and poor dental health
- recent heart surgery
- Most of these cause problems with the left side of the heart, except for IVDA which affects the right side.

Pathophysiology of Infective Endocarditis

The pathogen enters the blood stream → organisms colonize heart valve vegetations (in acute the lesions develop on healthy valves for unknown reasons, in subacute the lesions develop on damaged valves) → vegetations enlarge as platelets and fibrin are attracted to the site. This forms a covering over the bacteria and keeps the immune system from attacking. Vegetations can be singular or multiple... AND, they can break or shear off → embolus travels through blood stream to other organ systems. Ultimately the vegetations scar and deform the valves causing turbulence of blood flowing through the heart. Heart valve function is affected causing problems in forward blood flow or regurgitation.

Endocarditis is classified by its acuity...acute and subacute

- Acute infective endocarditis (RARE)
 - abrupt onset
 - rapidly progressive
 - severe
 - staphylococcus aureus
- Subacute infective endocarditis
 - gradual onset
 - systemic manifestations
 - streptococcus viridans
 - more likely to occur in the presence of heart disease
 - usually caused by previous strep infection, but can also be caused by enterococci, yeasts and fungi.

Manifestations of infective endocarditis

- fever, chills (if an adult is running a fever, need lab work!)
- flu-like symptoms (generalized malaise, fatigue)
- cough and shortness of breath
- joint pain that migrates
- anorexia/abdominal pain
- heart murmur
- petechiae and splinter hemorrhages
- splenomegaly

Course of the disease (infective endocarditis)

Complications of infective endocarditis can occur...these are embolization, heart failure, abscess, ventricular aneurysms. Without treatment, BE is usually fatal. Antibiotic therapy is usually effective, and from then on that person will always require prophylactic Abx (dental procedures, surgeries, bronchoscopy, cystoscopy, urinary catheterization, infected tissue and maybe even childbirth).

Diagnostics for endocarditis

- blood cultures (+ when typical pathogen ID from two or more separate blood culture)
- echocardiography (can detect vegetation)
- CBC, ESR, chem panel
- CXR and ECG

Treatment for endocarditis

- Antibiotics that are specific for the pathogen...so not the broad-spectrum ones
 - penicillins
 - gentamycin
 - cephalosporins

- vancomycin
- Surgery
 - may be required to replace damaged valves
 - can remove vegetations that are at risk for embolization
 - can remove a valve that does not respond to abx therapy
 - can repair stenotic valve
 - can replace regurgitative valves

Nursing care for endocarditis

The first thing you can do is *promote good health!* Address high risk behaviors and encourage exquisite dental health...your pt is going to be getting cleanings q 3 months and will need to take really good care of his teeth. You will also want to teach your pt to avoid respiratory infections (maybe they need to wear a mask when in crowds), and may also need teaching on anticoagulant therapy if warranted. When *assessing* this pt, you will take a complete health history and ask about any flu-like symptoms, SOB or heart defects. You will also take VS, listen to lung sounds and heart tones, look for petechiae or splinter hemorrhages.

Nursing Diagnosis for endocarditis

- R/F imbalanced body temperature: treat and report fevers, obtain blood cultures as ordered
- R/F ineffective tissue perfusion: assess and document changes in neuro status, renal status, pulmonary and CV function.
- Ineffective health maintenance: pt will need prophylactic abx, and you need to address IVDA if present.
- Knowledge deficit: teach pt to get flu shot, report SOB, report decreases in activity tolerance

Myocarditis

This disease is inflammation of the heart muscle. It usually results from an infectious process (often viral, i.e. coxsackievirus B). **It can also occur from radiation or toxic drugs as in chemotherapy.**

Incidence and Risk Factors for Myocarditis

It is more common in men than women. The risks are:

- malnutrition
- alcohol use
- immunosuppressive drugs
- exposure to radiation
- stress
- complications of rheumatic fever

Pathophysiology of myocarditis

The inflammatory process damages myocardial cells → local or diffuse swelling of the myocardium → abscesses form within the myocardium. It is usually self limiting with supportive care, though it may become chronic and can lead to heart failure. When heart failure occurs contractility decreases → ↓ EF. This disease can take several months to resolve.

Manifestations of myocarditis

- fever, fatigue, general malaise (flu-like)
- dyspnea
- palpitations
- arthralgias
- murmur
- pericardial friction rub
- chest pain

Diagnosis for myocarditis

- ECG will show ST elevation and T-waves changes that are non-specific
- Cardiac markers will be elevated
- Endomyocardial biopsy will differentiate between AMI and myocarditis.

Treatment for myocarditis

- antiviral therapy
- Immunosuppressive therapy
- corticosteroids
- treat heart failure as needed: ACEi, digitalis, anti-arrhythmics (amiodarone), anticoagulants if a-fib present
- possible bedrest during inflammatory process (3-4 weeks), definite activity intolerance/restriction for 6-8 months

Nursing Care for myocarditis

Care is directed at reducing myocardial work load and maintaining cardiac output.

- ECG monitoring
- Activity tolerance
- Perfusion of organs
- Monitor for and report any early signs of heart failure

Nursing Diagnosis for myocarditis

- Activity intolerance
- Decreased cardiac output (monitor pt for any signs of CHF)
- Fatigue
- Anxiety
- Excess fluid volume
- Impaired nutrition

Pericarditis

Pericarditis is more common than both myocarditis and endocarditis combined. It is an inflammation of the pericardium (the sac around the heart), and can be primary or secondary. It is usually due to a viral infection and it affects men more than women. It is seen in 40-50% of pts with end-stage renal disease. It also occurs post-infarction (Dressler's Syndrome) and post-cardiotomy (Postcardiotomy Syndrome). The difference between these two is the type of fluid in the sac.

Causes of pericarditis

- Infections: virus, bacteria, TB, fungi, syphilis, parasites
- Non-infectious: injury to heart, uremia, neoplasms, radiation, autoimmune disorders, rheumatic fever, drugs, trauma/surgery

Pathophysiology of pericarditis

Inflammatory response to tissue damage → vasodilation, hyperemia, edema. The capillaries get permeable and leak fluid and WBCs amass at the site of the injury. Exudate is formed! Fibrosis and scarring of the pericardium may restrict cardiac function (STIFF!), and pericardial effusions may develop. You will hear a pericardial friction rub every time the heart beats, and can obliterate S1 and S2. Have the pt sit up and lean forward so you can hear better...this rub is an expected finding in this disease! Is this "A STIF WALL" we learned in N-16?

Manifestations of pericarditis

- Chest pain that is sharp (as opposed to the dull, crushing pain of an MI), increases with inspiration, and is reduced by sitting upright and leaning forward.
- Pericardial friction rub is heard best at left lower sternal border, have client sit up and lean forward
- Fever (low-grade, around 100)
- Dyspnea
- Tachy...over 120

Course of the disease

Complications of pericarditis are:

- Pericardial effusion: fluid between the pericardial layers that threatens normal cardiac function
- Cardiac tamponade: this is a MEDICAL EMERGENCY! It results from effusion, trauma, rupture of a vessel, hemorrhage. It restricts cardiac function! Manifestations are:

- paradoxical pulse (difference between SBP on inspiration and expiration...drops on inspiration, rises on expiration. Can see on arterial line...the waveform changes)
 - narrowed pulse pressure (difference between SBP and DBP narrows)
 - hypotension with skin sign changes
 - tachycardia
 - weak peripheral pulses
 - distant, muffled heart tones
 - JVD (have pt sit at 30-degrees and look at JV)
 - decreased LOC and lethargic
 - decreased urine output
- Constrictive pericarditis: scar tissue contracts which restricts diastolic filling, can cause ascites

Fluid in the Pericardium

If it collects slow, can hold *up to 1 L*

If it collects quickly, will become restricted with around *100 ml of fluid*

Diagnosis of pericarditis

- CBC shows elevated WBC and ESR
- Cardiac markers slightly elevated
- ECG shows diffuse ST segment elevation in all leads, no Q waves
- Echocardiography may show effusion
- CXR may show cardiac enlargement and may show effusion

Nursing Care of pt with pericarditis

- Get a detailed health history...look for c/o chest pain, SOB, DOE, meds, chronic illnesses such as renal failure, DM and HTN
- Assess: VS, strength of pulses, apical pulse, JVD, heart tones, perfusion

Nursing Diagnoses for pericarditis

- Acute pain (give NSAIDS with food, assess chest pain carefully...it will be CONSTANT!)
- Ineffective breathing pattern (maintain a calm environment, encourage deep breathing use of incentive spirometer. Document respiratory rate, effort and breath sounds every 2 – 4 hours. Report adventitious or diminished breath sounds. Shallow guarded respirations may lead to increased respiratory rate and effort.)
- Risk for decreased cardiac output d/t increased work on heart
- Decreased tissue perfusion (watch urine output, LOC and skin signs)
- Activity intolerance (pace nursing activities)

Disorders of Cardiac Structure: Valve Diseases and Cardiomyopathy

Valvular Disease

Valvular disease occurs as two main disorders: stenosis and regurgitation. In stenosis, the valve leaflets fuse together via vegetation or a congenital defect. This causes the valve opening to narrow and become rigid which impedes forward blood flow ultimately leading to decreased cardiac output. Sometimes the opening can be as narrow as a few millimeters! Imagine how hard your heart has to work to push against that huge resistance...work of the heart goes up and this is never good news for your heart. Regurgitation occurs when the valves do not close completely and this is also usually caused by vegetation...could also be that the heart has grown (which is bad) and pulled the valves out of shape (can also be congenital). This causes blood to flow back and you'll hear a murmur with this.

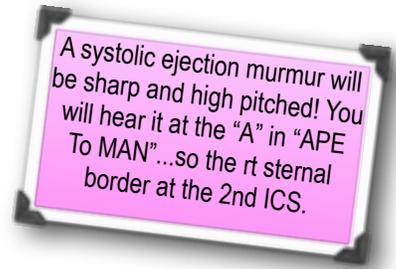
Obviously these diseases are going to cause hemodynamic changes, and the most common of these is decreased cardiac output and all the bad stuff that ensues. It can lead to heart failure or pulmonary complications (so make sure you listen to those lung sounds!). Stenosis increases workload of the chamber behind the valve, and regurgitation causes excess blood volume in chamber behind the valve...got that? Basically, both of these interfere with the smooth flow of blood through the heart (and to think we take it for granted!). The flow becomes turbulent and causes murmurs...yes, I know I just said that up above, I'm just following my notes on the PPT here.

The main ones we are focusing on are MITRAL REGURGITATION and AORTIC STENOSIS. Ignore the others!

Mitral Regurgitation

Mitral regurgitation allows blood to flow backward into the left atrium during SYSTOLE. The valve does not close fully (recall that the vegetation has affected its function or the enlarged heart has pulled the valves out of position). This can be caused by either rheumatic heart disease, congenital defects, an MI or CHF. Men have this more often than women.

Note: When your murmur is during SYSTOLE, you will hear a slushy sound at the left sternal border or MCL at the 4th-5th ICS. This slushy sound can obliterate S1 and S2. Just an FYI, mitral stenosis would cause a diastolic murmur (not on test)



When the blood flows back into the left atrium during systole, this increases left atrial volume. Manifestations of this are:

- fatigue, weakness
- DOE, orthopnea
- left sided heart failure → pulmonary edema
- holosystolic murmur

Aortic Stenosis

Aortic stenosis obstructs blood flow from the left ventricle into the aorta. This is also more common in males. It can be idiopathic (from an unknown cause) or due to a congenital defect. As you can imagine, this increases the workload of the left ventricle which causes it to get bigger (hypertrophy). This bigger muscle increases myocardial oxygen demands. Cardiac output is going to go down and your pt may present with syncopal episodes...a common finding!

Aortic stenosis is a progressive disease. Manifestations of LV failure are:

- DOE
- angina
- exertional syncope (a classic sign)
- harsh systolic murmur heard at 2nd RICS
- pulse pressure narrowed to 30 mm Hg

Cardiomyopathy

There are three types of cardiomyopathy and they all affect the heart muscle in both systolic and diastolic functions. It can be primary, secondary or idiopathic, and they are each categorized by their pathophysiology: dilated, hypertrophic or restrictive. The secondary cardiomyopathy can be caused by rheumatic fever!

The diagram to the right shows (from top left) normal heart, dilated cardiomyopathy, hypertrophic cardiomyopathy and restrictive. Cool, huh?

Dilated Cardiomyopathy

This is the MOST COMMON type (87% of cases) and is the most common cause of heart failure. It is a disease of middle aged males that is caused by:

- toxins, alcohol, cocaine
- metabolic conditions
- systemic HTN (this would be long-term HTN...and it is a big cause of this disease)
- infection
- genetic
- chemotherapy
- pregnancy (one more reason, in case you needed one!)

Pathophysiology of Dilated Cardiomyopathy

Scarring and atrophy of the myocardial cells → heart chambers dilate which requires a bigger preload to fill up the ♥, so end diastolic and systolic volumes increase → reduced ejection fraction and decreased cardiac output.

Manifestations of Dilated Cardiomyopathy

- s/s of heart failure
- mitral regurgitation murmur (a systolic murmur) d/t heart valves getting stretched out

Prognosis for Dilated Cardiomyopathy

- 75% of patients die within 10 years, 50% within 5 years
- Pts get dysrhythmias (a-fib, v-fib and v-tach)
- Mural thrombi may form in left ventricular apex and embolize to other parts of the body

Treatment for Dilated Cardiomyopathy

- Management of heart failure
- Implantable cardioverter/defibrillator (one of my pts had one of these)
- Heart transplant

Hypertrophic Cardiomyopathy

Decreased compliance of the left ventricle → hypertrophy of ventricular muscle → small left ventricular volume. Septal hypertrophy obstructs LV outflow and you also get atrial dilation. EF will be below 40%.

Hypertrophic cardiomyopathy is unique in that the muscle may not hypertrophy equally. In the majority of clients the interventricular septal mass especially the upper portion, increases to a greater extent than the free wall of the ventricle. The enlarged upper septum narrows the passageway of blood into the aorta, impairing ventricular outflow. For this reason, this disorder is also known as IHSS or idiopathic hypertrophic subaortic stenosis. Surgical intervention for hypertrophic cardiomyopathy might include excision of part of the ventricular septum.

Manifestations:

- dyspnea
- anginal pain
- syncope
- LV hypertrophy, dysrhythmias, sudden death

Management of Hypertrophic Cardiomyopathy

- BB (cause ↓ HR, ↓ contractility, so can be contraindicated...watch closely)
- Antidysrhythmics (amiodarone)
- Dual-chamber pacing

Restrictive Cardiomyopathy

This is the least common form. Rigid ventricular walls impair DIASTOLIC filling d/t stiff/rigid ventricles and decreased ventricular size/decreased CO. It is caused by myocardial fibrosis and infiltrative processes like amyloidosis. Can also occur with SLE.

Manifestations of Restrictive Cardiomyopathy

- S/S of heart failure
- Mostly right-sided (will see heart on right side of chest on CXR)
- Cardiomegaly

Management of Restrictive Cardiomyopathy

- Little that can be done :-)
- Treatment focuses on symptoms of heart failure

Nursing Care for Cardiomyopathy

- Management of heart failure
- Support and coping skills
- Adaptation to restrictive lifestyle
- Nitrates and vasodilators are AVOIDED in hypertrophic cardiomyopathy (b/c don't want to decrease pre-load)

Nursing Dx for Cardiomyopathy

- Decreased cardiac output
- Fatigue
- Ineffective breathing pattern
- Fear
- Ineffective role performance
- Anticipatory grieving

Diagnostics for Cardiomyopathy

- Echocardiography: assess chamber size and wall motion
- ECG: will show cardiac enlargement via dysrhythmias
- CXR: will show cardiomegaly, pulmonary congestion
- Cardiac catheterization: assess perfusion/ejection fraction/cardiac output
- Myocardial biopsy: infiltration, fibrosis or inflammation

Meds Used to Treat Cardiomyopathy

- ACEi to decrease remodeling
- Vasodilators to decrease afterload
- Digitalis to increase contractility
- BB to reduce cardiac workload
- Anticoagulants to prevent emboli
- Anti dysrhythmics to prevent sudden cardiac death
- Nitrates to manage chest pain and increase perfusion

Black, Joyce M., and Jane Hokanson Hawks. *Medical-Surgical Nursing: Clinical Management for Positive Outcomes - Single Volume (Medical Surgical Nursing- 1 Vol (Black/Luckmann))*. St. Louis: Saunders, 2009. Print.

Brady, D. (2009). Inflammatory Heart Disease Advanced Med/Surg. Lecture conducted from CSU Sacramento, Sacramento.

Deglin, Judith Hopfer, and April Hazard Vallerand. *Davis's Drug Guide for Nurses, with Resource Kit CD-ROM (Davis's Drug Guide for Nurses)*. Philadelphia: F A Davis Co, 2009. Print.

Kelly, K. (2009). Inflammatory Heart Disease. Advanced Med/Surg. Lecture conducted from CSU Sacramento, Sacramento.

Medical-Surgical Nursing Made Incredibly Easy! (Incredibly Easy! Series). Philadelphia: Lippincott Williams & Wilkins, 2008. Print.

Nettina, Sandra M. *Lippincott Manual of Nursing Practice*. Philadelphia: Lippincott Williams & Wilkins, 2006. Print

Springhouse. *Pathophysiology Made Incredibly Easy! (Incredibly Easy! Series)*. Philadelphia: Lippincott Williams & Wilkins, 2008. Print