

RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

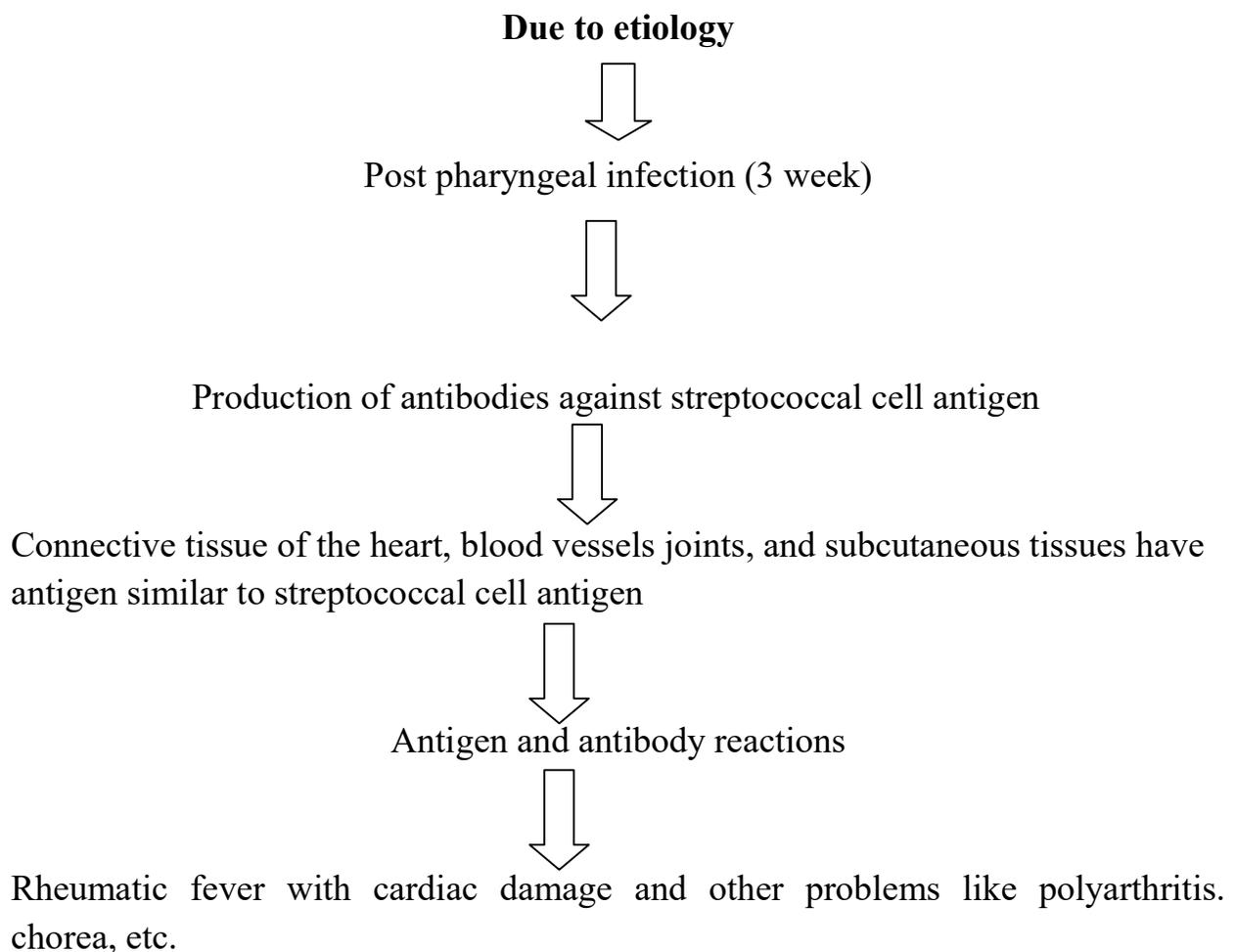
Definition of Rheumatic fever: Rheumatic fever is an inflammatory disease of the heart involving all layers (endocardium, myocardium, and pericardium) of the heart.

Definition of Rheumatic heart disease: Rheumatic heart disease is an autoimmune disorder resulting from rheumatic fever and it is characterized by scarring and deformity of the heart valves.

Causes Or Etiology:

Group A- β -hemolytic streptococcal pharyngitis

Patho-physiology of rheumatic heart disease:



Clinical manifestations:

Major criteria	Minor criteria	Plus
Carditis	Fever	Supporting evidence of preceding streptococcal infection-recent scarlet fever, raised antistreptolysin "O" or other streptococcal antibody titre, positive throat culture.
Polyarthrititis	Arthralgia	
Chorea	Raised erythrocyte sedimentation rate or C-reactive protein	
Erythema marginatum	Leucocytosis	
Subcutaneous nodules	First-degree AV block	

Major Criteria

Carditis: Carditis is the most important manifestation of ARF and results in three signs-(1) an organic heart murmur or murmurs of mitral or aortic regurgitation, or mitral stenosis; (2) cardiac enlargement and HF secondary to myocarditis; and (3) pericarditis resulting in muffled heart sounds, chest pain, pericardial friction rub, or signs of effusion. Carditis, meaning that all layers of the heart (endocardium, myocardium, and pericardium) is involved.

Mono- or polyarthrititis is the most common finding in rheumatic fever. The inflammatory process affects the synovial membranes of the joints, causing swelling, heat, redness, tenderness, and limitation of motion. The larger joints are most frequently affected, particularly the knees, ankles, elbows, and wrists.

Chorea (sydenham's chorea) is the major CNS manifestation of ARF and often a delayed sign occurring several months after the initial infection. It is characterized by involuntary movements, especially of the face and limbs, muscle weakness, and disturbances of speech and gait.

Erythema marginatum lesions are a less common feature of ARE. The bright pink, nonpruritic, maplike macular lesions occur mainly on the trunk and proximal extremities, and may be exacerbated by heat (e.g. warm bath).

Subcutaneous nodules usually associated with severe carditis, are firm, small, hard, painless swellings located over extensor surfaces of the joints, particularly knees, wrists, and elbows.

Diagnostic evaluation:

1. History Collection
2. Physical Assessment or Examination
3. Blood investigations: Leucocytosis, raised ESR, raised CRP, Antistreptolysin 'O'antibodies (ASO titres)
4. Chest X-ray
5. ECG
6. Echocardiogram

Complications:

- Heart failure. This can occur from either a severely narrowed or leaking heart valve.
- Bacterial endocarditis.
- Ruptured heart valve.

Management

Medical Management:

Treatment of the acute attack: A single dose of benzyl penicillin 1.2 million U IM or oral phenoxymethyl penicillin 250 mg 6-hourly for 10 days should be given on diagnosis to eliminate any residual streptococcal infection. If the patient is penicillin-allergic, erythromycin or a cephalosporin can be used.

Bedrest and supportive therapy: Bedrest is important as it lessens joint pain and reduces cardiac workload. The duration of bedrest should be guided by symptoms

and markers of inflammation (e.g. temperature, leucocyte count and ESR) and should be continued until these have settled.

Aspirin: This will usually relieve the symptoms of arthritis.

Corticosteroids: These produce more rapid symptomatic relief than aspirin, and are indicated in cases with carditis or severe arthritis.

Nursing Management

- Reducing Fever
- Assess for effectiveness of drug therapy.
- Take and record temperature every 3 hours
- Evaluate patient's comfort level every 3 hours
- Maintaining Adequate Cardiac Output
- Assess for signs and symptoms of acute rheumatic carditis.
- Be alert to patient's complaints of chest pain, palpitations, and/or chest tightness
- Monitor for tachycardia (usually persistent when patient sleeps) or bradycardia.
- Be alert to development of second-degree heart block.
- Auscultate heart sounds every 4 hours.
- Document the presence of murmur or pericardial friction rub.
- Document the extra heart sounds (S₃, gallop. S₄, gallop).
- Monitor for development of chronic rheumatic endocarditis, which may include valvular disease and heart failure.
- Maintain bedrest for duration of fever or if signs of active carditis are present.
- Provide ROM exercise program.
- Provide diversional activities that prevent exertion.

Nursing diagnosis:

- Acute pain related to Arthralgia as evidenced by Verbal description of pain
- Hyperthermia **related to** Illness or inflammatory disease **as evidenced by** Increase body temperature above normal range

- Activity Intolerance **related to** Decrease cardiac output **evidenced by** Imbalanced oxygen supply and demand
- Anxiety related to hospitalization
- Deficient knowledge related to diseases condition; follow up care, treatment regimen.

INFECTIVE ENDOCARDITIS

Definition: Endocarditis is an infection of the endocardial surface of the heart. The endocardium, the innermost layer of the heart.

Causes:

Bacteria

- Chlamydiae
- Enterococci
- HACEK group (Hemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella)
- Methicillin-resistant Staphylococcus aureus
- Rickettsiae
- Staphylococcus aureus
- Staphylococcus epidermidis
- Streptococcus bovis
- Streptococcus groups A, B, and C
- Streptococcus pneumoniae
- Streptococcus viridans

Fungi

- Candida albicans
- Candida parapsilosis

Viruses

- Coxsackie B virus

Predisposing Conditions for Infective Endocarditis

- Cardiac Conditions
- Prior endocarditis
- Prosthetic valves
- Acquired valve disease (e.g. mitral valve prolapse with murmur, calcified aortic stenosis)
- Cardiac lesions (e.g. ventricular septal defect)
- Rheumatic heart disease (e.g. mitral valve regurgitation)
- Congenital heart disease
- Pacemakers
- Cardiomyopathy.

Non-cardiac Conditions

- Intravenous drug abuse
- Nosocomial bacteremia

Pathophysiology of infective endocardial

Damage to endothelial surface may be due to turbulent blood flow that erodes that erodes the normal infection-resistant endocardium, mitral valve prolapsed with regurgitation, rheumatic heart disease, congenital defects, and valve replacements are especially prone to bacterial invasion.



Causative organisms (bacteria, virus, fungi) adhering to endothelial surface



Multiplication of organisms

Leukocytes, platelets and fibrin covers the multiplying organism and forms vegetative lesion on valve surface.



Local valve damage (mitral or aortic commonly affected)



As blood flows through the heart, these vegetations may break off and become emboli.



Embolization to the spleen, liver, brain, kidney resulting from left sided endocarditis, pulmonary emboli may occur in right-sided endocarditis

Classification of Infective Endocarditis:

- **Native valve endocarditis (NVE)** is an infection seen in patients those have valvular or heart disease that predisposes to infective endocarditis (IE).
- **Prosthetic valve endocarditis (PVE)** it occurs in 1 to 6 percent of all patients with prosthetic cardiac valves. The rates of infection of mechanical and tissue valves are similar.
- **Nosocomial endocarditis** is usually a complication of bacteremia induced by an invasive procedure or a vascular device and accounts for nearly 10% of IE in some areas. IE can occur after pacemaker implantation and has a mortality rate as high as 24%.

Clinical Manifestations

- ✓ **General manifestations:** Chills, weakness, malaise, fatigue, and anorexia. Arthralgias, myalgias, back pain, abdominal discomfort, weight loss, headache And clubbing of fingers.
- ✓ **Cardiac manifestations:**
 - The onset of a new or changing murmur is noted in most patients with IE, with the aortic and mitral valves most commonly affected
 - The mitral murmur of endocarditis is generally a mild-to late systolic regurgitation type.
 - The aortic murmur may be early diastolic.
 - Murmurs are often absent in tricuspid endocarditis because right-sided heart pressures are too low to be heard.

- HE occurs in up to 80% of patients with aortic valve endocarditis and in approximately 50% of patients with mitral valve endocarditis.
- ✓ **Vascular manifestations:**
 - Splinter hemorrhages (black longitudinal streaks) that may occur in the nail beds.
 - Petechiae may occur as a result of fragmentation and microembolization of vegetative lesions and are common in the conjunctivae, the lips, the buccal mucosa, and the palate and over the ankles, the feet, and the antecubital and popliteal areas
 - Osler's nodes (painful, tender, red or purple, pea-size lesions) may be found on the fingertips or toes.
 - Janeways lesions (flat, painless, small, red spots) may be found on the palms and soles.
 - -Roth's spots funduscopic examination may reveal hemorrhagic retinal lesions.
- ✓ **Embolization:** Embolization in various body organs may also be present. Embolization to the spleen may result in sharp, left upper quadrant pain and splenomegaly. Local tenderness and abdominal rigidity may be present. Embolization to the kidneys may cause pain in the flank, hematuria, and azotemia. Emboli may lodge in small peripheral blood vessels of arms and legs and may cause gangrene. Embolization to the brain may cause neurologic damage resulting in hemiplegia, ataxia, aphasia, visual changes, and change in the level of consciousness. Pulmonary emboli may occur in right-sided endocarditis.

Assessment and Diagnostic Studies

1. History Collection
2. Physical Assessment or Examination
3. Blood investigations: blood culture and sensitivity
4. ECG
5. Echocardiogram
6. Chest X-ray
7. Cardiac catheterization

Management

Antimicrobial treatment of common causative organisms in infective endocarditis Benzyl penicillin IV and gentamicin IV

Nursing Management :

Nursing diagnosis:

MYOCARDITIS

Myocarditis is an inflammatory process involving the myocardium. Myocarditis can cause heart dilation, thrombi on the heart wall (mural thrombi), infiltration of circulating blood cells around the coronary vessels and between the muscle fibers, and degeneration of the muscle fibers themselves.

Causes

Viral (Most Common)

- Adenovirus
- Coxsackievirus, enterovirus
- Cytomegalovirus
- Hepatitis C virus
- Influenza virus
- Human immunodeficiency virus
- Herpes virus
- Epstein-Barr virus
- Mixed infections.

Bacterial

- Mycobacterial species
- Chlamydia pneumoniae
- Streptococcal species
- Mycoplasma pneumoniae

- Treponema pallidum.

Fungal

- Aspergillus
- Candida
- Coccidioides
- Cryptococcus
- Histoplasma
- Protozoal

Parasitic

Larva migrans.

Toxins

Cocaine

Hypersensitivity

- Clozapine
- Sulfonamides
- Cephalosporins
- Penicillins

Autoimmune Activation

- Smallpox vaccination
- Giant cell myocarditis
- Churg-Strauss syndrome
- Inflammatory bowel disease
- Celiac disease

Pathophysiology of myocarditis

Due to etiology





Invasion of microorganisms to the myocardium and myocytes.



Inflammatory reactions (the immune response is activated and cytokines and oxygen free radicals are released)



Cellular damage and necrosis



As the infection progresses, an autoimmune response are activated leads to further destruction of myocytes.



Cardiac dysfunction leads to development of dilated cardiomyopathy



Clinical Manifestations

Clinical Manifestations:

Fever, fatigue, malaise, myalgias, pharyngitis, dyspnea, lymphadenopathy, and nausea and vomiting are early systemic manifestations of the viral illness.

Early cardiac manifestations appear 7 to 10 days after viral infection. These include pleuritic chest pain with a pericardial friction rub and effusion because pericarditis often accompanies myocarditis. Late cardiac signs relate to the development of HF and may include an S₃ heart sound, crackles, Jugular venous distention, syncope, peripheral edema, and angina.

Assessment and Diagnostic Studies:

1. History Collection

2. Physical Assessment or Examination
3. Blood investigations: blood culture and sensitivity
4. ECG
5. Echocardiogram
6. Chest X-ray
7. Cardiac catheterization

Management:

Management

The specific treatment for myocarditis has yet to be established, and treatment usually cardiac decompensation. Oxygen therapy, bedrest, and restricted activity are general supportive measures used for management of myocarditis.

Antimicrobial treatment of common causative organisms in infective endocarditis Benzyl penicillin IV and gentamicin IV

Lanoxin is often used to treat ventricular failure because it improves myocardial contractility and reduces ventricular rate.

Diuretics may be used to reduce fluid volume and decrease preload.

Digoxin should be used cautiously in patients with myocarditis because it improves Vasodilators such as nitroprusside (Nitropress), inamrinone (Inocor), and milrinone (primacor) are used to reduce afterload and improve CO by decreasing systemic arterial resistance.

Anticoagulation therapy

Immunosuppressive therapy with agents such as prednisone, azathioprine (Imuran), and used to reduce myocardial inflammation and to prevent irreversible myocardial damage.

Intravenous immunoglobulin (IVIG) is being used on an experimental basis to treat myocarditis.

Nursing Management

Reducing Fever

Administer antipyretics as directed

Check temperature every 4 hours

Administer antibiotics as directed.

Maintaining Cardiac Output

Record daily intake and output

Record daily weight.

Check for peripheral edema

Elevate head of bed, if necessary, to enhance respiration

Treat the symptoms of heart failure as prescribed

Evaluate patient's pulse and apical rate for signs of tachycardia and gallop rhythm indications that heart failure is recurring

Evaluate for evidence of dysrhythmia patients with myocarditis are prone to develop dysrhythmias.

Reducing Fatigue

Ensure bedrest to reduce heart rate, stroke volume, BP, and heart contractility; also helps to decrease residual damage and complications of myocarditis, and promotes healing

Provide diversional activities for patient

Allow patient to use bedside commode rather than bedpan (reduces cardiovascular workload)

Discuss patient activities that can be performed after discharge.

Pericarditis

Pericarditis: Pericarditis is a condition caused by inflammation of the pericardial sac (the pericardium), which may occur on an acute basis. The pericardium is composed of the inner serous membrane (visceral pericardium) that closely adheres to the epicardial surface of the heart and the outer fibrous (parietal layer). The pericardial space is the cavity between these two layers, and, in the normal state, it contains 10 to 30 mL of serous fluid. Although the pericardium may be congenitally absent or surgically removed, it serves a useful anchoring function, provides lubrication to decrease friction during systolic and diastolic heart movements, and assists in preventing excessive dilation of the heart during diastole.

Classification of Pericarditis

Clinical Classification

- **Acute pericarditis (<6 weeks)**
- **Subacute pericarditis (6 weeks to 6 months)**
- **Chronic pericarditis (6 months)**

Etiologic Classification

Infectious pericarditis

Viral (coxsackievirus A and B, echovirus, mumps, adenovirus, hepatitis, HIV)

Pyogenic (pneumococcus, streptococcus, staphylococcus, Neisseria, Legionella)

Tuberculous

Fungal (histoplasmosis, coccidioidomycosis, Candida, blastomycosis)

Other infections (syphilitic, protozoal, parasitic).

Non infectious pericarditis

- Acute myocardial infarction
- Uremia
- Neoplasia
- Primary tumors (benign or malignant)
- Tumors metastatic to pericardium (lung and breast cancer, lymphoma, leukemia)
- Myxedema
- Cholesterol
- Chylopericardium
- Trauma: Penetrating chest wall, Nonpenetrating
- Aortic dissection (with leakage into pericardial sac)
- Post-irradiation
- Familial pericarditis
- Acute idiopathic

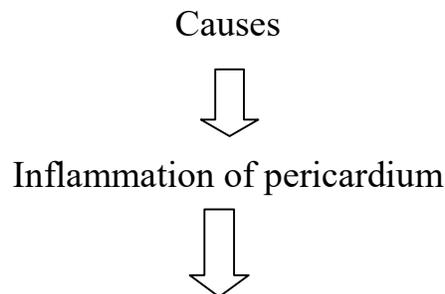
Pericarditis Presumably Related to Hypersensitivity or Autoimmunity

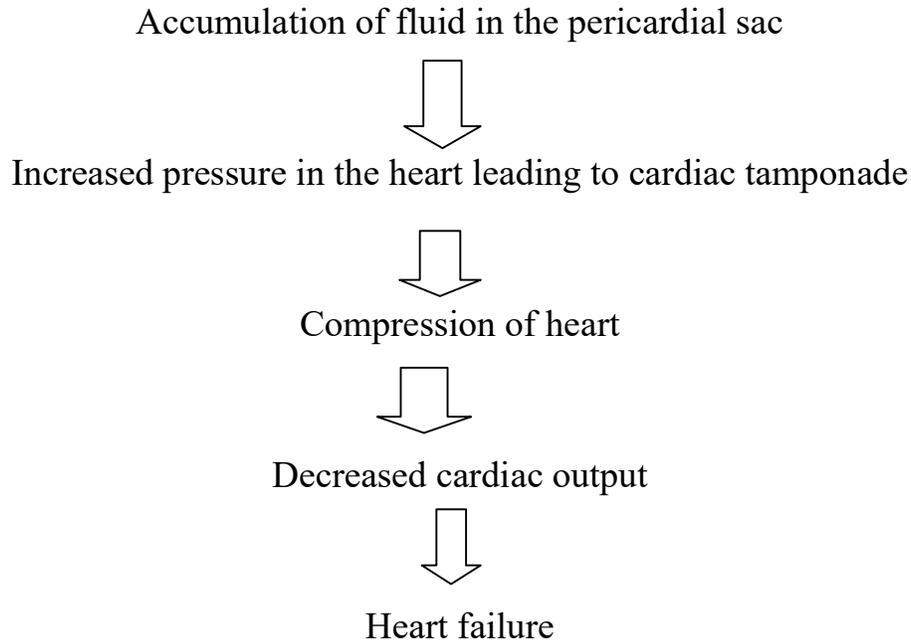
- Rheumatic fever
- Collagen vascular disease (SLE, rheumatoid arthritis, ankylosing spondylitis, scleroderma, acute rheumatic fever)
- Drug -induced (e.g. procainamide, hydralazine, phenytoin, isoniazide, minoxidil, anticoagulants, methysergide).

Postcardiac Injury

- Postmyocardial infarction 1 to 12 weeks (Dressler's syndrome)
- Postpericardiotomy
- Posttraumatic.

Pathophysiology of pericarditis





Clinical Manifestations

A progressive, frequently severe chest pain that is sharp and pleuritic in nature. The pain is generally worse with deep inspiration and when lying Supine. It is relieved by sitting. The pain may radiate to the neck, arms, or left shoulder, making it difficult to differentiate from angina. One distinction is that the pain from pericarditis can be referred to the trapezius muscle (shoulder, upper back) as the phrenic nerve innervates these two regions.

The dyspnea that accompanies acute pericarditis is related to the patient's need to breathe in rapid, shallow breaths to avoid chest pain and may be aggravated by fever and anxiety.

The hallmark finding in acute pericarditis is the pericardial friction rub. The rub is a scratching, grating, high pitched sound believed to arise from the friction between the roughened pericardial and epicardial surfaces.

Assessment and Diagnostic Studies

1. History Collection

2. Physical Assessment or Examination
3. Blood investigations: blood culture and sensitivity, include leukocytosis and elevation of CRP and ESR.
4. ECG
5. Echocardiogram
6. Chest X-ray
7. Cardiac catheterization

Complications: pericardial effusion and cardiac tamponade.

Pericardial effusion is an accumulation of excess fluid in the pericardium. It can occur rapidly (e.g. chest trauma) slowly (eg tuberculous pericarditis).

Cardiac tamponade develops as the pericardial effusion increases in volume, causing an increase in intra pericardial pressure and results in compression of the heart. The speed of fluid accumulation affects the severity of clinical manifestations. Cardiac tamponade can occur acutely (e.g. rupture of heart, trauma) or subacutely (e-g: secondary to uremia, malignancy).

Management:

Antimicrobial treatment of common causative organisms in infective endocarditis Benzyl penicillin IV and gentamicin IV

Administer Corticosteroids to the client.

Pericardiocentesis is usually performed for pericardial effusion with acute cardiac tamponade, purulent pericarditis and a high suspicion of a neoplasm.

Nursing Management

Assess heart rate, rhythm, BP, respirations at least hourly in the acute phase; continuously if hemodynamically unstable.

Assess for signs of cardiac tamponade increased heart rate, decreased BP presence of paradoxical pulse, distended neck veins, restlessness, muffled heart sounds.

Prepare for emergency pericardiocentesis or surgery. Keep pericardiocentesis tray at bedside.

Assess for signs of heart failure.

Monitor closely for the development of dysrhythmias.