Endocarditis/Pericarditis/Myocarditis/Valvular Disease

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ENDOCARDITIS/PERICARDITIS/MYOCARDITIS/
VALVULAR HEART DISEASE

I. PERICARDITIS

A. Etiology-multiple

1. Idiopathic
2. Infectious
   a. Viral (coxsackie, echo/adenovirus, influenza, HIV)
   b. Bacterial (staph, strep, TB)
   c. Fungal (histo, aspergillosis)
   d. Parasite
   e. Rickettsia
3. Malignancy (leukemia, lymphoma, breast, lung)
4. Post-MI
5. Radiation-induced
6. Post-traumatic
7. Uremia
8. Autoimmune
9. Drug-induced (procainamide, hydralazine)
10. Myxedema
11. Aortic dissection

B. Symptoms

1. Substernal chest pain
2. Varies with respiration
3. Improves with leaning forward
4. Fever, myalgias, anorexia
5. Dyspnea related to pain with respiration

C. Signs—essentially the same for all etiologies except when noted

1. Rub heard best over the left sternal border (25%)
2. Intermittent, migratory
3. Rub varies with respiration, position
4. EKG
   a. Sinus tachycardia
   b. Nonspecific ST-T wave changes
   c. ST ↑ diffuse except AVR/V1 represents subepicardial ventricular injury
   d. PR ↓ represents subepicardial atrial injury
5. EKG stages
a.  I:    ST ↑ and PR ↓, reciprocal ST ↓ AVR, V1
b.  II:   Transiently normal - T waves may flatten
c.  III:  Deep symmetrical TWI
d.  IV:  Normal or permanent TWI
e.  May be difficult to distinguish from AMI-ST segments
   concave instead of convex and no anatomical pattern present
6.  Elevated WBC, ESR
7.  Elevated cardiac enzymes including troponin I

D.  Diagnosis

1.  Clinical symptoms
2.  Echocardiogram
3.  CT/MRI
4.  Pericardioscopy

E.  Treatment

1.  NSAIDS
2.  Prednisone for those patients who cannot tolerate NSAIDS
3.  Inpatient treatment
   a.  Cannot r/o MI
   b.  Severe pain
   c.  Pericardial effusion

F.  Complications

1.  Chronic pericarditis (treated with colchicines, prednisone)
2.  Pericardial effusion
3.  Constrictive pericarditis

G.  Recovery

1.  Complete: 60% in one week
2.  Prolonged course in 3%
3.  16% recurrence in the first year
4.  3% recurrence after first year
5.  Recurrent may require additional work-up to exclude malignancy

H.  Specific etiologies

1.  Uremia - etiology unknown
   a.  Possible toxic metabolites
   b.  Bleeding diathesis
   c.  Immunologic mechanism
2.  Findings in uremic pericarditis
a. Pericardial effusion (fibrinous, bloody)
   b. Diagnostic pericardiocentesis may be required to rule out an infectious cause
   c. Cardiac tamponade (one of the most common causes)
   d. No EKG changes (very little epicardial inflammation)
   e. Otherwise typical findings
3. Treatment in uremic pericarditis
   a. 2-6 weeks intensive hemodialysis
   b. Systemic steroids for 1-2 weeks
   c. Possible intrapericardial steroids
   d. Treat tamponade if present
   e. Careful with NSAIDS because of bleeding tendency
4. Post-MI pericarditis
   a. Early: 2-4 days post MI
      i. Chest pain is of different quality than MI pain
      ii. Friction rub, fever
      iii. EKG findings rarely seen (masked by the new EKG changes)
      iv. Short-lived
      v. Treat with NSAIDS
   b. Late: Dressler’s syndrome
      i. Autoimmune
      ii. Anti myocardial antibodies
      iii. NSAIDS
      iv. Resolution complete/rapid
      v. May discontinue anticoagulants to reduce risk of hemorrhagic effusion
5. Post-traumatic pericarditis
   a. 4-12 days post injury
   b. Autoimmune
   c. Same symptoms
   d. NSAIDS
6. Neoplastic pericarditis
   a. Mostly metastatic
   b. Nonspecific symptoms
   c. May contribute to patient’s demise/tamponade
   d. Treatment: chemo, pericardiocentesis
   e. Prognosis dependent on tumor type (breast and lymphoma better; lung worse)
7. Radiation-induced pericarditis: typical symptoms, high survival rate, treat supportive, most spontaneously resolve
8. Collagen vascular disease: RA/lupus, sarcoidosis, scleroderma, amyloidosis - treat with steroids
9. Purulent pericarditis
   a. Acute, life-threatening
   b. Febrile illness, chest pain, friction rub, tachycardic, toxic-
appearing
c. Elevated WBC, CVP and hepatomegaly
d. Suspect in patients treated for a systemic illness, improves and then c/o chest pain, fever, dyspnea
e. Diagnosis by pericardiocentesis
f. Treatment with drainage, IV antibiotics
g. Complicated by constrictive pericarditis

10. TB pericarditis
   a. Rare but incidence increasing secondary to HIV
   b. Triple drug therapy for 9 months, surgery

11. Miscellaneous etiologies
   a. Mycoplasma pneumoniae
   b. Chlamydia trachomatis
   c. EBV, CMV, HIV

II. CARDIAC TAMponade

A. Etiology

1. Pericardial effusion ⇒ impaired filling ⇒ ↓ cardiac output
2. Dependent on the rate of accumulation of fluid
3. End result is equalization of pressures in all chambers

B. Symptoms

1. Nonspecific
2. Chest pain, cough, dyspnea

C. Signs

1. Spectrum of minimal cardiac decompensation to shock
2. Pulsus paradoxus
3. Beck’s triad: hypotension, JVD, muffled heart sounds (may not be present if tamponade develops quickly)
4. Kussmaul’s sign: JVD with inspiration
5. EKG: low voltage, electrical alternans is pathognomonic (20%)
6. CXR-cardiomegaly with large accumulation of fluid (200-250ml)

D. Diagnosis

1. Echocardiogram
2. Right atrial and ventricular collapse during diastole

E. Treatment

1. Volume augmentation to right ventricle with IVF’s (increase
III. CONSTRUCTIVE PERICARDITIS

A. Etiology: viral, idiopathic, uremia, radiation, cardiac surgery, trauma, complication from pericarditis
   1. Thickening and fibrosis of pericardium
   2. Impaired ventricular filling ⇒ equalization of right/left heart pressures

B. Symptoms of CHF (right sided > left sided)

C. Signs
   1. Friction rub
   2. Pericardial knock in early diastole
   3. Third heart sound (filling complete after 1/3 of diastole)
   4. EKG: low voltage, nonspecific ST-T wave, atrial dysrhythmias
   5. Small heart size
   6. Pericardial calcification
   7. LFT’s consistent with passive congestion
   8. Pulsus paradoxus absent (no fluid to transmit the respiratory variation)

D. Diagnosis: echo, CT scan/MRI, cardiac cath

E. Treatment: disease state progresses slowly, pericardectomy

IV. MYOCARDITIS

A. Incidence unknown

B. Classified as inflammatory cardiomyopathy

C. Multiple etiologies
   1. Coxsackie B (summer outbreaks)
   2. Adenovirus
   3. Influenzae A and B
   4. Strep
   5. Mono
   6. Post-vaccine
   7. CMV/toxo in transplant/immunocompromised

Endocarditis/Pericarditis/Myocarditis/Valvular Heart Disease
8. Chagas’ disease-worldwide #1 cause

D. Symptoms

1. Flu-like/fever/URI
2. Myalgias
3. Vomiting/diarrhea
4. CHF symptoms

E. Signs

1. Dysrhythmias
2. Elevated cardiac enzymes
3. Elevated WBC/ESR
4. Unexplained CHF
5. EKG changes
   a. Sinus tachycardia
   b. AMI pattern
   c. ST segment depression, TWI
   d. Prolonged Q-T interval, AV block
   e. Low voltage

F. Diagnosis

1. Gold standard is endocardial biopsy
2. Echocardiogram (multi chamber dysfunction)
3. Gallium/Technetium scan
4. Viral titers (low yield)
5. Indium III antimyosin antibodies - bind to exposed myosin indicating ongoing myocardial necrosis

G. Treatment

1. Supportive, bedrest
2. Antivirals still being tested for efficacy
3. Immunosuppressive drugs being tested in specific populations
4. IV immunoglobulin testing in children

H. Complications

1. Dilated cardiomyopathy
2. Ventricular dysrhythmias
3. LV aneurysm formation
I. Prognosis

1. Variable
2. 1/3 complete recovery
3. 1/3 ongoing cardiac dysfunction
4. 1/3 chronic heart failure

J. Mortality

1. 20% 1 year
2. 56% 4 years
3. Fulminant myocarditis best prognosis
4. Patients transplanted for myocarditis - decreased survival and increased rejection

K. Specific Etiologies

1. Parasitic
   a. Chagas’ disease (Trypanosoma cruzi)
   b. Leading cause of death in Central America
   c. ¾ no cardiac symptoms
   d. ¼ arrhythmias
   e. Angina-like chest pain, syncope
   f. Parasites in serum = extent of tissue damage
   g. 2D echo shows a peculiar apical LV aneurysm/scar
2. Lyme disease
   a. Unexplained heart block from an endemic area
   b. Treat with antibiotics - they may reverse the block
   c. The role of antibiotics in preventing Lyme carditis is unknown
3. HIV
   a. 46% have myocarditis on postmortem
   b. Pathogenesis is multifactorial
   c. CMV/toxo most common etiologies
   d. Treatment is with HIV meds
4. Kawasaki syndrome
   a. 50% develop syndrome
   b. Same classic symptoms
   c. Treatment: high dose ASA and IV gammaglobulin

V. INFECTIVE ENDOCARDITIS

A. Overall review

1. Infection of the endothelial lining of the valve and heart
2. Acute and subacute forms
3. All organisms implicated - bacteria most common
4. Average age 55-60
5. Mortality rate 20-40%
   a. Native valve: 16-27%
   b. Prosthetic valve: early 30-80%, late 20-40%
   c. IVDU: decreased mortality due to right-sided lesions

B. Risk factors

1. Rheumatic fever/congenital heart disease (most common)
2. Calcific/degenerative valve disease
3. History of endocarditis
4. IVDU
5. Prosthetic heart valve (same risk for mechanical and prosthetic valve
6. MVP with regurgitation
7. Intracardiac pacemaker
8. Intravascular devices (catheters and shunts)

C. Pathophysiology

1. Initial insult creates a lesion ⇒ sterile thrombus formation ⇒
   microorganisms adhere and colonize
2. Subclinical bacteremia within one week
3. Embolizations of vegetations

D. Causative organisms

1. Native valve
   a. Strep: 65% (viridans most common), enterococci
   b. Staph: 10-30%
   c. Most often present as subacute, left-sided lesions
2. IVDU
   a. Primarily right-sided lesions but may affect any valve
   b. Staph: 80%
   c. Pseudomonas, Serratia, Haemophilus, gram negatives
   d. Presents acutely
   e. Rarely have predisposing lesion - thought to be due to
      adjunctive compounds like talc
3. Prosthetic valves
   a. Early: <60 days post-op, Staph epidermidis, HACEK, culture
      negative
   b. Late: >60 days post-op, resembles native valve endocarditis,
      may result in leaks, CHF, dehiscence
4. Fungal
   a. Candida
   b. Aspergillus
   c. Seen in long term indwelling IV catheters, pacemakers, AICD’s, immunosuppression, IVDU

E. Symptoms

1. Nonspecific
2. Most common intermittent fever 85%
3. Classic triad of fever (>38°), anemia, and murmur (new or evolving) - RARE
4. Malaise (80%)
5. Neurologic symptoms (30-40%)

F. Signs

1. Fever
2. Murmur (absent in 15%)
3. CHF
4. Vasculitic lesions: petechiae (mucosal surface or skin), splinter hemorrhages, Osler nodes, Janeway lesions (35%)
5. Splenomegaly (30%)
6. Ocular findings: conjunctival/retinal hemorrhages, Roth spots
7. Laboratory findings are nonspecific
   a. Elevated WBC/ESR
   b. Anemia
   c. Hematuria (>50%)
   d. Blood culture positive
8. CXR: CHF, septic emboli
9. EKG: infarction, conduction defects

G. Diagnosis

1. Often mistaken for other illnesses
2. Inquire about high risk procedures/IVDU
3. Obtain at least 3 sets of blood cultures (90-95% positive)
4. Echocardiogram
   a. TEE more sensitive especially in prosthetic valves
   b. Reveals vegetations, evaluates valve and myocardial function
   c. Start with TTE-more cost effective (sensitivity 60-70%)

H. Treatment - IV ABX 4-6 weeks

1. Native valve
   a. PCN 5 million units IV q 4 hr OR Ceftriaxone 2 gm q 24 hr
2. IVDU
   a. Vancomycin 1g q 12 hr PLUS
   b. Gentamicin 3 mg/kg IV q 24 hr

3. Prosthetic valve
   a. Vancomycin 15mg/kg IV q 12 hrs PLUS
   b. Gentamicin 3mg/kg q 24 hr PLUS
   c. Rifampin 900mg q 24 hr

4. Surgery indications
   a. Life threatening CHF or cardiogenic shock secondary to valvular insufficiency
   b. Annular or aortic abscesses
   c. Major embolic complications
   d. Continued infection despite ABX treatment
   e. Fungal disease
   f. Arrhythmia/new conduction defects

I. Prophylaxis indications

1. High risk category (Class IIa, Level of Evidence B)
   a. Prosthetic valves or prosthetic material used
   b. Previous bacterial endocarditis
   c. Complex cyanotic congenital heart disease, unrepaired
   d. Surgically constructed systemic-pulmonary shunts or conduits
   e. Completely repaired congenital heart disease with prosthetic material or device (either by catheter or surgery) during first 6 months after procedure
   f. Repaired congenital heart disease with residual defect
   g. Cardiac transplant recipients who develop valvulopathy (level of evidence C)

J. Procedures requiring prophylaxis

1. Dental procedures which involve manipulation of either gingival tissue or the periapical region of teeth or perforation of oral mucosa
2. High risk patients plus incision of respiratory tissue such as tonsillectomy and adenoidectomy
3. High risk patients with current infections in the GI or GU tract to prevent worsening infection/sepsis. Antibiotic choice is guided to infection source.
K. Procedures not requiring prophylaxis

1. Prophylaxis is not recommended for nondental procedures in the absence of active infection (Level of Evidence B)
2. Administration of antibiotics to prevent endocarditis is not recommended for patients who undergo a GI or GU tract procedure

L. Antibiotic regimens for prophylaxis - see attached handout

VI. VALVULAR HEART DISEASE

A. Overview

1. 90% chronic
2. Decades between structural abnormalities and symptoms
3. Chronic adaptation by dilation and hypertrophy

B. Mitral valve prolapse (MVP)

1. Affects 2.5% of population, mostly women
2. Etiology unknown
3. Myxomatous proliferation which allows abnormal stretching of the valve leaflet
4. Commonly affects the posterior leaflet
5. Both leaflets → mitral regurgitation
6. Symptoms of MVP
   a. Palpitations (PVC’s, rarely SVT and VT)
   b. Chest pain (typically sharp and localized)
   c. Fatigue, lightheadedness, SOB
7. Signs of MVP
   a. Click secondary to snapping of chordae tendineae during prolapse of the valve (20% classic)
   b. Midsystolic click followed by late systolic crescendo murmur heard best between apex and left sternal border
8. Diagnosis by echo
9. Treatment with beta blockers
10. Complications
    a. Mitral regurgitation
    b. Infective endocarditis
    c. Dysrhythmias
    d. Sudden death
C. Mitral stenosis

1. Latency period 20 years
2. Most common cause is rheumatic fever
3. Other causes: congenital, thrombus, atrial myxoma, calcification of annulus/leaflets
4. Course: stenosis → impedes filling → left atrial enlargement → left ventricular failure → pulmonary hypertension → right-sided failure
5. Symptoms of mitral stenosis
   a. DOE (80%)
   b. Orthopnea
   c. Hemoptysis
   d. Increased demands may precipitate symptoms (pregnancy, anemia, infection)
6. Signs of mitral stenosis
   a. Palpable diastolic thrill over apex
   b. Loud S1
   c. Opening snap early diastole followed by low-pitched rumble at apex (mid-diastole), accentuated in left lateral decubitus position
   d. Prominent a wave in the neck
   e. EKG: a. fib (40%), left atrial enlargement
7. Complications of mitral stenosis
   a. Atrial fibrillation
   b. Embolic events (total incidence 20% and 75% of these are neurologic)
   c. CHF

D. Mitral regurgitation

1. Acute
   a. Catastrophic event
   b. Rupture of chordae tendineae, papillary muscle, or valve perforation
   c. Regurgitant flow can be 3-4X the forward flow
   d. Seen in inferior wall MI 2° RCA occlusion (also infective endocarditis and trauma)
   e. Presents with severe pulmonary edema and new murmur
   f. Loud murmur: crescendo-decrescendo systolic murmur ending before S2 heard best at the apex
   g. Treatment: cardiac cath to assess severity and need for emergent surgery, treat pulmonary edema, possible intra-aortic balloon pump
2. Chronic
   a. Usually from rheumatic heart disease
b. Often coexists with mitral stenosis
c. Course: left ventricle compensates
d. Most remain asymptomatic but eventually develop DOE(#1),
a. fib, fatigue
e. Murmur: high-pitched holosystolic heard at apex with
radiation to axilla
f. Palpable left ventricular heave
g. Treatment: medical management

E. Aortic stenosis

1. < 65 congenital bicuspid valve
2. > 65 calcific degeneration
3. Rheumatic disease second most common
4. Significant obstruction when orifice < 1.0 cm or pressure gradient
   > 50 mmHg
5. LVH will compensate prior to this
6. Symptoms of aortic stenosis
   a. DOE with LV failure
   b. Angina
c. Exertional syncope (decreased cerebral blood flow and
   arrhythmias)
   d. Once symptoms begin, life expectancy ≈ 5 years without
   operative repair
7. Signs of aortic stenosis
   a. Murmur: raspy, low-pitched crescendo-decrescendo systolic
      murmur heard best at the base, radiates to carotids
   b. Paradoxical splitting of S2
c. Carotid pulse diminished and slow rising
d. Pulse pressure may be reduced
e. Brachioradial delay
8. Treatment of aortic stenosis
   a. Immediate referral for operative repair (medical therapy
   futile)
   b. Increased risk of sudden death 2o arrhythmia (25%)
c. Avoid vasodilators (NTG)

F. Aortic regurgitation

1. Usually results from rheumatic fever
2. May be acute in infective endocarditis, aortic dissection at root,
   Marfan’s, syphilis, trauma
3. Acute presentation results in pulmonary edema (in new onset
   CHF, consider possible aortic or mitral regurgitation)
4. Chronic aortic regurgitation → left ventricle compensates with
   hypertrophy
5. #1 symptom is again dyspnea
6. Signs of aortic regurgitation
   a. Rapidly rising/falling carotid pulse (waterhammer or Corrigan’s pulse)
   b. Nail pulsations (Quincke’s sign)
   c. Head bobbing
   d. Widened pulse pressure
   e. High-pitched blowing diastolic murmur at left sternal border
   f. Austin-Flint murmur (soft diastolic rumble - regurgitant stream hitting mitral valve)
   g. Duroziez’s murmur (a to-and-fro murmur over the femoral artery)
7. Treatment of aortic regurgitation
   a. Acute → surgical
   b. Chronic → medical with ACE inhibitors, diuretics, then surgical when left ventricle failure occurs

G. Tricuspid stenosis

1. Rheumatic in origin
2. Corresponds with aortic/mitral disease
3. Symptoms of tricuspid stenosis
   a. Fatigue
   b. Venous congestion
4. Signs of tricuspid stenosis
   a. Prominent jugular venous a wave
   b. High-pitched diastolic murmur at left sternal border
   c. Murmur increases with inspiration
   d. Hepatosplenomegaly
   e. Atrial fibrillation
5. Treatment of tricuspid stenosis
   a. Fluid/salt restriction
   b. Surgical replacement

H. Tricuspid regurgitation

1. Usually 2° pulmonary hypertension
2. Also seen in rheumatic fever, infective endocarditis, trauma
3. Symptoms of tricuspid regurgitation
   a. Dyspnea
   b. Painful hepatomegaly
   c. Ascites
   d. Peripheral edema
4. Signs of tricuspid regurgitation
   a. Prominent c-v wave in jugular vein
   b. Right ventricular heave
c. Murmur: high-pitched pansystolic in 4th intercostal space
d. Prominent P2 and S3
e. RBBB
f. Atrial fibrillation

5. Treatment
   a. Fluid/salt restriction
   b. Surgical replacement

I. Valve dysfunction in the ED - immediate evaluation required
   1. New murmur
   2. Diastolic murmur
## MEDICATIONS

### Prophylactic Regimens for Dental Procedures

<table>
<thead>
<tr>
<th>Situation</th>
<th>Agent</th>
<th>Regimen*</th>
</tr>
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<tbody>
<tr>
<td>Standard general prophylaxis</td>
<td>Amoxicillin</td>
<td>Adults: 2.0 gm; children: 50 mg/kg orally 1 h before procedure</td>
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<tr>
<td>Unable to take oral medications</td>
<td>Ampicillin Or Cefazolin Or Ceftriaxone</td>
<td>Adults: 2.0 gm intramuscularly (IM) or intravenously (IV); children: 50 mg/kg IM or IV within 30 min. before procedure 1 gm IM or IV; children 50 mg/kg IM or IV</td>
</tr>
</tbody>
</table>
| Allergic to penicillin                         | Clindamycin or Cephalexin or Azithromycin or clarithromycin | Adults: 600 mg; children: 20 mg/kg orally 1 h before procedure  
Adults: 2.0 gm; children: 50 mg/kg orally 1 h before procedure  
Adults: 500 mg; children: 15 mg/kg orally 1 h before procedure |
| Allergic to penicillin and unable to take oral medications | Clindamycin or Cefazolin Or Ceftriaxone | Adults: 600 mg IM or IV; children 20 mg/kg IM or IV within 30 min. before procedure  
Adults: 1.0 gm; children 25 mg/kg IM or IV within 30 min. before procedure |
ENDOCARDITIS/PERICARDITIS/MYOCARDITIS/VALVULAR HEART DISEASE

PEARLS

1. The most common etiology of pericarditis is idiopathic/viral.

2. Symptoms of pericarditis include chest pain that improves with leaning forward.

3. EKG changes in pericarditis include diffuse ST segment elevation, PR depression, and nonspecific ST-T wave changes.

4. Dressler’s syndrome is pericarditis 2-3 weeks post-MI.

5. Myocarditis should be considered in any patient with a viral syndrome and signs of cardiac disease.

6. Cardiac tamponade results in equalization of pressures in all chambers.


8. Electrical alternans is pathognomonic in tamponade.

9. Fever is the most common presenting sign in patients with infective endocarditis.

10. Strep viridans is the most common cause of native valve endocarditis.

11. Staph aureus is the most common cause of IVDU endocarditis.

12. IVDU endocarditis is most commonly right-sided.

13. Prophylaxis for endocarditis is recommended in high risk patients that are undergoing a dental procedure that involve manipulation of the gingival tissue.

14. 90% of valvular heart disease is chronic.

15. Acute mitral regurgitation may occur from rupture of the chordae tendineae or valve perforation seen in IWMI secondary to RCA occlusion.

17. Treatment for aortic stenosis is immediate operative repair - medical treatment is futile.

18. Avoid vasodilators in aortic stenosis.

19. New onset CHF: consider aortic or mitral regurgitation.

20. Immediate evaluation is required for new murmur or diastolic murmur.
REFERENCES


