Infective Endocarditis

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CVS Module

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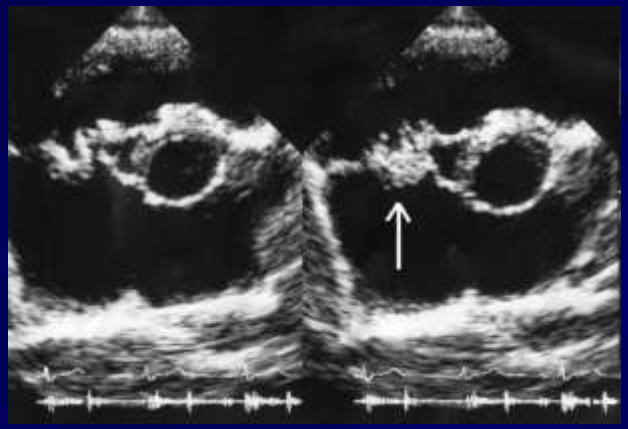
Infective endocarditis

- Microbial infection of the endothelial surface
  - Valves
  - Septal defect
  - chordae tendineae
  - mural endothelium
- NVE: native valve endocarditis
- PVE: prosthetic valve endocarditis
  - Acute
  - Subacute

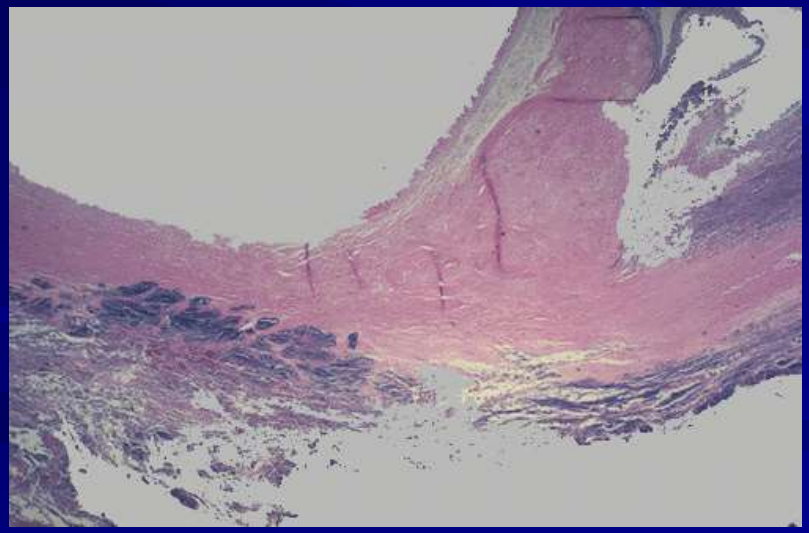
Infective endocarditis- classification

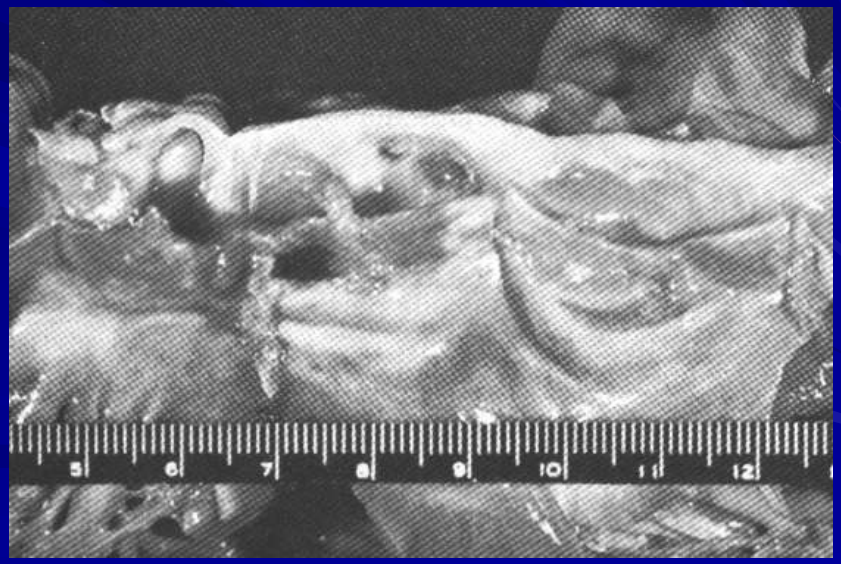
- Duration (incubation)
- Culture results
- Heart side
- Infection setting
- Valve type



Infection

Growth of vegetation by platelet-fibrin deposition yields a sanctuary for bacteria.





Infective endocarditis

- Occurs on
  - Defective valves
  - Prosthetic valves
  - Normal valves
  - Congenital heart defects e.g.
    - Ventricular septal defect
    - Patent ductus arteriosus
- 40% with IE have
  - Normal heart
  - Undiagnosed defect

Infective Endocarditis

- Febrile illness
- Persistent bacteremia
- Characteristic lesion of microbial infection of the endothelial surface of the heart

the vegetation

  - Variable in size
  - Amorphous mass of fibrin & platelets
  - Abundant organisms
  - Few inflammatory cells

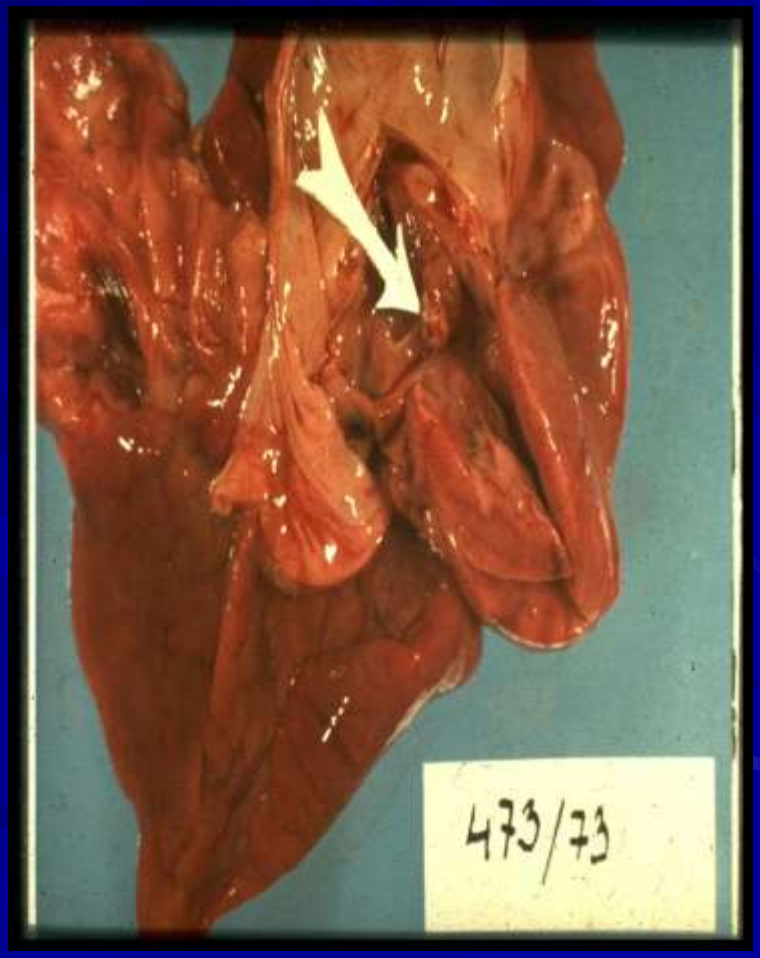
Infective Endocarditis

- Acute
  - Toxic presentation
  - Progressive valve destruction & metastatic infection developing in days to weeks
  - Most commonly caused by *S. aureus*

Infective Endocarditis

- Subacute
  - Mild toxicity
  - Presentation over weeks to months
  - Rarely leads to metastatic infection
  - Most commonly

    viridans streptococci or enterococcus



Infective endocarditis

Incidence: 2 / 100,000 patient-years,
15—30 / 100,000 patient-years (>60 y/o)

- Rheumatic heart disease
- Congenital heart disease
- Mitral valve prolapse with regurgitation
- Degenerative heart disease
- Asymmetrical septal hypertrophy
- Intravenous drug abuse
- Prosthetic valve (7—25%)

Infective endocarditis:
patient groups

- Children with IE:
  - Congenital heart disease (aortic valve),
  - Normal structure (tricuspid valve),
  - Staphylococcus (neonate),
  - Streptococcus group B (children), *S. pneumonia* (rare)
- Adults with IE:
  - Rheumatic heart disease
  - Congenital heart disease

Infective Endocarditis

- Intravenous Drug Abuse
  - Risk is 2 – 5% per pt./year
  - *S. aureus* predominant organism (>50%, 60-70% of tricuspid cases)
- Prosthetic Valve Endocarditis (PVE)
  - Early PVE – within 60 days
    - Nosocomial (*S. epidermidis* predominates)
  - Late PVE – after 60 days
    - Community (same organisms as NVE)

Infective endocarditis:
nosocomial

- Infected intracardiac device and catheter
- GI or GU tract surgery or instrumentation
- High mortality (40—56%)
- (*S. aureus*, CONS, Enterococcus)
- *S. aureus* catheter related bacteremia (23%)

Infective endocarditis:

microorganism

- viridans streptococcus:
  - 35 — 65% NVE
  - normal inhabitants of the oropharynx
  - penicillin sensitive
  - penicillin plus aminoglycoside

Infective endocarditis:
microorganism

- *Streptococcus pneumoniae*:
  - alcoholism
  - aortic valve
  - concurrent pneumonia or meningitis
  - Penicillin / Rocephin → Vancomycin
- Enterococcus:
  - normal GI tract flora and cause GU infection
  - 5—15% NVE and PVE
    - Penicillin / Ampicillin / Vancomycin / Teicoplanin + GM

Infective endocarditis: microorganism

- Staphylococcus:

  Coagulase-positive: *S. aureus*

  highly toxic febrile

  30—50% CNS involvement

  Mortality: 16—46% (L't), 2—4% (R't)

  Oxacillin / 1st cephalosporin

  Coagulase-negative: *S epidermidis*

  Major cause of PVE

Infective Endocarditis

- Gram negative organisms
  - *P. aeruginosa* most common
  - HACEK - slow growing, fastidious organisms that may need 3 weeks to grow out of culture
    - Haemophilus sp.
    - Actinobacillus
    - Cardiobacterium
    - Eikenella
    - Kingella
- Other Organisms like Aspergillus species, Brucella species, *Coxiella burnetii*, Chlamydia species

Pathogenesis

- Vegetations along valve edges: mass of fibrin, platelets and infectious organisms

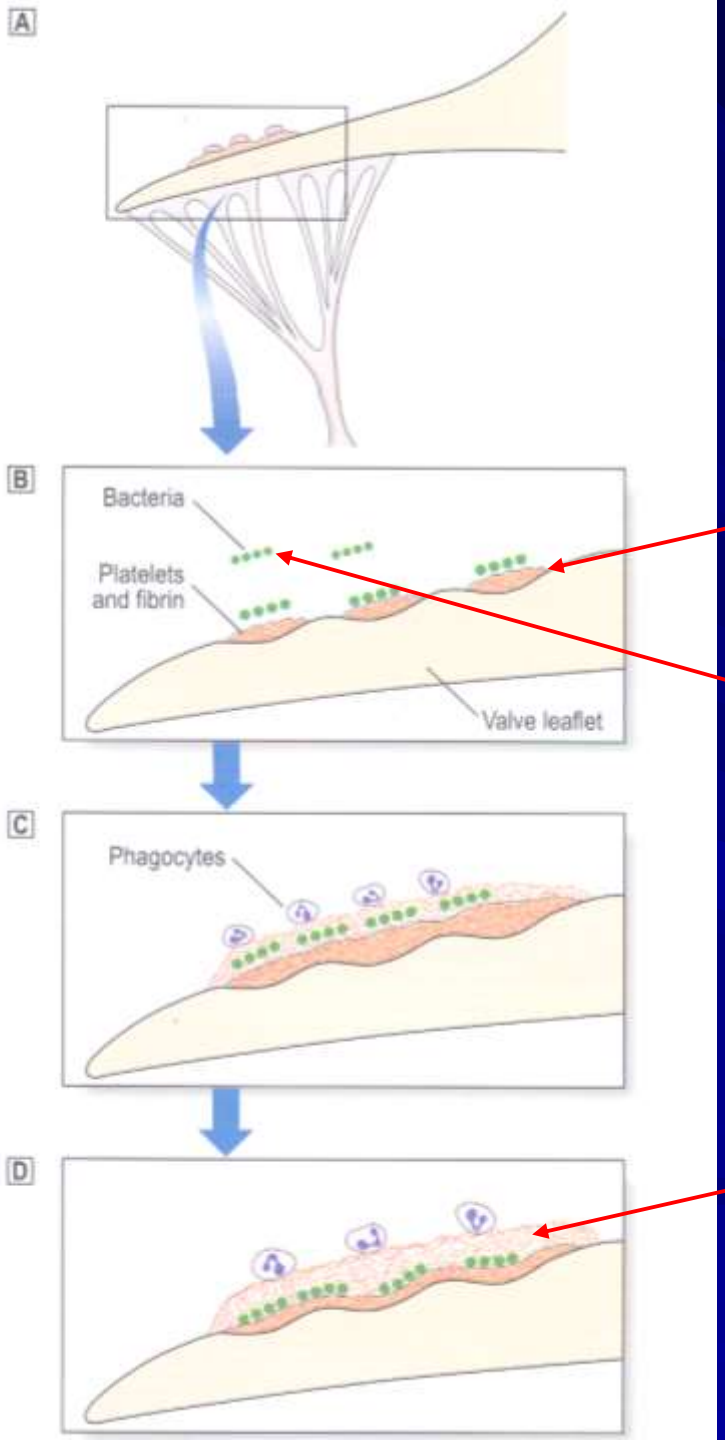
Endothelial damage

↓

Platelet-fibrin thrombi

↓

Microorganism adherence



Turbulent blood flow traumatises endothelium

Nonbacterial thrombotic endocarditis

Bacteraemia

Further deposition of fibrin and platelets

Underwood 2004

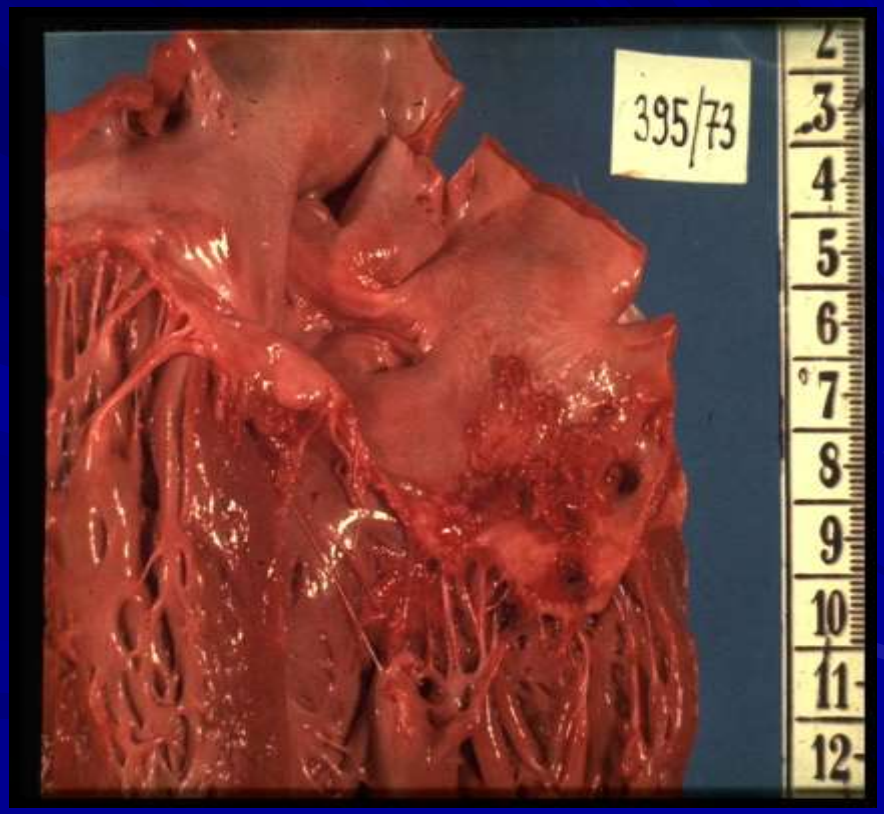
Pathophysiology

- Clinical manifestations
  - Direct
    - Constitutional symptoms of infection (cytokine)
  - Indirect
    - Local destructive effects of infection
    - Embolization – septic or bland
    - Hematogenous seeding of infection
      - N.B. may present as local infection or persistent fever, metastatic abscesses may be small, miliary
    - Immune response
      - Immune complex or complement-mediated

Infective Endocarditis

Outcome

- Fatal 10-70% of cases



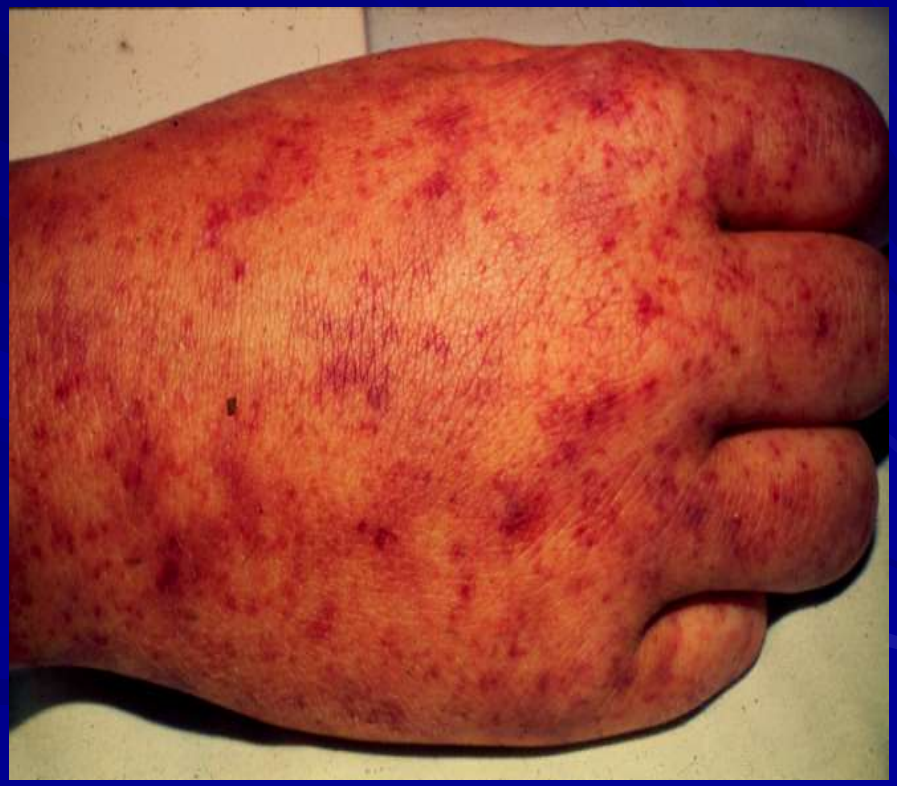
Clinical Features

- Malaise, fever, night sweats, weight loss, anaemia, Chills, Anorexia, Arthralgia

- Valve destruction → heart failure
  → new/changing murmurs
- Embolic events → abscesses in brain, liver
- Immune complex deposition → vasculitis
  → arthralgia
  → glomerulonephritis

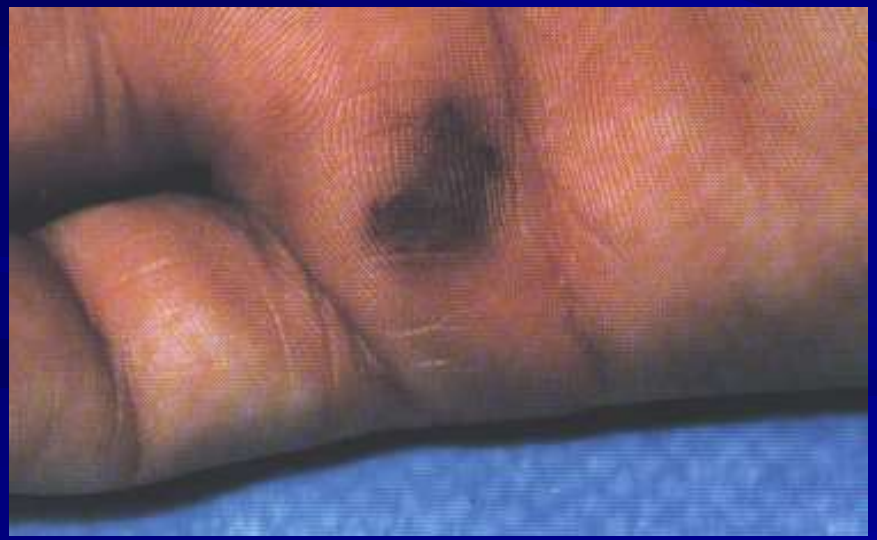
Infective Endocarditis

- Petechial Hemorrhages
- Linear Hemorrhages
- Osler Nodes
- Janeway Lesions
- Retinal Hemorrhages
- Splenomegaly



Peripheral Manifestations

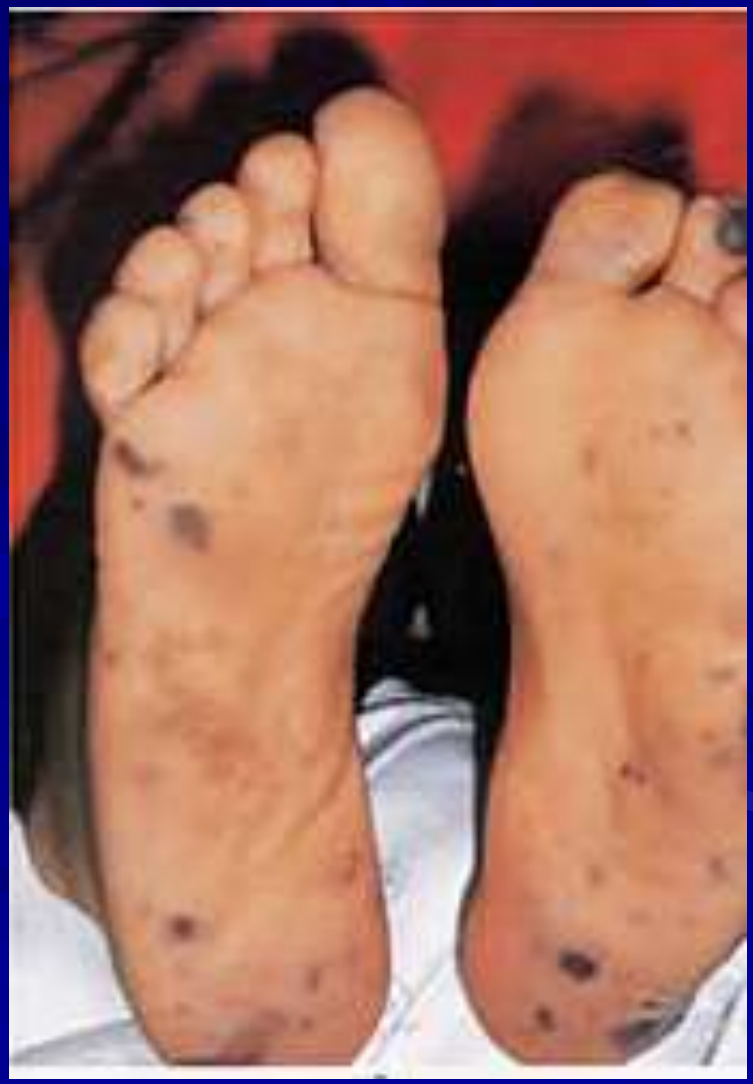
- Janeway Lesions:
  - erythematous, macular, non tender.
  - septic emboli?



- Osler's Nodes:
  - Tender, subcutaneous nodules.
  - 4 P's:
    - Pink
    - Painful
    - Pea-sized
    - Pulp of the fingers/toes.
  - Immunologic origin?

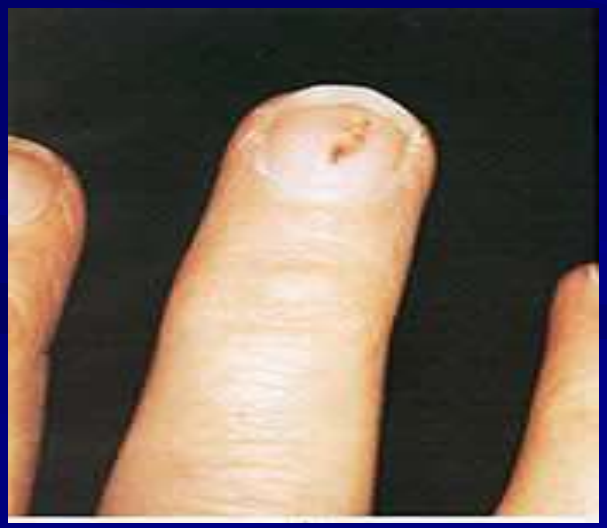
Osler's Nodes

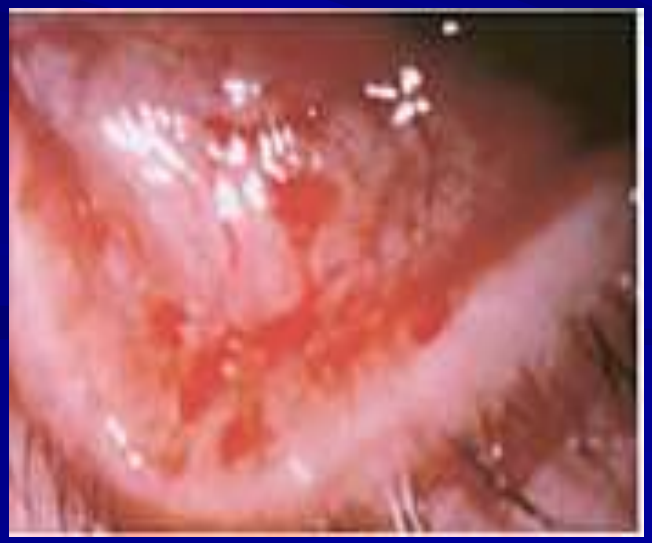
Janeway Lesions

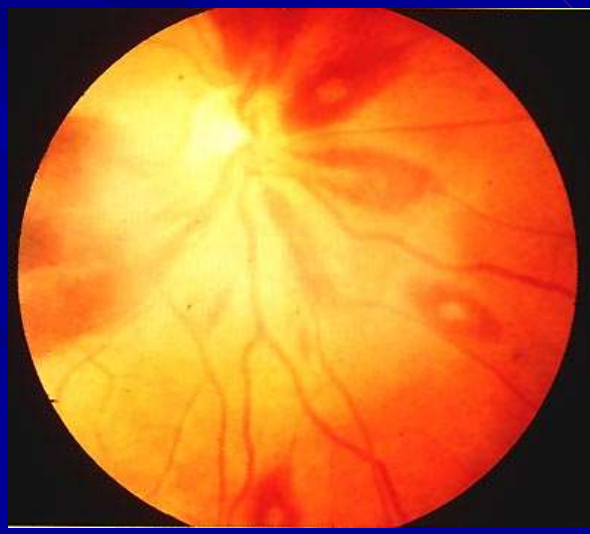


Bleeding

- Subungual (splinter) hemorrhage
- Conjunctival hemorrhage
- Retinal hemorrhage: Roth Spot

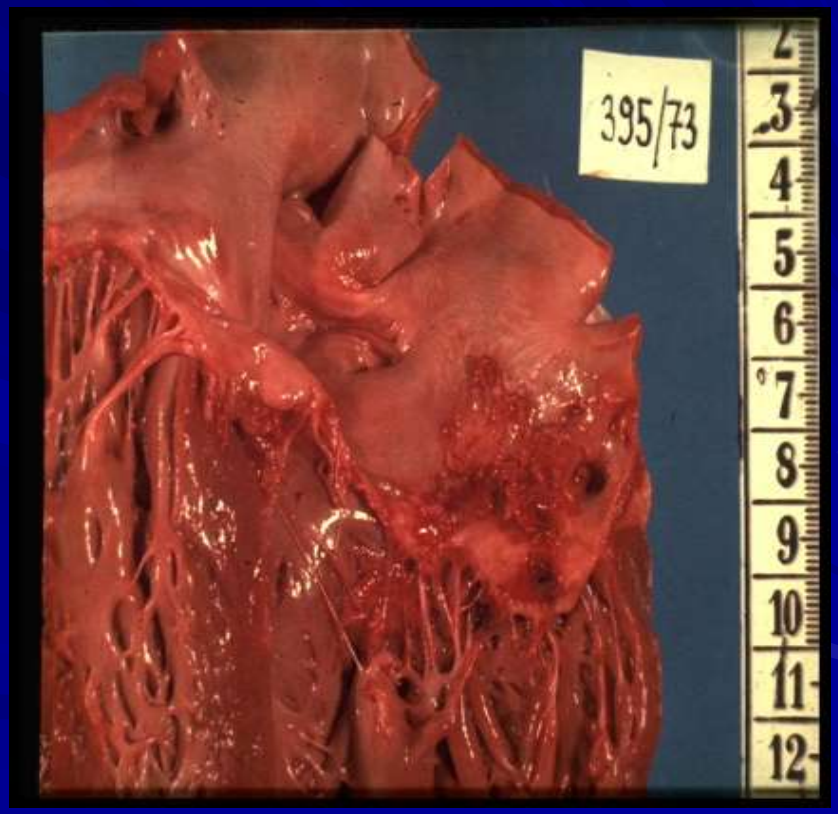






Infective Endocarditis
Complications

- Reinfection
- Systemic emboli
- Neurological sequelae
- Congestive heart failure
  - Due to mechanical disruption
- Renal insufficiency
  - Immune complex mediated
  - Impaired hemodynamics/drug toxicity



Lab Investigations

- Normochromic normocytic anaemia
- ↑ WCC – white cell count
- ↑ ESR – erythrocyte sedimentation rate
- Blood cultures – repeated samples, 3/24h
- Echocardiography

Surgical Treatment of Intra-Cardiac Complications

- Valve dysfunction
- Unstable prosthetic valve
- Uncontrolled infection
- Unavailable effective antimicrobial therapy
  - Fungal endocarditis
  - Brucella
- *S. aureus* PVE with any intra-cardiac complication
- Relapse of PVE after optimal therapy

Questions