Infective Endocarditis

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Outline

1. Introduction
2. Pathogenesis & risk factors
3. Classification systems
4. Microbial Etiology
5. Clinical presentation
6. Diagnosis and management

What is Infective Endocarditis?

- Infective endocarditis (IE) is **a microbial infection** of the inner lining of the heart **(endocardium)**, most commonly affecting the heart valves.

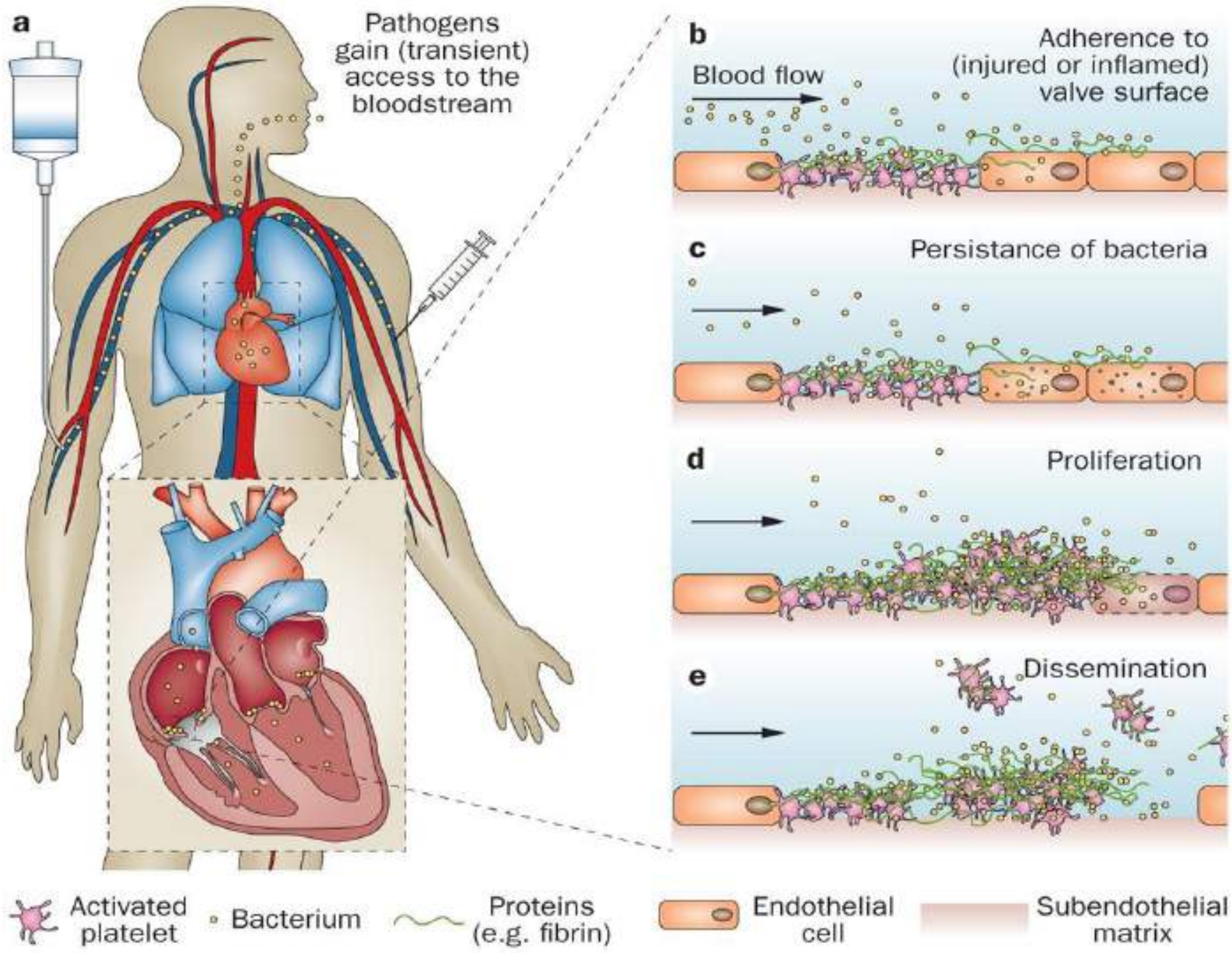
- **Epidemiology:**
  - Incidence: 11–15 cases per 100,000 persons per year
  - Mean age: 60.8 years (> 50% are > 50 years of age)
  - 3 times more common in men

Risk Factors

- **Cardiac Risk Factors:**
  - Rheumatic heart disease
  - Valvular abnormalities, Congenital defects
  - Presence of a prosthetic valve
  - Previous history of endocarditis
- **Patient Risk Factors:**
  - Age > 60 years
  - IV drug use (most commonly affects the tricuspid valve)
  - Poor dentition
  - Immunosuppression
- **Iatrogenic Risk Factors** → Implanted devices or catheters

Infective endocarditis: Pathophysiology

- Infective endocarditis: Predisposing factors:
  - Endocardial abnormality or injury
  - Bacteraemia
- Damaged endothelium → platelet and fibrin deposition → adherence by microorganisms
- Proliferation and invasion by organisms → inflammation → vegetation development → valve destruction
- Release of septic emboli → embolic complications and/or metastatic infection


a
Pathogens gain (transient) access to the bloodstream
b
Blood flow
Adherence to (injured or inflamed) valve surface
c
Persistance of bacteria
d
Proliferation
e
Dissemination
Activated platelet
Bacterium
Proteins (e.g. fibrin)
Endothelial cell
Subendothelial matrix

Classification: By clinical course

Infective endocarditis can be further classified based on the

- Clinical course
- Type of valve
- Location

Classification: By clinical course

Acute infectious endocarditis:

- **Onset and Progression:** More sudden onset of symptoms → Progresses more rapidly → Fatal if not treated promptly
- **Vegetations:** Larger vegetations
- **Organism virulence:** HIGH (aggressive pathogens) → More commonly affects normal valves
- Most common cause is *S. aureus*
- Other causes include *Streptococcus pneumoniae*, *Streptococcus pyogenes* (Group A Strep)

Classification: By clinical course

Subacute infectious endocarditis:

- More gradual onset of symptoms → Progresses more slowly (weeks to months) → Patients may survive for months untreated.
- Smaller vegetations
- Organism virulence: LOW to MODERATE → More commonly affects congenitally abnormal or diseased valves
- Most common cause is *S. viridans*.

Classification: By clinical course

Chronic infectious endocarditis:

- **Onset:** Months to years
- **Organism virulence:** Very low, intracellular
- **Typical Organisms:** *Coxiella burnetii*, *Bartonella* species

Classification: By valve type

Native valve endocarditis:

- Accounts for 78% of cases
- Further subdivided into:
  - Community acquired (most common)
  - Healthcare associated
  - IV drug use
- Most often associated with: Staphylococcus, Streptococcus, and HACEK organisms

Classification: By valve type

Prosthetic valve endocarditis:

- Early:
  - < 60 days after valve placement
  - Often from contamination during surgery
- Intermediate: 60–365 days after valve placement
- Late:
  - > 1 year after valve placement
  - Usually involves similar organisms to native valve endocarditis
  - Associated with a higher risk of complications and mortality

Classification: By location

Left-sided endocarditis:

- Left-Sided IE is more common:
  - More turbulent flow → more shear stress on valves → more endothelial microtrauma
  - Most pre-existing valve abnormalities affect the left side
- Mitral valve > Aortic valve
- Complications: Left-sided IE leads to **systemic emboli**
  - Brain → Stroke
  - Kidneys → Renal infarcts, glomerulonephritis

Classification: By location

Right-sided endocarditis

(most common in IV drug use):

- Tricuspid valve (90%) > Pulmonic valve (10%)
- Right-sided IE risk factors:
  - IV drug use – 70-80% of right-sided IE occurs in IV drug users
  - Indwelling venous catheters (central lines)
- Complications: Right-sided IE leads to **pulmonary emboli** (not systemic)

Infective Endocarditis Etiology: Staphylococci

General features:

- Gram stain: Gram-positive cocci in clusters ("grape-like")
- Non-motile, non-spore forming
- Facultative anaerobe
- Catalase positive: differentiates from Streptococcus

A. *Staphylococcus aureus* (most common)
   - Coagulase positive: differentiates from CoNS
   - β-haemolytic (complete haemolysis)

B. Coagulase-negative staphylococci (cons)
   - Main Species: *Staphylococcus epidermidis*
   - Coagulase negative
   - Non-hemolytic or weakly hemolytic

Infective Endocarditis Etiology: Streptococci

General features:

- Gram-positive cocci in CHAINS or PAIRS
- Non-motile, non-spore forming
- Catalase negative

<u>Viridans group streptococci</u>

- **α-haemolytic** (partial haemolysis)
- Optochin Resistant: Differentiates from *S. pneumoniae* (sensitive)
- Bile solubility: Negative → Differentiates from S. pneumoniae (positive)

Infective Endocarditis Etiology: HACEK Group

HACEK organisms – a group of fastidious gram-negative bacteria that cause about 5% of IE cases.

HACEK Acronym:

- **H:** *Haemophilus parainfluenzae, H. aphrophilus*
- **A:** *Aggregatibacter* (*Actinobacillus*) *actinomycetemcomitans*
- **C:** *Cardiobacterium hominis*
- **E:** *Eikenella corrodens*
- **K:** *Kingella kingae*

Clinical Presentation: General signs and symptoms

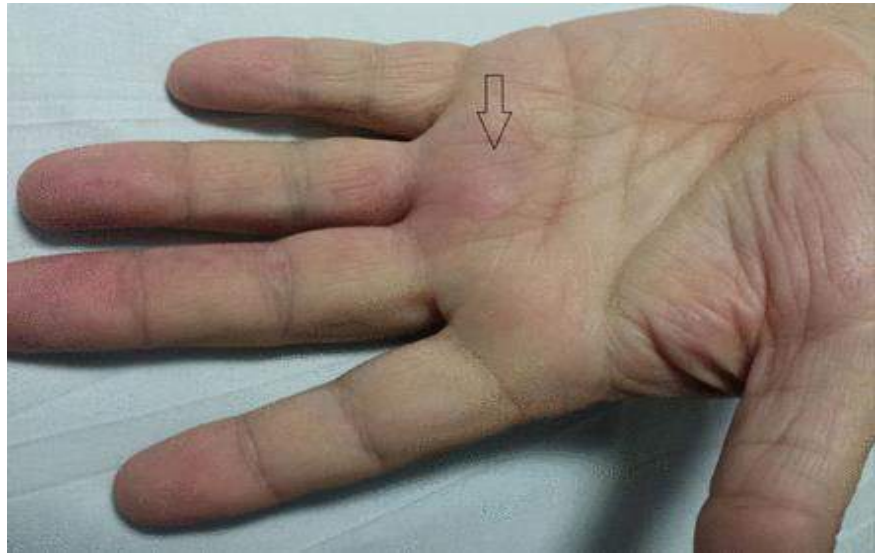
- Presentation and course depend on the etiology, location of vegetations, and severity.
- **Fever:** (endocarditis should be suspected in a patient with a fever of unknown origin)
- Night sweats
- Fatigue
- Loss of appetite
- Weight loss
- Myalgias and arthralgias

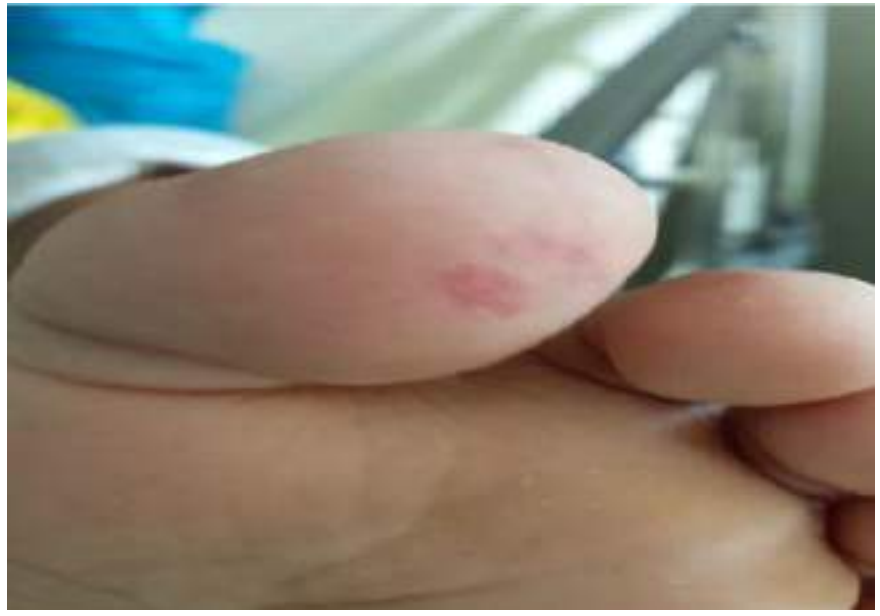
Clinical Presentation: Cardiac findings

- **New or changed cardiac murmur**:
  - Mitral regurgitation, Tricuspid regurgitation, Aortic regurgitation
- **Tachycardia**
- **Arrhythmia:**
  - Potentially due to spread of the infection
  - Results in disruption of the atrioventricular conduction system → conduction delay or heart block

Clinical Presentation: Extracardiac Findings

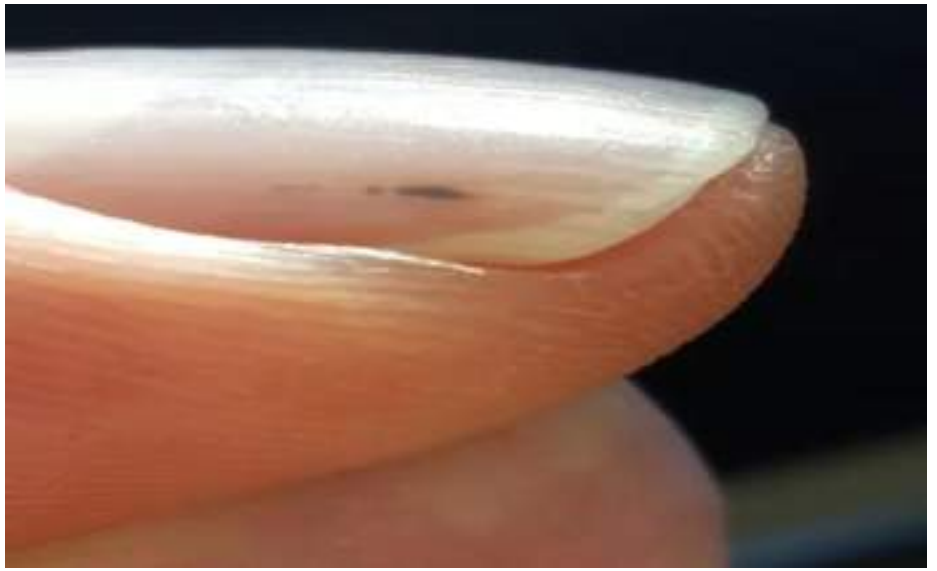
- **Osler nodes:**
  - Painful red nodules on pads of the fingers and toes
  - Due to immune complex deposition and inflammation
- **Janeway lesions:**
  - Small, painless, erythematous lesions on the palms or soles
  - Due to septic emboli and microabscesses

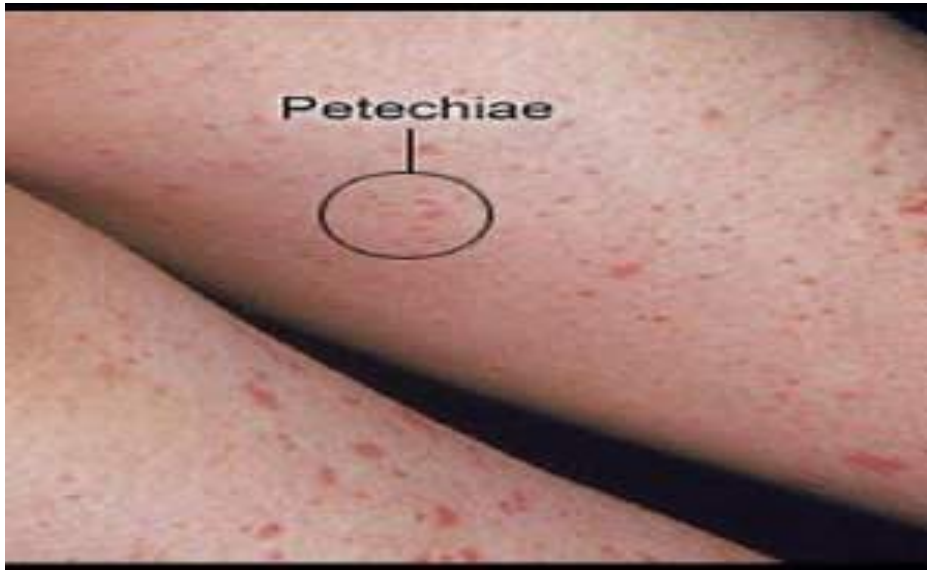




Clinical Presentation: Extracardiac Findings

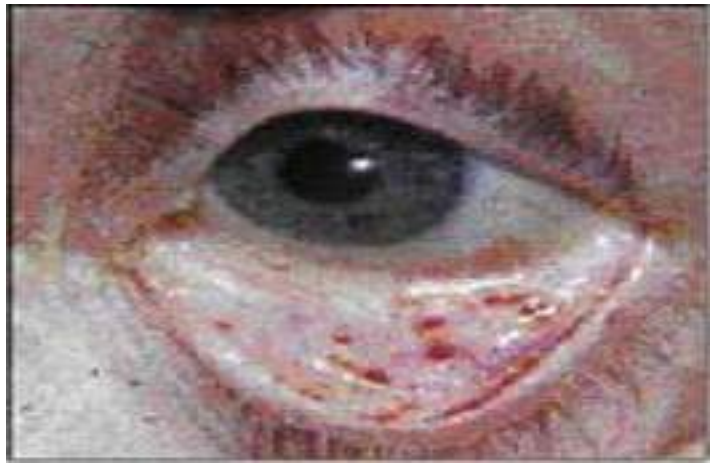
- **Splinter haemorrhage:**
  - Small areas of red discoloration under the nails
  - Due to micro emboli in capillaries
- **Petechiae:** Small, pinpoint, non-blanching, red-purple spots on the skin or mucous membranes.

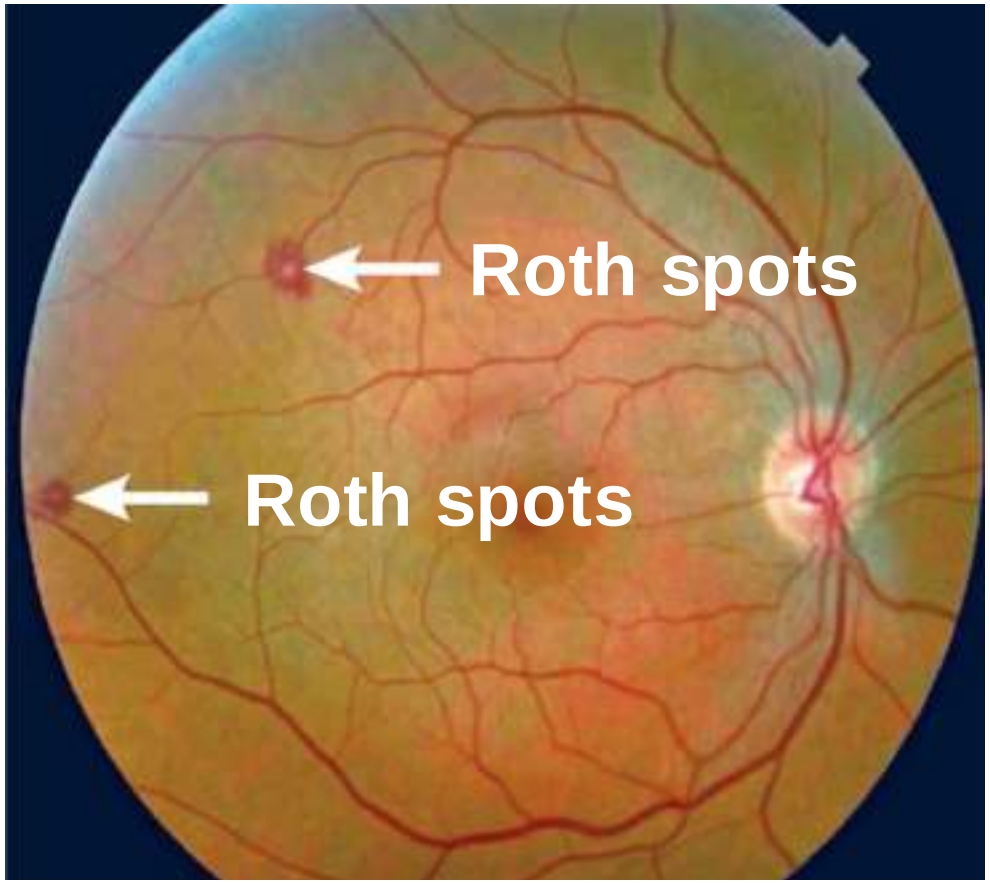




Clinical Presentation: Extracardiac Findings

- **Roth spots:**
  - Red spots with pale centers on fundoscopic exam
  - Due to retinal haemorrhages
- **Conjunctiva haemorrhage**

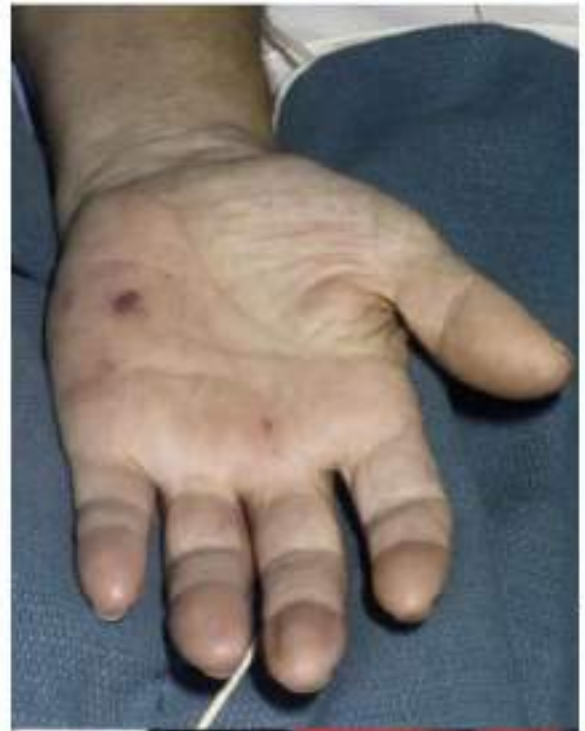


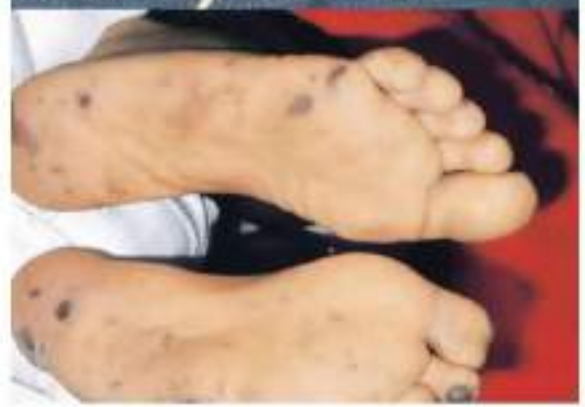


Splinter hemorrhages

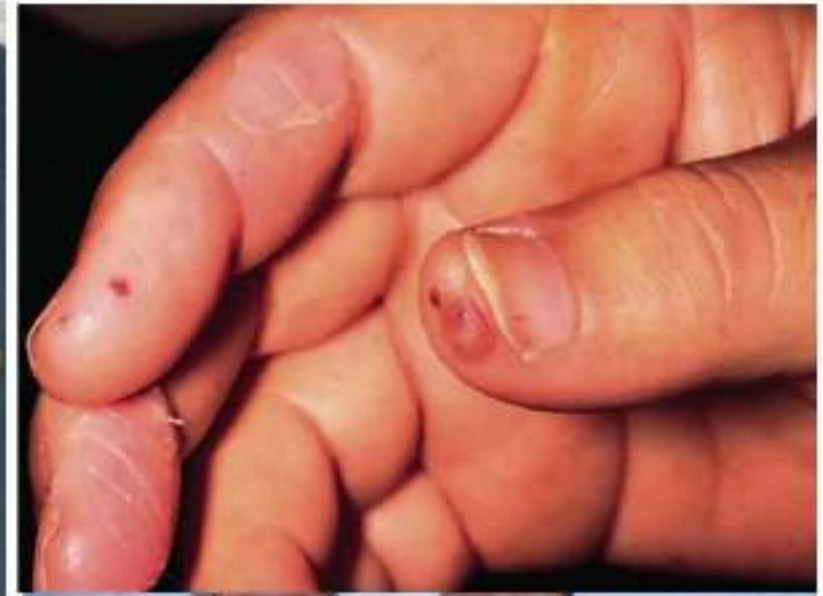
Conjunctival hemorrhages

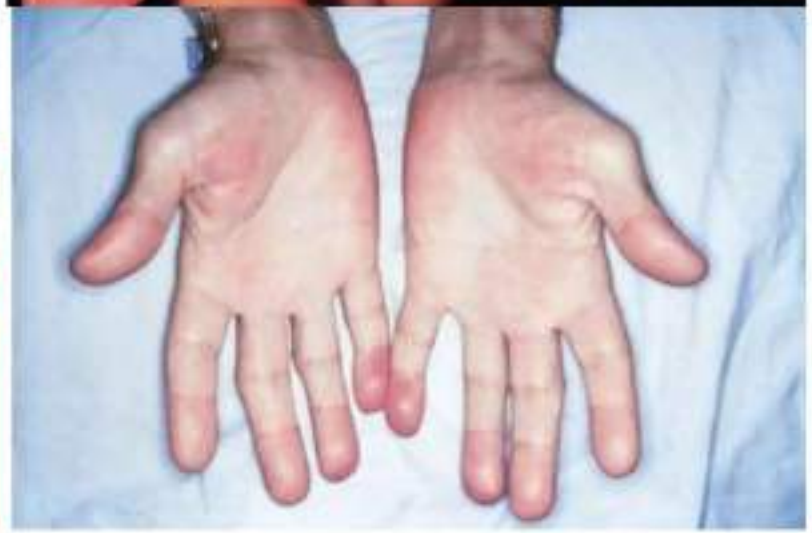
Janeway lesions
(painless spots on palms/soles of feet)





Osler's Nodes
(painful nodules in pulp of fingers/toes)





Diagnosis: Duke diagnostic criteria

- The Duke diagnostic criteria is a set of clinical criteria that can aid in the diagnosis of IE.
- Must meet 1 of the following for a definitive diagnosis of IE:
  - 2 major criteria, or 1 major plus 3 minor criteria, or 5 minor criteria
- **Major criteria**:
  - Positive blood cultures (1 of the following):
    - Typical organism for IE in 2 separate blood cultures
  - Findings of endocardial involvement (1 of the following):
    - By echocardiogram
    - New regurgitation murmur

Diagnosis: Duke diagnostic criteria

- **Minor criteria**:
  - Fever > 38°C
  - Risk factors: IV drug use, or Predisposing heart condition
  - Vascular findings:
    - Major arterial emboli
    - Janeway lesions
    - Conjunctival haemorrhages
    - Septic infarcts
  - Immunologic findings: Osler nodes, Roth spots, Glomerulonephritis
  - Microbiologic findings by culture that do not meet major criteria

Management

Antibiotic therapy:

- Prolonged IV therapy: Typically, 4-6 weeks (native valve) or 6-8 weeks (prosthetic valve)
- Bactericidal antibiotics
- Start empiric therapy while waiting for blood culture results, initial therapy typically vancomycin

Surgical management

Valve repair or replacement is indicated for: Heart failure, Persistent infection, Embolic events, Prosthetic valve dehiscence

Thank you