Infective Endocarditis

- Most serious of all infections- febrile illness, persistent bacteremia
- Most cases are bacterial, some fungi, rickettsiae, chlamydiae

Bacterial Endocarditis

- Serious infection of valvular and mural endocardium caused by different organisms and is characterised by infected and friable vegetations associated with destruction of the underlying tissues
- Typically involves the valves, may involve chordae tendinae, sites of shunting or mural lesions
- Divided into 2 clinical forms:

  Acute bacterial endocarditis

  Subacute bacterial endocarditis

Etiology

ABE:

- Staph aureus
- Gonococci
- Pneumococci
- Strep and enterococci

SABE: organisms are those with low virulence or commensals

- Strep viridans (present in oral cavity)
- Staph epidermidis (skin)
- Hacek group (Haemophillus, Actinobacilus, Cardiobacterium, Eikenella, and Kingella)

10% culture negative

Etiology

- Acute
  - Toxic presentation
  - Progressive valve destruction & metastatic infection developing in days to weeks
  - Previously normal heart valve
  - Most commonly caused by S. aureus
- Subacute
  - Mild toxicity
  - Presentation over weeks to months
  - Rarely leads to metastatic infection
  - Most commonly S. viridans or enterococcus

  - 55-75% of patients have underlying valve abnormalities- Rheumatic, Congenital, prosthetic, myxomatous mitral valve

	Acute	**Subacute**
Duration	<6 wks	>6 wks-months-yrs
Organism	Staph aureus B Strep	Strep viridans
Virulence	Highly (+++)	Less (+)
Previous Valves	Normal	Damaged
Lesions	Invasive, destructive, suppurative	Not invasive or suppurative
Clinical features	Acute systemic infection; 50% fatal	Splenomegaly, clubbing, petechiae

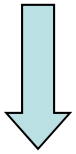
Predisposing factors

- Bacteremia, pyemia, and septicemia: Transient and clinically silent entry of bacteria into the blood stream
  - periodontal infections
  - genito-urinary infections
  - skin infections
  - I.V drug abuse (rt side valves, staph aureus)
  - respiratory tract infections
- Underlying heart disease: SABE occurs much more frequently in previously damaged valves- RHD, CHD
- Impaired host defenses- lymphomas, leukemias, chemotherapy and transplant patients

Pathogenesis

Endothelial damage

Platelet-fibrin thrombi



Microorganism adherence

Pathologic changes

Gross - Friable, bulky and destructive vegetations containing fibrin, inflammatory cells and bacteria are present on the heart valves

- Involve one or more valves, commonly mitral and aortic
- Atrial surface of AV valves, ventricular surface of semilunar valves
- Size of vegetation few mm-cms, depends on organism, antibiotic use, degree of host reaction
- Grey tan, irregular, single or multiple, flat, filiform or fungating
- Erode into surrounding myocardium to produce abscess (ring abscess)

M/E: 3 zones

- Outer cap consisting of eosinophilic material (fibrin, platelets)
- Basophilic zone of colonies of bacteria
- Non specific inflammation

ABE- neutrophils, tissue necrosis

SABE- vegetations cause less destruction, simultaneous healing by granulation tissue, mononuclear cells, later fibrosis, calcification

Clinical Features

- Interval between bacteremia & onset of symptoms usually < 2 weeks
- ABE: Fever is the most consistent sign with rapidly developing chills, weakness and lassitude
- SABE: fever may be slight or absent, nonspecific fatigue, loss of weight, flu-like illness, may be absent in elderly/debilitated patient
- **Cardiac:**

  - Regurgitation

  - CHF

  - Murmur present in 80 – 85%, indication of underlying lesion

Complications

Cardiac

- valvular stenosis or insufficiency
- Perforation, rupture
- Paravalvular abscess
- Conduction abnormalities
- Purulent pericarditis

Complications

Extra cardiac:

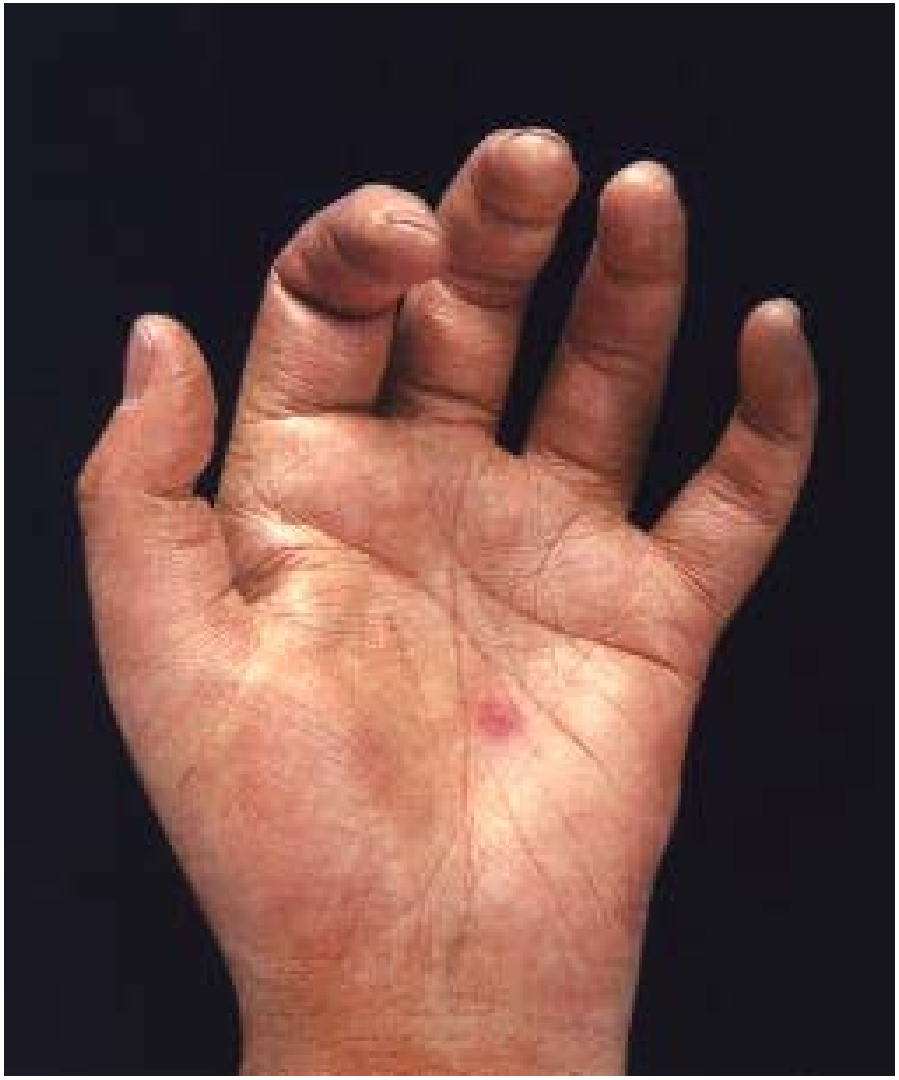
- Embolization: High risk for embolization
  - » Large > 10 mm vegetation
  - » Hypermobile vegetation
  - » Mitral vegetations (esp. anterior leaflet)

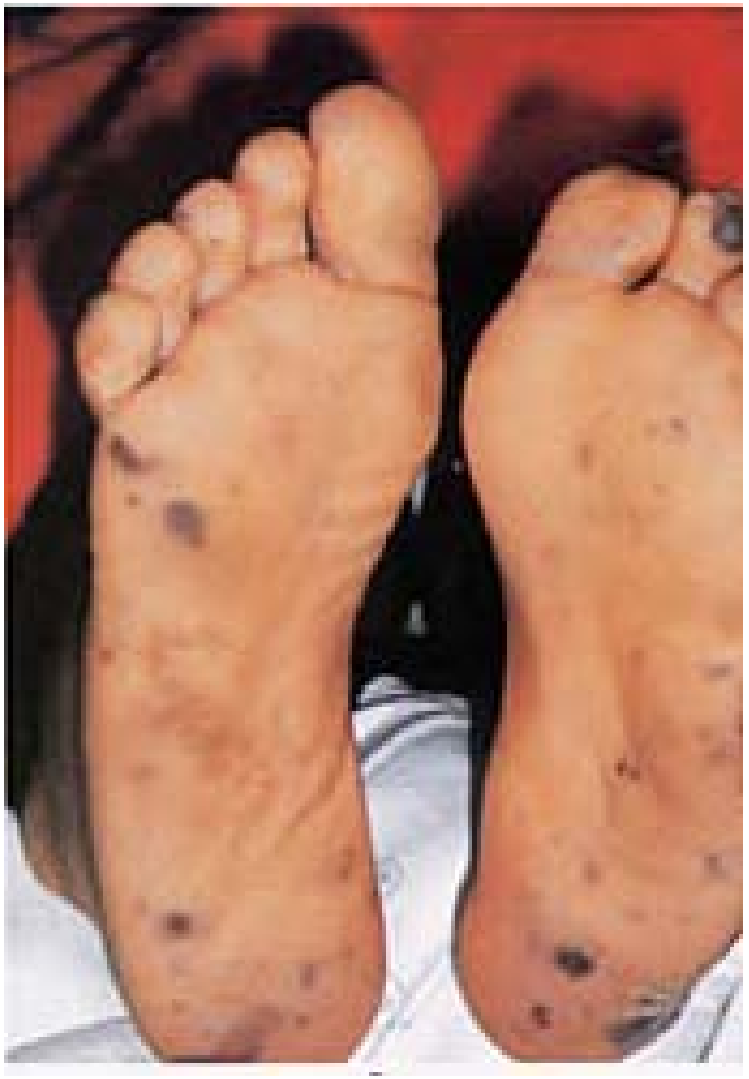
Pulmonary (septic) – 65 – 75% of i.v. drug abusers with tricuspid IE

Systemic emboli may occur anytime d/t friable nature → cause infarcts of brain, spleen, kidney, myocardium which are infected (Septic infarcts)

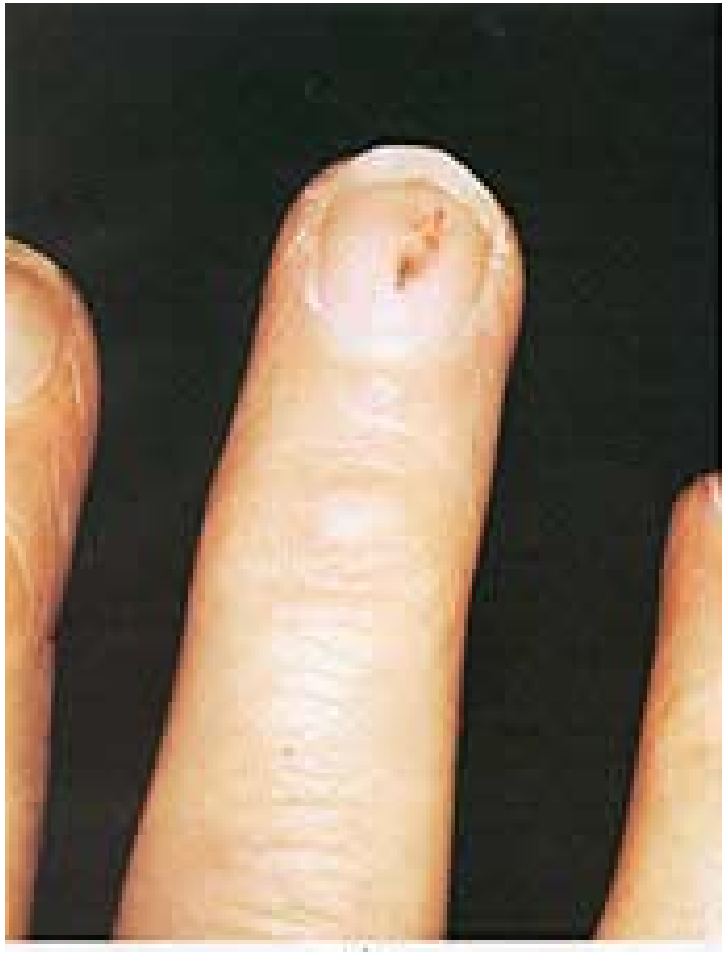
- Secondary to microthrombi: Osler's nodes- s/c nodules in the pulp of digits
- Roth spots: retinal hemorrhages
- Janeways lesions: erythematous/ hemorrhagic lesions on palms and soles
- Splinter/subungual haemorrhages
- Renal: glomerulonephritis occcur after 1 week, immunologically mediated d/t trapping of Ag-Ab complexes

Janeway Lesions

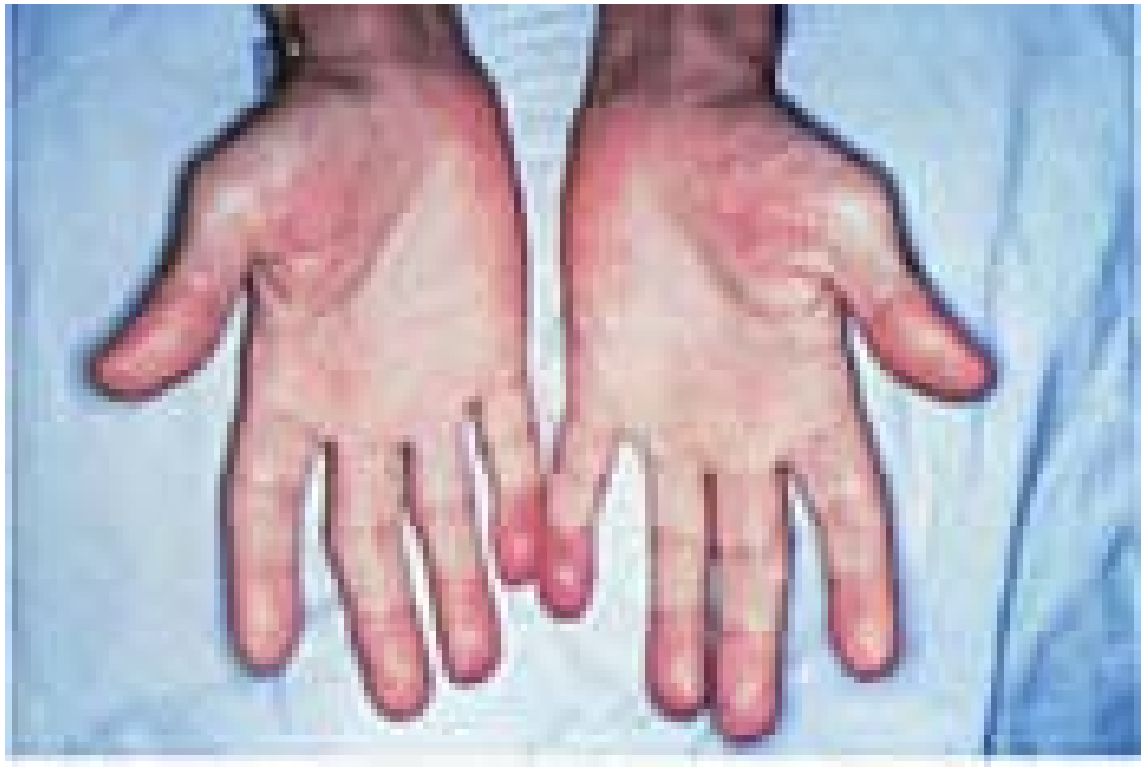




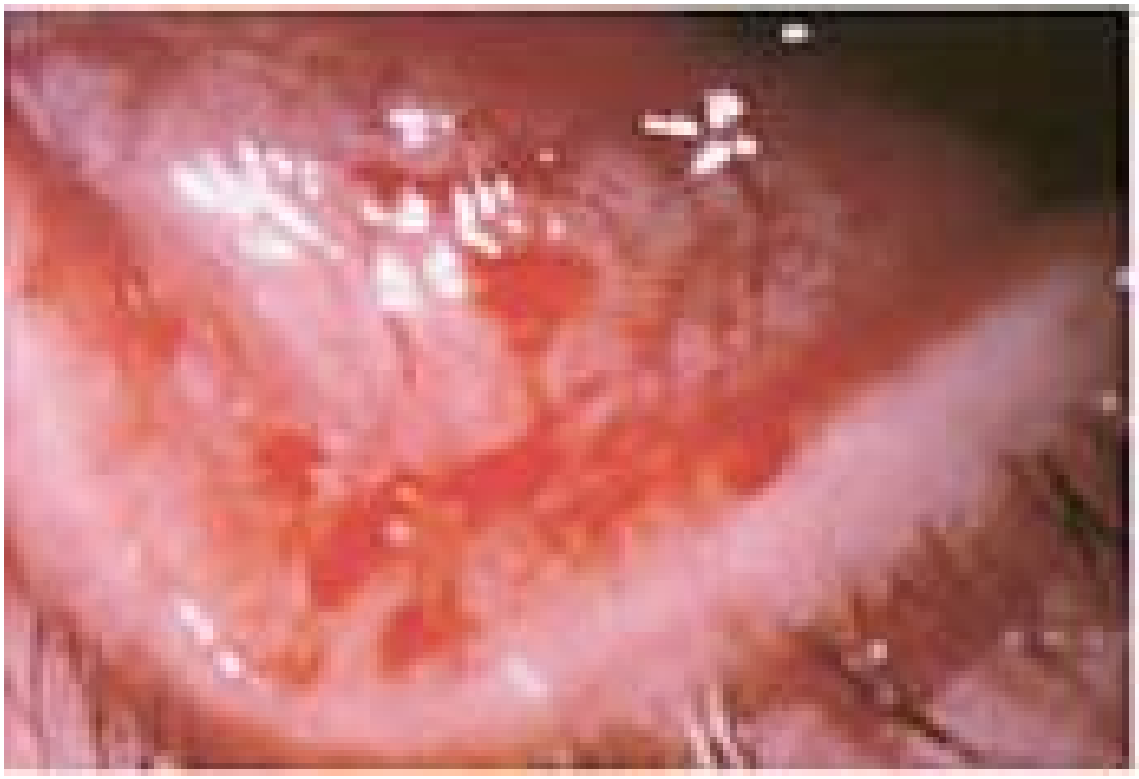
Splinter Hemorrhage



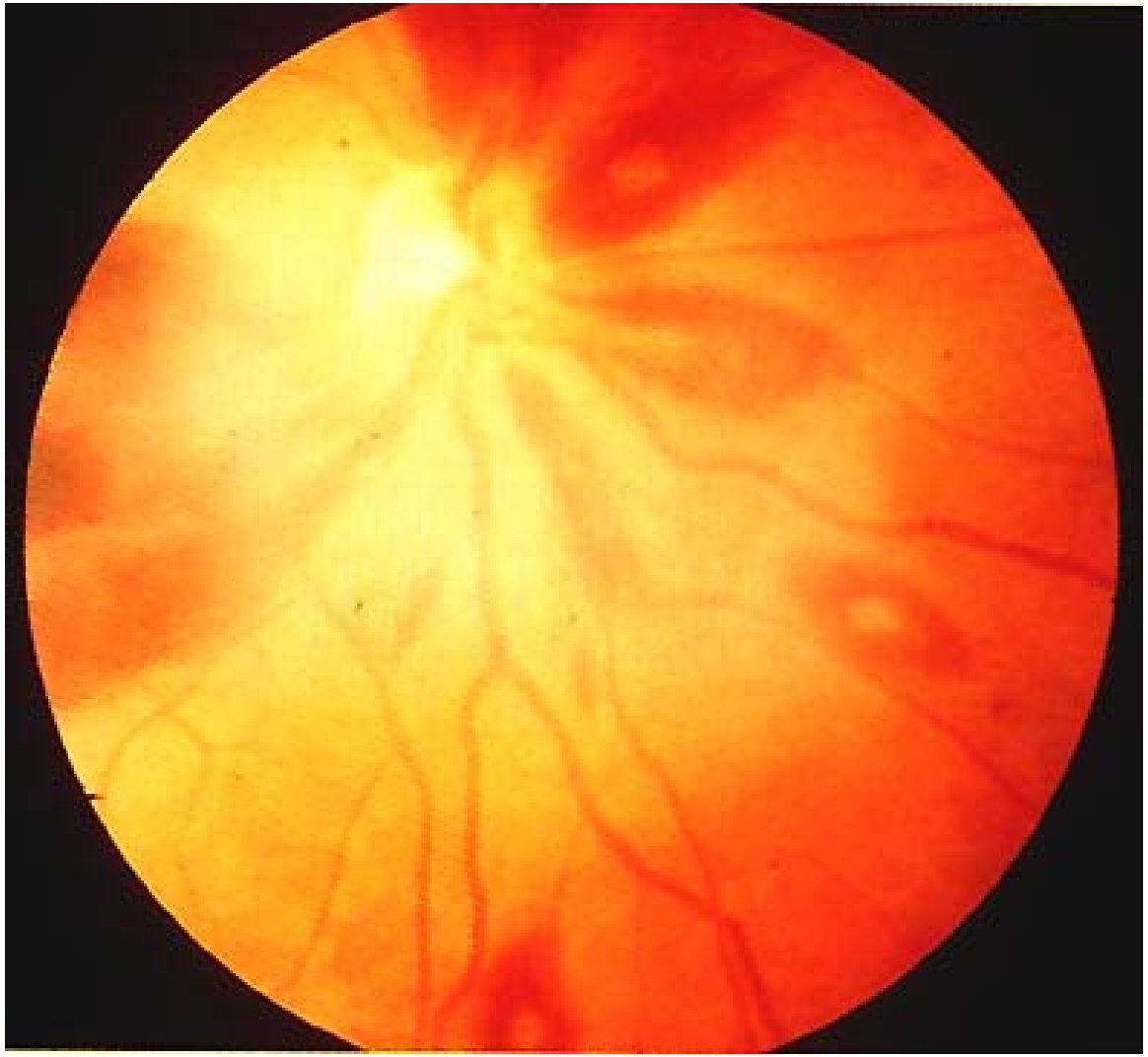
Osler’s Nodes



Subconjunctival Hemorrhages



Roth’s Spots



Dukes Criteria for diagnosis of IE

Pathologic:

- Microorganism demonstrated by culture or histologically from vegetation/ intracardiac abscess/ embolus
- Histologic confirmation of active endocarditis in vegetation/ abscess

Clinical criteria

Major

- Positive blood culture
- Echocardiographic findings: valve related mass/ abscess
- New valvular regurgitation

Dukes Criteria for diagnosis of IE

Minor

- Predisposing heart lesions/ I.V drug abuse
- Fever
- Vascular lesions: petechiae, hemorrhages, septic infarcts, mycotic aneurysms
- Immunologic phenomena: GN, RF
- Echocardiographic findings consistent but not diagnostic of IE
- Microbiologic evidence of single culture showing uncharacteristic organism

(2 major, 1 major+ minor, 5 minor)

- Causes of death
  - cardiac failure
  - embolism
  - renal failure