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 (Haemophilus, 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
<table>
<thead>
<tr>
<th></th>
<th>Acute</th>
<th>Subacute</th>
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<tbody>
<tr>
<td><strong>Duration</strong></td>
<td>&lt;6 wks</td>
<td>&gt;6 wks-months-yrs</td>
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<tr>
<td><strong>Organism</strong></td>
<td>Staph aureus</td>
<td>Strep viridans</td>
</tr>
<tr>
<td></td>
<td>B Strep</td>
<td></td>
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<tr>
<td><strong>Virulence</strong></td>
<td>Highly (+++)</td>
<td>Less (+)</td>
</tr>
<tr>
<td><strong>Previous Valves</strong></td>
<td>Normal</td>
<td>Damaged</td>
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<tr>
<td><strong>Lesions</strong></td>
<td>Invasive, destructive,</td>
<td>Not invasive or suppurative</td>
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<tr>
<td></td>
<td>suppurative</td>
<td></td>
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<tr>
<td><strong>Clinical features</strong></td>
<td>Acute systemic infection; 50% fatal</td>
<td>Splenomegaly, clubbing, petechiae</td>
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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
• Janeway’s lesions: erythematous/ hemorrhagic lesions on palms and soles
• Splinter/subungual haemorrhages
• Renal: glomerulonephritis occur 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