

- Pathology of Endocarditis

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- **Endocarditis** includes: 1- Infective endocarditis (IE)  
2- nonbacterial thrombotic

endocarditis

3- Libman - Sacks endocarditis:

- ***1- Infective endocarditis:***

- is a serious infection requiring early diagnosis and intervention

- is characterized by: 1- microbial invasion of endocardium (valves)

- 2- destruction of underlying cardiac tissues

- 3- formation of bulky friable bacterial vegetations (microorganisms, fibrin, platelets and inflammatory cells)

- is caused by: 1- bacteria (majority of cases)

- 2- fungi

- is classified into:

- 1- Acute: - infection by highly virulent organisms (S. aureus, beta hemolytic streptococci, pneumococci)

- occurs in previously normal heart

- causes death in more than 50% of patients despite therapy

- rapidly developing fever with rigors, malaise

- embolic complication is common

2- Subacute: - infection by low virulent organisms (St. viridans, enterococci)

- occurs in previously abnormal heart
- most patients recovering after therapy
- malaise, low grade fever, flu-like

symptoms

- embolic complication less common

- **Pathogenesis:**

- blood-borne bacteria reach the valvular endocardium, from:

- a) infections elsewhere in the body

- b) intravenous drug abuse

- c) dental or surgical procedures

- damage to endocardium, exposure of subendothelium connective tissue to blood, formation of (sterile) small thrombi

- Bacterial invasion of thrombi and bacterial vegetations formation

- The vegetations may: 1- erode into underlying myocardium (ring abscess)

- 2- detach and impact distant sites (septic emboli = septic infarct)

- neutropenia, immunodeficiency, malignancy, immunosuppression therapy, DM, prosthetic valves, cardiac catheter increase the risk of IE

- **Morphology:**

- friable bulky vegetations are present on valves (single or multiple)

- mitral and aortic valves are most commonly involved

- tricuspid valve involved commonly in intravenous drug abuse

- **Clinical features:**

- fever

- vegetations can embolize producing abscess and infarctions in distant sites (e.g. embolic stroke, splenic and kidney infarcts etc.)

- valve destruction leads to regurgitation murmurs and CHF.

- extension of infection into heart ( abscess)

- immune complex vasculitis:

- 1- Roth's spot (hemorrhages) in retina

- 2- Splinter hemorrhages in nail beds

- 3- Osler's node (painful) on hands and feet

- 4- Janeway lesions in hand and feet (painless)

- 5- Glomerulonephritis

- valve destruction
- immune complex vasculitis: Roth's spot, Splinter hemorrhage, Osler's node, Janeway lesion
- **Investigations:**
  - 1- blood culture
  - 2- CBC (leucocytosis, increased ESR)
  - 3- echocardiography
- **Diagnosis:-** confirmed by Duke criteria (2 major, 1 major + 3 minors or 5 minors are required for diagnosis)

**Duke criteria:**

- Major:
- 1- positive blood culture
  - 2- echocardiography findings (vegetations, abscess)
  - 3- new valvular regurgitation
- Minor:
- 1- predisposing heart lesion
  - 2- intravenous drug abuse
  - 3- vascular lesions (hemorrhage, emboli)
  - 4- immunological phenomena (glomerulonephritis)
  - 5- blood culture (showing uncharacteristic organisms)
  - 6- echo findings (not diagnostic of endocarditis)

- **Complications:**
  - 1- valve regurgitation
  - 2- myocardial ring abscess or perforation
  - 3- myocarditis

- 4- congestive heart failure
- 5- arrhythmias
- 6- septicemia
- 7- glomerulonephritis and so renal failure
- 8- systemic embolization with development of septic infarct

***II- Nonbacterial thrombotic endocarditis:***

- is characterized by deposition of thrombi (fibrin, platelets, other blood components) on valves
- occurs in previously normal valves
- no microorganisms (sterile vegetations)
- not lead to valve damage
- can embolize
- Pathogenesis:
  - predisposed by:
    - hypercoagulable states:
      - 1- sepsis with DIC
      - 2- hyperestrogenic state
      - 3- underlying malignancy (mucinous adenocarcinoma)
    - endocardial trauma ( catheters)
  - the diagnosis based largely on: 1- predisposing conditions 2- embolic stroke

***III- Libman -Sacks endocarditis:***

- occurs in SLE due to immune complex deposition
- involves mitral valve
- embolization is uncommon

