* Pathology of Endocarditis
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* **Endocarditis** includes: 1- Infective endocarditis (IE)

 2- nonbacterial thrombotic endocarditis

 3- Libman - Sacks endocarditis:

 ***I- Infective endocarditis:***

 - is a serous 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