INFECTIVE ENDOCARDITIS

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OBJECTIVES

- Definition
- Types of Infective endocarditis
- MORPHOLOGIC FEATURES.

Classification of Endocarditis

A. NON-INFECTIVE

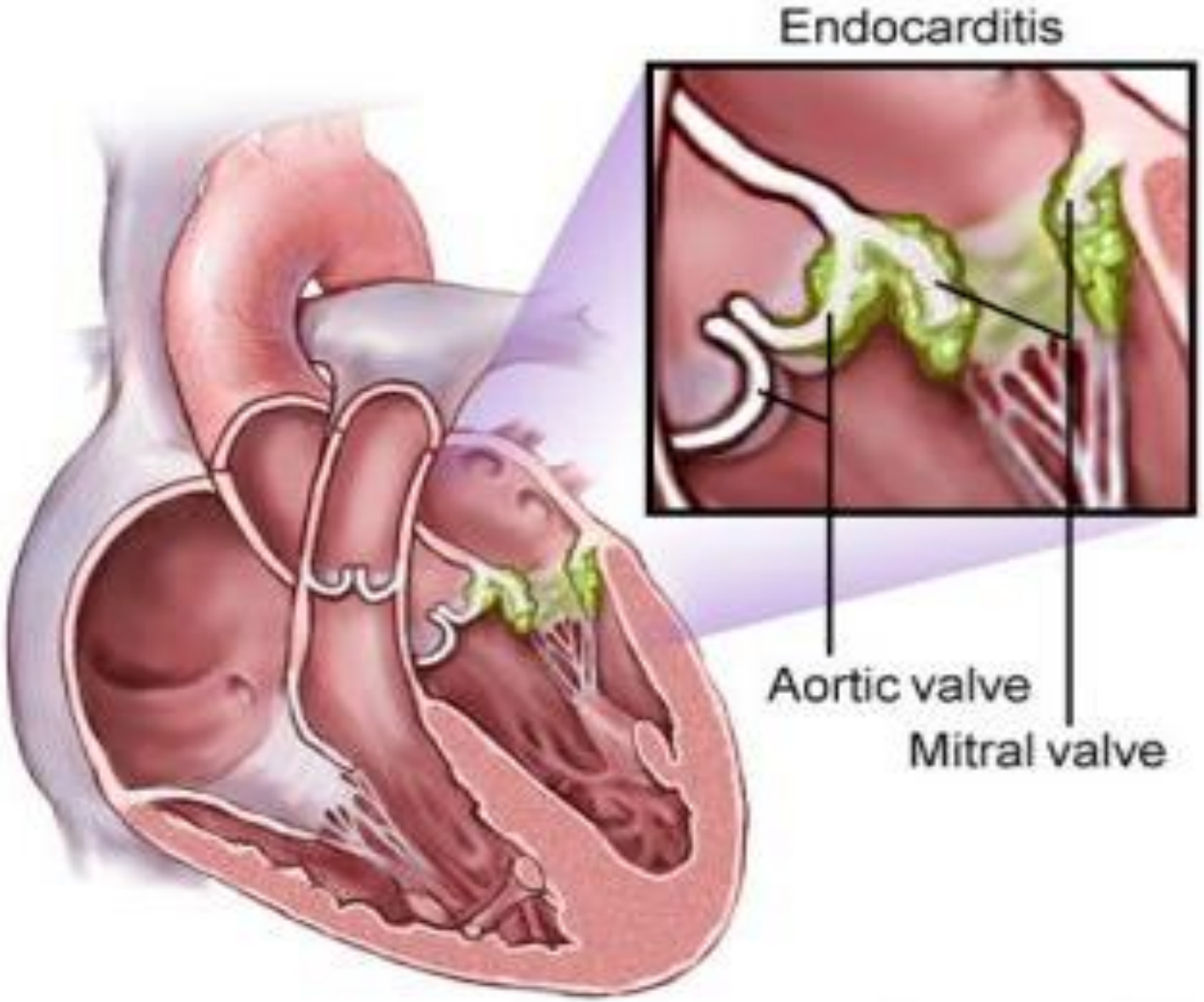
1. Rheumatic endocarditis
2. Atypical verrucous (Libman-Sacks) endocarditis
3. Non-bacterial thrombotic (cachectic, marantic) endocarditis

B. INFECTIVE

1. BACTERIAL ENDOCARDITIS
2. Other infective types (tuberculous, syphilitic, fungal, viral, rickettsial)

BACTERIAL ENDOCARDITIS

- Infective or bacterial endocarditis **(IE or BE)** is serious infection of the valvular and mural endocardium caused by different forms of microorganisms and is characterised by **typical infected** and **friable vegetation.**


Endocarditis
Aortic valve
Mitral valve

TYPES OF BACTERIAL ENDOCARDITIS (BE)

1. Acute bacterial endocarditis (ABE)

2. Subacute bacterial endocarditis (SABE) or endocarditis lenta (lenta = slow)

1) Acute bacterial endocarditis (ABE)

- This is **fulminant and destructive** acute infection of the endocardium by **highly virulent bacteria** in a previously normal heart and almost invariably runs a **rapidly fatal course** in a period of 2-6 weeks.

2)Sub-acute bacterial endocarditis (SABE)

- Endocarditis lenta (lenta = slow) is caused by less virulent bacteria in a previously diseased heart and has a **gradual downhill** course in a period of **6 weeks to a few months** and sometimes years.

	Distinguishing Features of Acute and Subacute Bacteria Endocarditis		
	Features	**Acute**	**Subacute**
1	**Duration**	< 6 weeks	> 6 weeks
2	**Most common organism**	Staph. Aureus, β-streptococci	Streptococcus Viridans
3	**Virulence of organisms**	Highly Virulence	Less Virulent
4	**Previous condition of valves**	Usually previously normal	Usually previously damaged
5	**Lesion on Valves**	Invasive, destructive, suppurative	Usually not invasive or suppurative
6	**Clinical features**	Features of acute systemic infection	Splenomegaly, clubbing of fingers, petechiae

Predisposing factors to BE

- There are 3 main factors which predispose to the development of both forms of BE:
  1. Conditions initiating transient bacteraemia, septicaemia and pyaemia
  2. Underlying heart disease.
  3. Impaired host defenses.

1. Bacteraemia, septicaemia and pyaemia

- Bacteria gain entrance to the bloodstream causing transient and clinically silent bacteraemia through many ways.
  i. **Periodontal infections** such as trauma from vigorous brushing of teeth, hard chewing, tooth extraction and other dental procedures.
  ii. **Infections of the genitourinary** tract such as in catheterisation, cystoscopy, obstetrical procedures including normal delivery and abortions.
  iii. **Infections of gastrointestinal and biliary tract.**
  iv. **Surgery** of the bowel, biliary tract and genitourinary tracts.
  v. **Skin infections** such as boils, carbuncles and abscesses.
  vi. **Upper and lower respiratory tract infections** including bacterial pneumonias.
  vii. **Intravenous drug abuse.**
  viii. **Cardiac catheterisation** and **cardiac surgery** for implantation of prosthetic valves.

2. Underlying heart disease:

- commonly associated underlying heart diseases are ;
  I. **Chronic rheumatic valvular disease** in about 50% cases.
  II. **Congenital heart diseases in** about 20% cases. These include VSD, subaortic stenosis, pulmonary stenosis, bicuspid aortic valve, coarctation of the aorta, and PDA.
  III. **Other causes are**
    - syphilitic aortic valve disease,
    - atherosclerotic valvular disease,
    - floppy mitral valve, and
    - prosthetic heart valves.

3. Impaired host defenses:

- All conditions in which there is depression of specific immunity, deficiency of complement and defective phagocytic function, predispose to BE.
  i. Impaired specific immunity in lymphomas.
  ii. Leukaemias.
  iii. Cytotoxic therapy for various forms of cancers and transplant patients.
  iv. Deficient functions of neutrophils and macrophages.

PATHOGENESIS

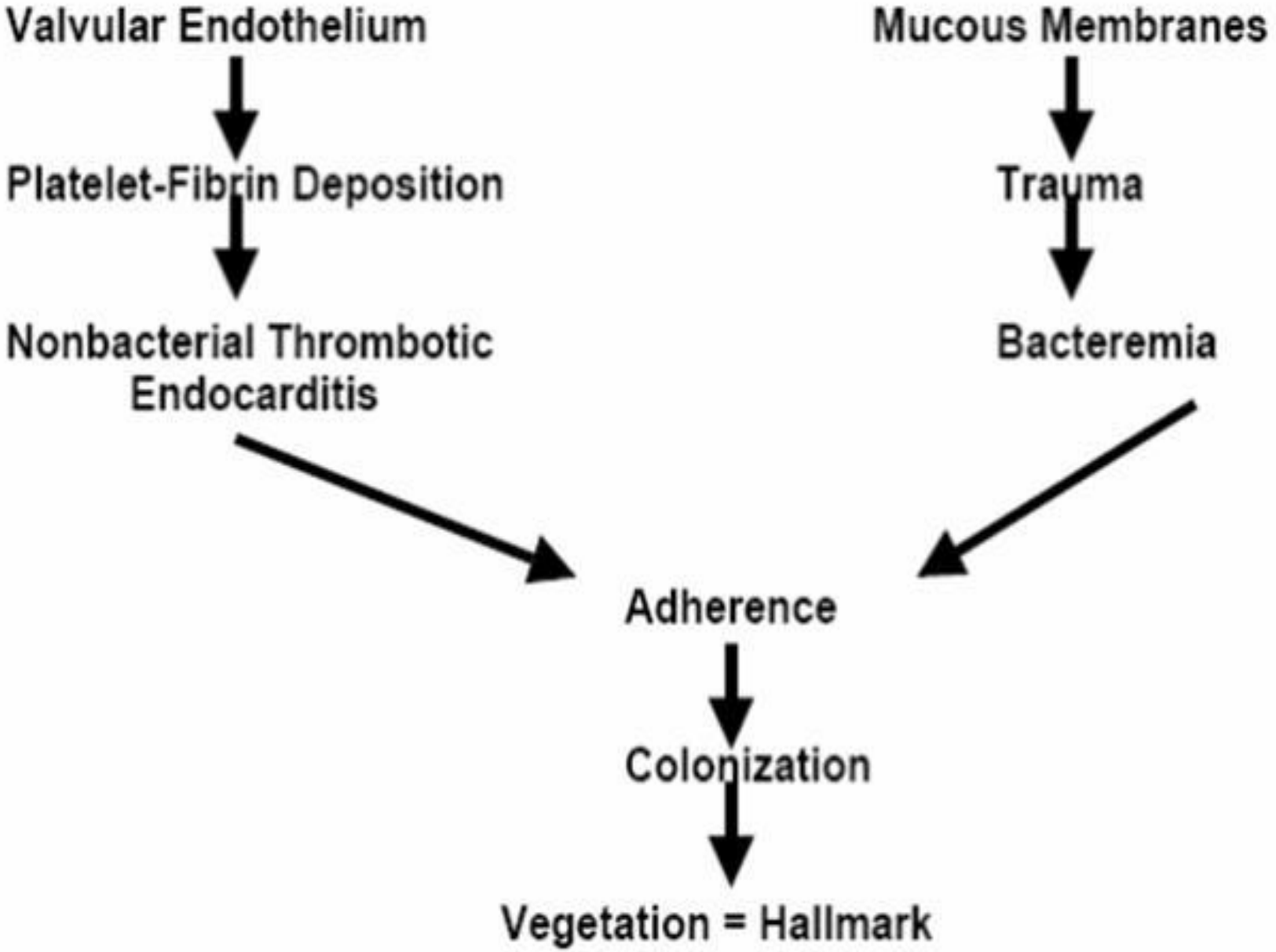
1. The circulating bacteria are lodged much more frequently on previously damaged valves from diseases, chiefly RHD, congenital heart diseases and prosthetic valves, than on healthy valves.

2. Conditions producing haemodynamic stress on the valves are liable to cause damage to the endothelium, favouring the formation of platelet-fibrin thrombi which get infected from circulating bacteria.

3. Another alternative hypothesis is the occurrence of nonbacterial thrombotic endocarditis from prolonged stress which is followed by bacterial contamination

MORPHOLOGIC FEATURES

- **GROSSLY,**
  - lesions are found commonly on the valves of the left heart, most frequently on the **mitral**, followed in descending frequency, by the **aortic**, simultaneous involvement of **both mitral and aortic valve**s, and quite rarely on the valves of the right heart.
  - The **vegetations in SABE** are more often seen on previously diseased valves, whereas the **vegetations of ABE** are often found on previously normal valves.
  - The **vegetations in ABE** tend to be **bulkier and globular** than those of SABE and are located more often on previously normal valves, may cause ulceration or perforation of the underlying valve leaflet, or may produce myocardial abscesses


Valvular Endothelium
Platelet-Fibrin Deposition
Nonbacterial Thrombotic Endocarditis
Mucous Membranes
Trauma
Bacteremia
Adherence
Colonization
Vegetation = Hallmark

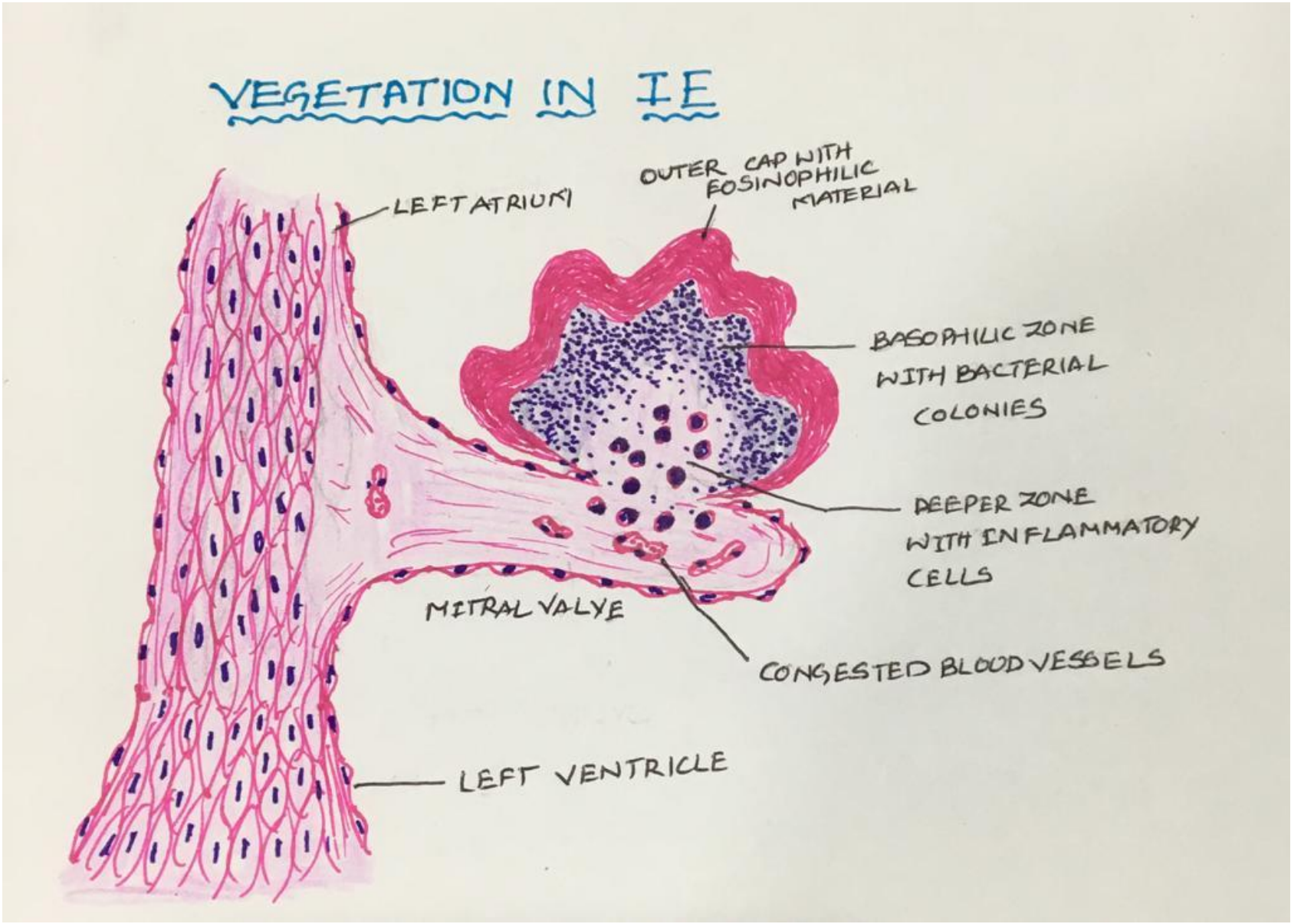
Microscopically,

- the vegetations of BE consist of 3 zones:

I. **The outer layer or cap** consists of eosinophilic material composed of fibrin and platelets.

II. **The basophilic zone** containing colonies of bacteria. However, bacterial component of the vegetations may be lacking in treated cases.

III. **The deeper zone** consists of non-specific inflammatory reaction in the cusp itself, and in the case of SABE there may be evidence of repair


VEGETATION IN IE
OUTER CAP WITH EOSINOPHILIC MATERIAL
LEFT ATRIUM
BASOPHILIC ZONE WITH BACTERIAL COLONIES
DEEPER ZONE WITH INFLAMMATORY CELLS
MITRAL VALVE
CONGESTED BLOOD VESSELS
LEFT VENTRICLE

COMPLICATIONS AND SEQUELAE OF B.E

A. **Cardiac complications.**

I. Valvular stenosis or insufficiency
II. Perforation, rupture, and aneurysm of valve leaflets
III. Abscesses in the valve ring
IV. Myocardial abscesses
V. Suppurative pericarditis
VI. Cardiac failure from one or more of the foregoing complications

B. Extracardiac complications.

- Since the vegetations in BE are typically friable, they tend to get **dislodged** due to rapid stream of blood and give rise to **embolism** which is responsible for very common and serious extra-cardiac complications.

Extra-cardiac complications

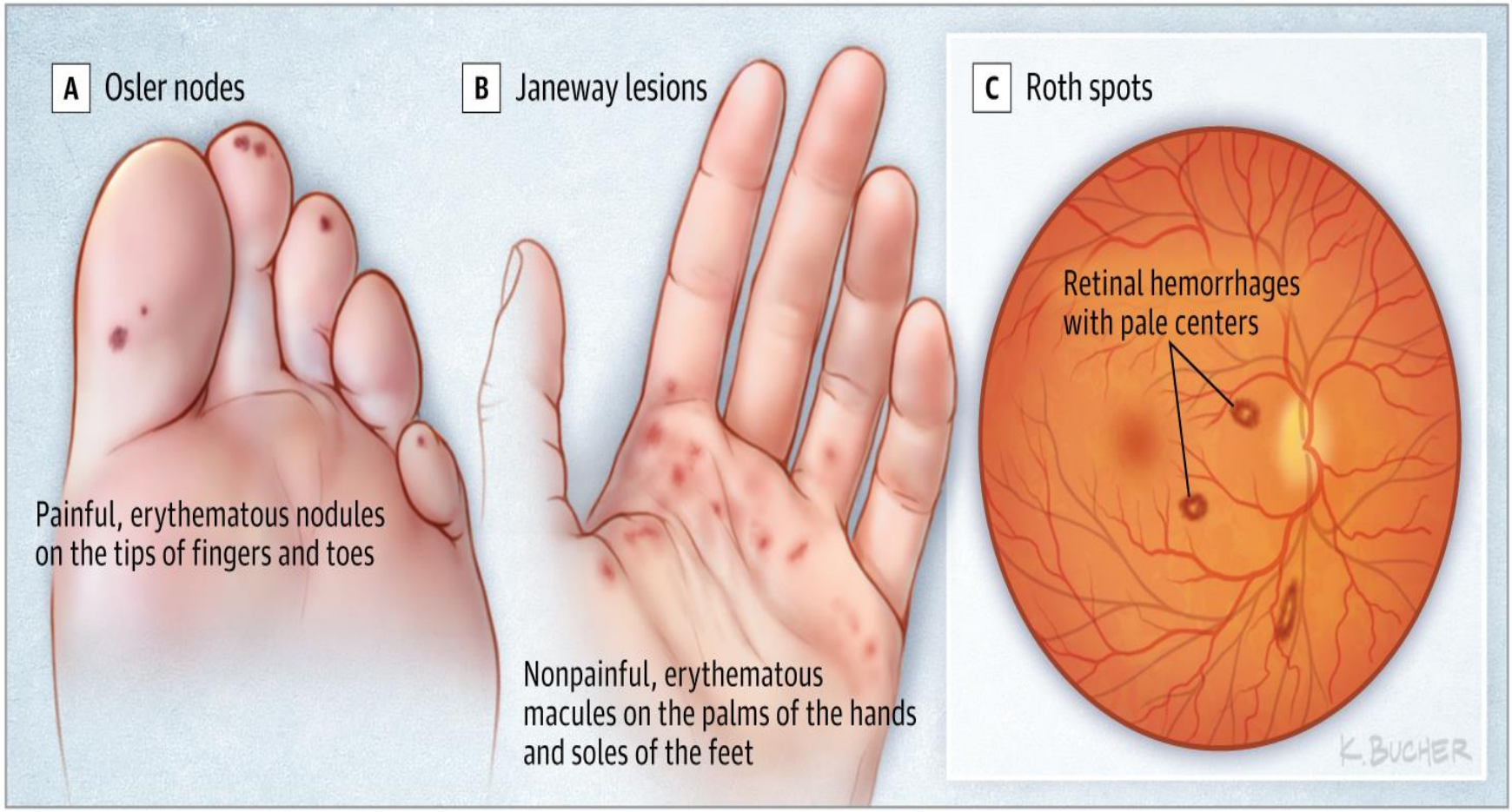
1. **Emboli originating from the left side of the heart** and entering the systemic circulation affect organs like the spleen, kidneys, and brain causing infarcts, abscesses and mycotic aneurysms

2. **Emboli arising from right side of the heart** enter the pulmonary circulation and produce pulmonary abscesses

Extra-cardiac complications

3**. Petechiae** may be seen in the skin and conjunctiva due to either **emboli or toxic damage** to the capillaries

4. **In SABE**, there are painful, tender nodules on the finger tips of hands and feet called **Osler's nodes**, while in **ABE** there is appearance of painless, non-tender subcutaneous maculopapular lesions on the pulp of the fingers called **Janeway's spots**. Both originate from toxic or allergic inflammation of the vessel wall


A Osler nodes
Painful, erythematous nodules on the tips of fingers and toes
B Janeway lesions
Nonpainful, erythematous macules on the palms of the hands and soles of the feet
C Roth spots
Retinal hemorrhages with pale centers
K. BUCHER

Extra-cardiac complications

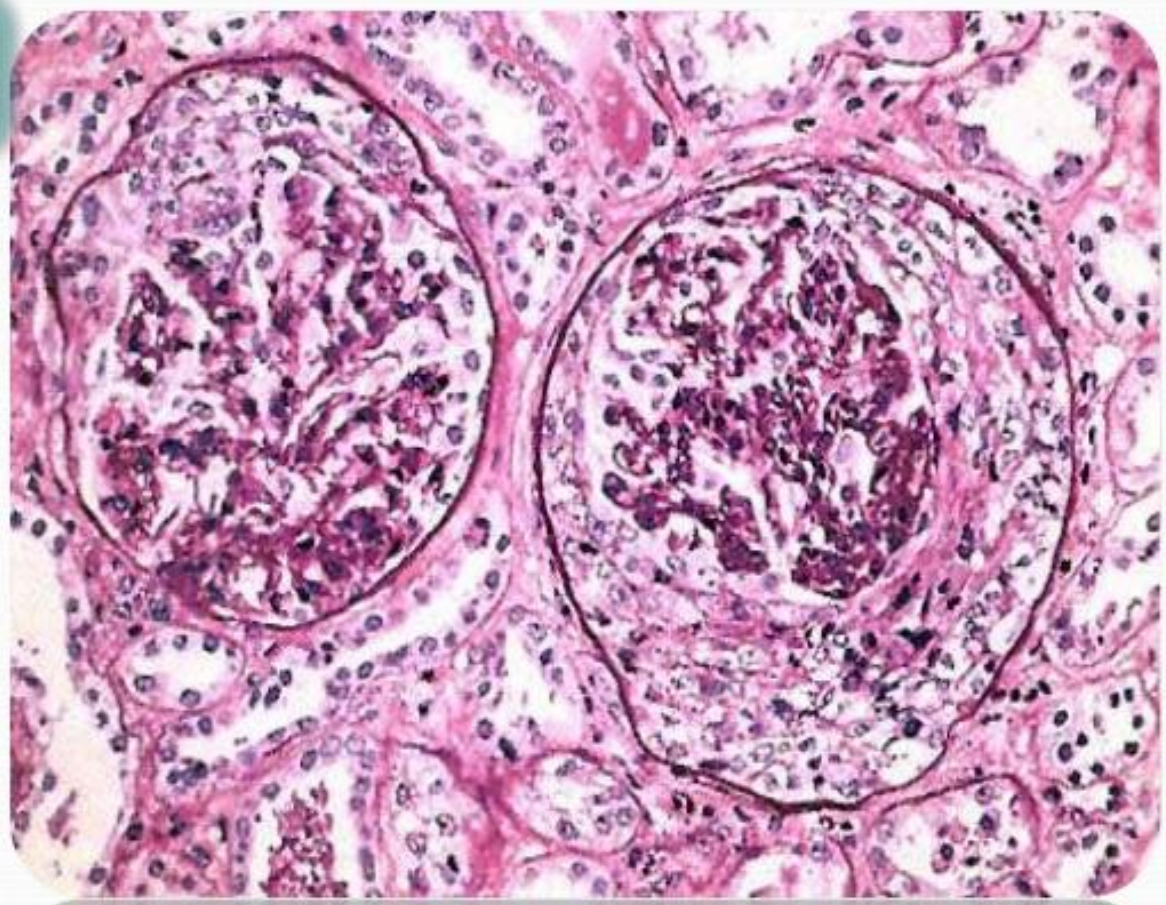
5. **Focal necrotising glomerulonephritis** is seen more commonly in SABE than in ABE. Occasionally diffuse glomerulonephritis may occur. Both have their **pathogenesis in circulating immune complexes** (hypersensitivity phenomenon)

RAPIDLY PROGRESSIVE GLOMERULONEPHRITIS

LIGHT MICROSCOPY

Glomeruli may show focal necrosis, diffuse or focal endothelial proliferation and mesengial proliferation.

The histological picture is dominated by distinctive crescents.



Crescentric glomerulonephritis. Collapsed glomerular tuft and crescent-shaped mass of proliferating parietal epithelial cells.

SPECIFIC TYPES OF INFECTIVE ENDOCARDITIS

1. Tuberculous endocarditis.
2. Syphilitic endocarditis
3. Fungal endocarditis.
4. Viral endocarditis
5. Rickettsial endocarditis.

Tuberculous endocarditis

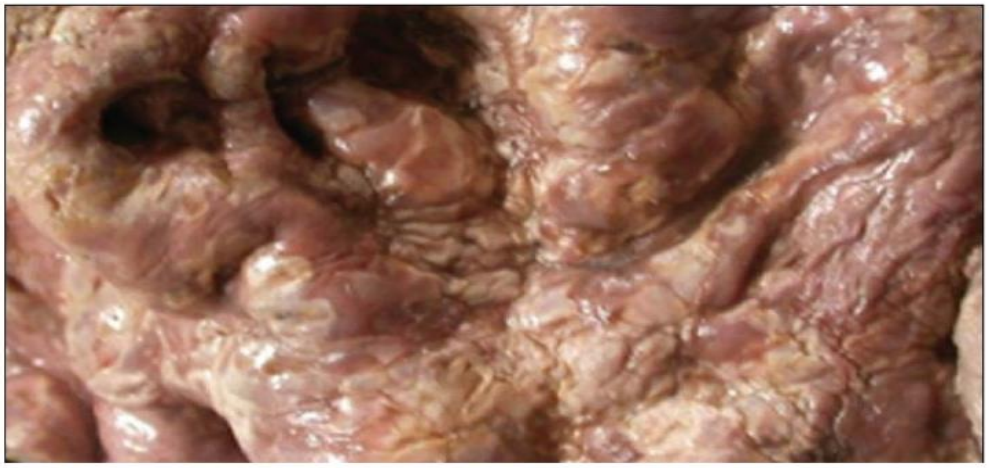
- Though tubercle bacilli are bacteria, tuberculous endocarditis is described separate from the bacterial endocarditis due to specific granulomatous inflammation found in tuberculosis.
- It is characterised by presence of **typical tubercles** on the valvular as well as mural endocardium and may form tuberculous thromboemboli

Syphilitic endocarditis

- The severest manifestation of cardiovascular syphilis is aortic valvular incompetence

Syphilitic Aortitis

- Aortic root scarring
- Loss of elasticity of aorta with progressive dilatation: This results in formation of aneurysm and aortic regurgitation
- Narrowing of coronary ostia, resulting in ischemic heart disease



Aortic root in syphilitic aortitis: Characteristic ***tree-bark appearance***

Fungal endocarditis.

- Rarely, endocardium may be infected with fungi such as from Candida albicans, Histoplasma capsulatum, Aspergillus, Mucor, coccidioidomycosis, cryptococcosis, blastomycosis and actinomycosis
- Opportunistic fungal infections like candidiasis and aspergillosis are seen more commonly in patients receiving long-term antibiotic therapy, intravenous drug abusers and after prosthetic valve replacement.
- Fungal endocarditis produces appearance similar to that in BE but the **vegetations are bulkier in fungal endocarditis**

Viral endocarditis

- There is only experimental evidence of existence of this entity

Rickettsial endocarditis

- Another rare cause of endocarditis is from infection with rickettsiae in Q fever.

QUESTIONS

1. Outline major difference between ABE and SABE (5MK)
2. Discuss the pathogenesis of BE. (6 MKS)
3. Outline the key morphological features found in BE, (5 MKS)
4. Describe the three rings of a Vegetation due to Infective Endocarditis (6mks)
5. Discuss 5 extra-cardiac complications of BE (10 MKs)

END