MICROBIOLOGY

بسم الله الرحمن الرحيم

MID | Lecture 2

Infective Endocarditis

وَلَقَدْ خَلَقْنَا الْإِنسَانَ وَنَعْلَمُ مَا تُوَسْوِسُ بِهِ نَفْسُهُ ۖ وَنَحْنُ أَقْرَبُ إِلَيْهِ مِنْ حَبْلِ الْوَرِيدِ

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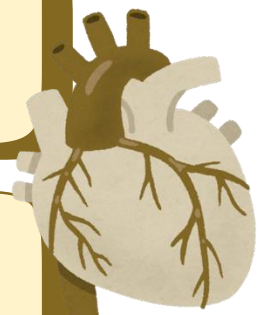

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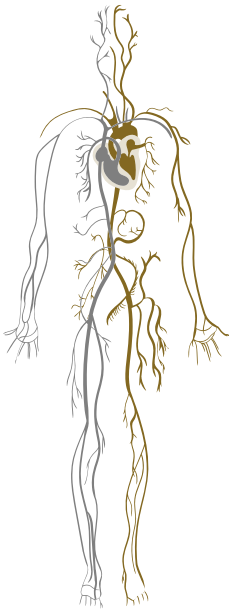


بسم الله الرحمن الرحيم
اللهم افتح علينا فتوح العارفين ونوّر بصرنا وبصيرتنا

Infective endocarditis (IE)

By Assis. Prof. Nader Alaridah MD , PhD

Introduction

- This lecture is important due to the significant medico-legal consequences; being found responsible for an infective endocarditis could lead to the loss of your medical license, regardless of your specialty.
- Regarding viral hemorrhagic fever lecture, a key clinical concept is to distinguish between arboviruses and non-arboviruses, and to know which viruses are capable of **interpersonal** transmission (this is high-yield information).
- For this lecture: keep in mind three factors for IE:
  1- Bacteremia: bacteria in blood due to surgical intervention or installation of a catheter, introducing commensal bacteria into the patient's blood. (Commensal bacteria are usually only present on the skin surface and the mucous membranes.)
  2- The patient has predisposition to the bacteria (the bacteremia) adhering to the leaflets (the epithelial layer) of the heart valves, for many reasons and risk factors, for eg: in our countries (LIC) rheumatic heart diseases, in the west degenerative valvular diseases that develop with age or patients with congenital heart anomalies are more disposed as well. This would lead to infective endocarditis that could lead to seeding in extracardiac sites.
  3- Nosocomial Factor: Increased life expectancy and more frequent hospital visits make patients more vulnerable to the first two factors.
- Infective endocarditis is rare; but the mortality rate is >30%.

IE will be used to refer to infective endocarditits.

Introduction

- IE used to be called **bacterial endocarditis**, but the name was changed due to other causative agents (fungi like *Candida* and *Aspergillus*, parasites like *Trypanosoma cruzi*) being responsible, especially in ICU patients who are on IV antibiotics and parenteral nutrition.
- **Infective** endocarditis (IE) is an inflammation of the endocardium.. Inner of the heart muscle & the epithelial lining of heart valves.
- Another type is **non-bacterial thrombotic endocarditis** (NBTE) - (non-infective). This rare form occurs primarily in immunocompromised, hospitalized cancer patients. It is initiated by endothelial injury and is considered a nosocomial complication.
- Infective endocarditis is a rare, extremely life-threatening disease that has long-lasting effects even among patients who survive and are cured. (**before antibiotics, mortality rate was 75%, after specified antibiotics mortality rate dropped to 20-30% which is worse than most cancers**)
- Infective endocarditis is caused by damage to the endocardium of the heart followed by microbial, usually bacterial, colonization.
- Once established, IE can involve almost any **organ system** in the body and can be fatal if left untreated, **an example is vascular-immunological phenomena that'll be explained later.**

Epidemiology

- The crude incidence ranged from 1 to 10 cases per 100,000 person-years (rare, but in our countries the incidence is higher).

- Infective endocarditis (IE) has undergone **two key epidemiological shifts**:
- **Age of Onset**: The typical patient age has shifted from 30-40 years to over 50-60 years.
- **Underlying Cause:** This is linked to a change in the primary causative agents and predisposing conditions. Previously, Streptococcus viridans and group A beta-hemolytic streptococci led to rheumatic fever, causing early structural heart defects in younger adults. Now, the main risk factor is degenerative valvular disease in the elderly.

- **Rheumatic heart disease** remains the key risk factor for infective endocarditis in **low-income countrie**s and underlies up to two-thirds of cases.
- In **high income** countries, However, **degenerative valve disease**, diabetes, cancer, intravenous drug use, and congenital heart disease have replaced rheumatic heart disease as the major risk factors for infective endocarditis. The mean age of patients with IE has increased significantly (past <30, now >50 years old).
- Untreated, mortality from IE is uniform. Even with best available therapy, contemporary mortality rates from IE are approximately 25%

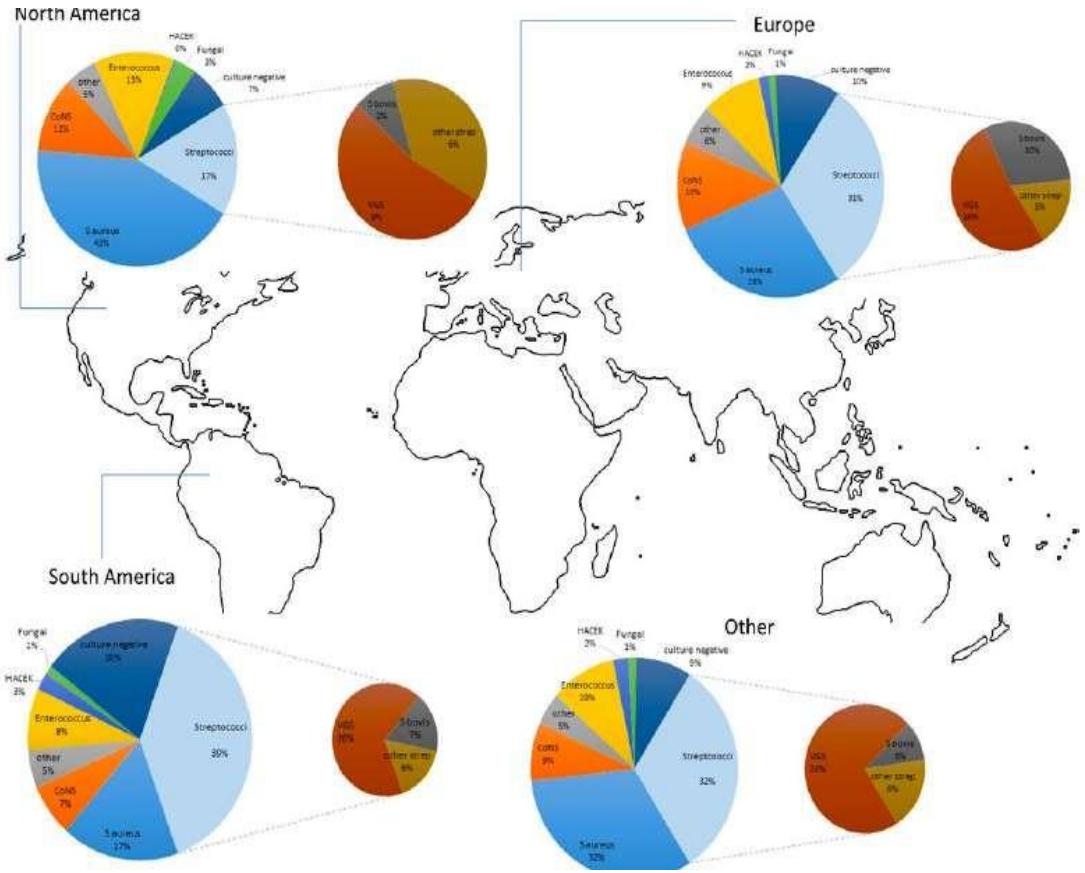
The causative agents **vary by region**

- **90% of the cases are caused by 3 main gram-positive cocci species:**

1. *Staph aureus* (most common in developed countries)
2. *Viridans strept.* (most common in developing countries)
3. *Enterococci* (3rd most common)

- **The rest 10%:**

1. Gram-negative: HACEK group (mentioned later)
2. Fastidious bacteria (less common) → Culture negative endocarditis that don't show as positive in culture.



- The **cornerstone** of IE diagnosis is positive blood culture.
- The **criteria** for diagnosis (modified duke criteria) of infective endocarditis is discussed later in this lecture.

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Predisposing Factors for Endocarditis

Refer to this slide when you read the prevention part of the lecture.

1. Historically and in our countries, rheumatic Disease caused by Group A Streptococci was considered frequent pre- disposing factor for endocarditis

   Treated with penicillin

   - This process is driven by molecular mimicry. A child with a Group A Streptococcus pharyngitis (strep throat) that is left untreated may, after about one week, develop an adaptive immune response. The antibodies produced against the bacteria can cross-react with the body's own tissues, specifically cardiac myosin, leading to rheumatic fever. Each subsequent infection with Group A strep can exacerbate this autoimmune reaction, progressively worsening the structural damage to the heart valves.

2. Congenital heart disorders, prosthetic heart valves, pacemaker
3. Following pneumonia and meningitis
4. Periodontal procedures/disease, Damaged gingival tissue due to plaque accumulation on teeth
5. Dental extractions, dental implants

   - Points 4 and 5 explain why antibiotic prophylaxis is standard for certain dental procedures. These procedures can introduce oral flora (like viridans streptococci) into the bloodstream, fulfilling the first factor for IE—bacteremia. This is why dental work was historically a leading cause of infective endocarditis in many regions.
   - Guidelines from AHA and NICE have changed. In the past, antibiotics were recommended after many procedures, but it was noticed that the incidence of IE didn't decrease. Then, the guidelines in Britain went to another extreme, where antibiotics were not used unless there was a specific indication.
     The current recommendation (as mentioned by the doctor) is to be **moderate**: do not give antibiotics to all patients, but those with specific risk factors should always be given antibiotics before and after the procedure.

Predisposing Factors for Endocarditis

6. Hemodialysis due to catheterization (factor 1), tonsillectomy , esophageal dilation
7. Skin infections.. Intravenous drug users
8. Cystoscopy. Colonoscopy, urethral dilation,

- The most common type of IE is left-sided endocarditis, which affects the left-sided heart valves.
- IV drug users get right sided IE, with the tricuspid valve being the most implicated.
- All these procedures associated with mucosal commensal flora may cause endogenous infections; thus antibiotic prophylaxis is recommended.
- There are also daily activities that may introduce bacteria into our systemic circulation like brushing teeth and chewing gum, but those are usually very transient and low quantity.

BETTER BE SAFE THAN SORRY !!

Microbiology Overview

- The microbiology of the disease has also changed, and staphylococci, most often associated with health-care contact and invasive procedures, , as well as community acquired, have overtaken streptococci as the most common cause of the disease.

- streptococci and staphylococci have collectively accounted for approximately 80% of IE cases, the proportion of these two organisms varies by region.

Microbiology outline (causative agents – mentioned in slide 4 + 6)

- The **Gram-positive cocci** of the staphylococcus, streptococcus, and enterococcus species account for 80–90% of infective endocarditis.
- ***S.aureus*** is the most frequently isolated microorganism associated with infective endocarditis in **high-income countries** and is reported in up to 30% of cases.
- **Streptococcal** infective endocarditis caused by the oral *viridans* group remains most common in **low-income countries.**
- **Enterococci** account for 10% of cases overall.
- The **HACEK** bacteria (*Haemophilus, Aggregatibacter, Cardiobacterium,Eikenella corrodens, kingella*), which cause about 3% of cases.
- **Fungal** endocarditis, usually *Candida* or *Aspergillus*, is rare but often fatal, arising in patients who are immunosuppressed or after cardiac surgery, mostly on prosthetic valves. (ICU patients who are on IV antibiotics and parenteral nutrition; broad-spectrum antibiotics can also have significant immunosuppression effects.)
- As mentioned, **parasites** like *Trypanosoma cruzi* can also be causative agents.
- Other causes include **fastidious bacteria** such as *Tropheryma, Pileliae, Brucella* (a cause in our region), *Bartonella*, and *Coxiella burnetii*. The diagnostic challenge with these pathogens is that they often present as culture-negative endocarditis, requiring diagnostic methods other than standard culture to confirm IE.

This table summarizes the most common causes of IE.

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➢ Staphylococcus Species:

- *S. aureus* is coagulase-positive.
- Coagulase-negative staphylococci (CoNS) include species like *S. epidermidis*, *S.saprophyticus*, *S. lugdunensis*, and *S. capitis.* These are common skin commensals, with *S. epidermidis* being the most clinically significant for IE.

➢ Streptococcus species

- We classify Streptococcus species based on their hemolytic patterns on blood agar.
  → Alpha-hemolysis refers to partial hemolysis.
- The specific strain that causes rheumatic fever is the Group A beta-hemolytic streptococcus (e.g., *S. pyogenes*).
- The grouping (A, B, C, etc.) is based on the carbohydrate antigen in the bacterial cell wall, according to the Lancefield classification system.

❖ Tests

- **The catalase test**: Staphylococcus species are catalase-positive, while Streptococcus and Enterococcus are catalase-negative.
- **The coagulase test** is then used to speciate: Staphylococcus aureus is coagulase-positive, distinguishing it from other staphylococci (e.g., S. epidermidis), as well as from streptococci and enterococci.

	Catalase	Coagulase	Hemolysis†	Distinguishing Features	Disease Presentations
***Staphylococcus* Species**					
S. aureus	+	+	β	Ferments mannitol Salt tolerant	Infective endocarditis (acute) Abscesses Toxic shock syndrome Gastroenteritis Suppurative lesions, pyoderma, impetigo Osteomyelitis
S. epidermidis	+	–	γ	Novobiocin^S Biofilm producer	Endocarditis in IV drug users Catheter and prosthetic device infections
Viridans group (not groupable)	–	–	α	Optochin^R	Infective endocarditis Dental caries
Enterococcus sp. (Group D)	–	–	α, β, or γ	PYR† Esculin agar	Infective endocarditis Urinary and biliary infections
S. bovis	–	–	γ	Bile esculin†	Endocarditis, especially in patients with colon cancer

- *S. aureus:* most common cause of **early AND late** prosthetic valve IE.
- *S. epidermidis:* most common cause of **early** prosthetic valve IE. Also in IV drug users.
- *Viridans* is abundant in oral cavity.
- *Enterococcus* is a significant pathogen, particularly following abdominal surgery. The main species are *E. faecium* and *E. faecalis*. It is important to be mindful of this risk in patients undergoing urinary or biliary tract surgeries.
- *S. bovis* is a common cause for IE especially in patients with colon cancer.

Microbial Causes-1

- Remember catalase test differentiate between staphylococci which is catalase positive and streptococci which is catalase negative depending on the ability to neutralize hydrogen peroxide into oxygen and water.

- While coagulase test is used to differentiate staphylococci into staphylococci aureus which is catalase positive and other coagulase negative staphylococci species.

- Gram-positive cocci.. facultative anaerobes, diplococci chains/clusters or pairs cocci.. Catalase +ve /Staphylococci group.. catalase-ve/ Streptococci & Enterococci groups.

Streptococci are subdivided depending on their pattern of hemolysis (partial-alpha hemolysis) into type A (true hemolysis) and type B (pseudo-hemolysis).

- Streptococci subdivided into groups according their hemolytic reaction on blood agar in vitro & by serotypes according to surface cell wall specific carbohydrate antigens.

They're mostly commensals in the oral cavity and they survive the aerobic environment there by:
1. Forming a biofilm where the outer layer is exposed to O2 and the inner is anaerobic
2. They reside in the gingival sulcus where oxygen is very low
3. Streptococci will consume O2 (through NADH oxidases) in the oral cavity creating a more favorable anaerobic environment so other obligate anaerobes can be established there (this is called metabolic cooperation)

Doctor mentioned being catalase +ve as another way to reduce O2 levels in the oral cavity but when discussing oral cavity the most common commensals are streptococci which are catalase -ve in addition to catalase enzyme neutralizing H_2O_2 to O_2 and H_2O (it can help reduce H_2O_2 levels as part of the 'metabolic cooperation' but it can't help lower O_2 levels in the oral cavity)

- Viridans comes from ''viridis'' which means green in latin as they produce green discolorations around their colonies in alpha hemolysis and Dr. Nader also called them 'dense' as they look solid and non translucent on their blood agar plate (not the physical density of bacterial cells)

  I added a picture of st.pneumoniae (both perform alpha hemolysis) to see how dense they look compared to the tranlucent St.pneumoniae

St.viridans

Streptococcus pneumoniae

Microbial Causes-1A

❖ Viridans streptococci Group (VGS)

- Normal oral-intestinal flora.. Common causes of dental caries.. Oral abscesses Gingivitis Deposit **dextran, adhesins, Fibronectin-binding protein.** They cause 'dental tartars' due to biofilm formation (mentioned in the previous slide) الجير حول الأسنان
- *St. mutans, St. mitis* accounted for many cases, and tend to be less susceptible to penicillins. Other examples are St. salivarius, St. anginosus, and St. sanguinis

❖ Group A Streptococci (*S. pyogenes*).. Repeat Sore throat infection.. Less skin infection.. Develop Pos-streptococcal Diseases ..Rheumatic heart disease.. Children. Observed later in young adults

- They can also cause IE but much more rarely.

Microbial Causes-1B

- An old way of classifying IE was based on symptoms into :
  - Acute In days to weeks (less than eight weeks)
  - Subacute (2–3 months)
  - Chronic (more than 3 months) if the patient survives

❖ ***S. aureus*** is a common cause of acute endocarditis, may result in a severe sepsis syndrome with a fatal outcome.

- Most endocarditis cases occurred within 2-month-1 year following vascular catheters & surgical wounds, skin injury/ invasive dental procedures and others. Because Staphylococci are commensals on our skin (they cause the problem when they reach the heart through blood circulation)

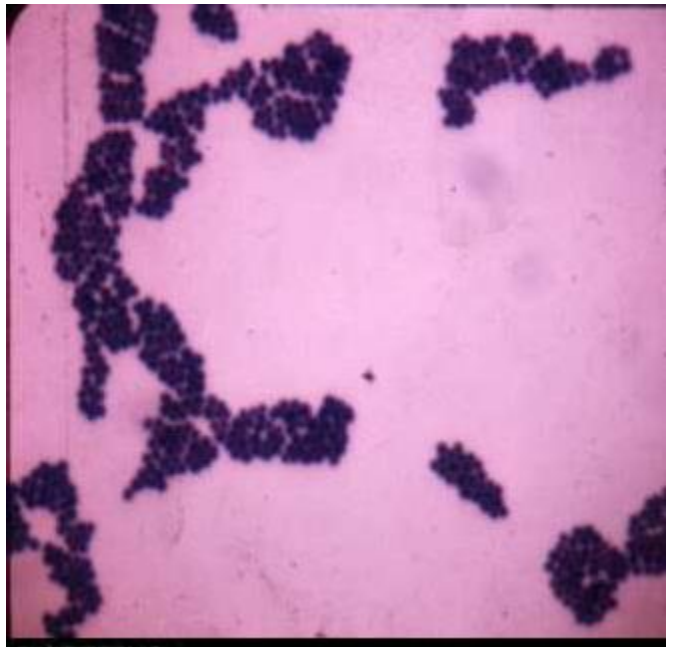
❖ *Enterococcus species* (*E. fecalis, E. faecium*) are responsible for up to 5-10% of cases; some strains may be resistant to penicillin,vancomycin.
So the empirical treatment for bacterial infections in hospitals wouldn't work (VREs)

- **Acute** → Staphylococci aureus + group A beta hemolytic streptococci + strep. pneumoniae . Acute = more fulminant with faster detoriation (vegetations expand at faster rates)
- **Subacute** → more subtle presentation caused by very dense group streptococci (Viridans streptococci)
- **Chronic** → with fungal infections or fastidious or gram negative bacteria

Again, S.Aureus are the most common cause of IE in developed countries + most common cause of prosthetic valve IE (if in the question the dr specified that IE happened early within 6 months of having the prosthetic valve then the causative agent would be staph epidermidis)

Streptococci-Staphyloccoci

- Streptococci (remember they grow in chains)
- Dense alpha (partial) hemolysis on blood agar



- Staphylococci: they grow in clusters like clusters of grapes
- Selective agar mannitol salt agar (turn it to yellow due to their ability to ferment mannitol)
- This feature is specific to **staph. aureus** and will turn negative for other coagulase negative strains

Microbial Causes-2

Make up 5–7% of IE causes worldwide

- A group of fastidious gram-negative bacteria can cause rarely endocarditis : Gram-ve *bacteria: Brucella, Salmonella, Haemophilus, Cardiobacterium, Eikenella, Gram-ve Actinobacillus* part of Normal oral flora .

- Clinically, these bacteria spp. causing subacute or chronic course, and often present with embolic lesions from large biofilm vegetations in heart valves .

- Most cases of fungal endocarditis occur in patients who are receiving prolonged antibiotics or intravenous nutrition through central vascular catheters.. Immuno-compromised patients.

Yeast & Filamentous Fungi

- The most common species is *Candida albicans*, followed by other less common *Candida spp.* (C. glabrata, C. krusei, C. Tropicals, C. parapsilosis). All can be causative agents for IE due to fungal infections
- Candida part of human normal flora.. Oral-intestinal-Urinary tract (Vagina).. Infection often followed often using catheters or respiratory intubation.
- Endocarditis due to *Histoplasma capsulatum* / *Aspergillus* species is very rare.. Immuno-suppressed patients.

➢ **Broad-spectrum antibiotics, parenteral nutrition, intubation, and urinary catheterization are all risk factors for candidal IE.**

Candida albicans Pseudohyphae

- Candida is also called “pleomorphic” as it can be in the form of yeast or both pseudo and true hyphae.

- This is a picture of **pseudohyphae** since you can see the yeast cells

- Sabouraud Dextrose Agar (**SDA**) it shows white, waxy, and creamy colonies

Pathophysiology

- The healthy cardiac endothelium is resistant to frequent bacteremia caused by daily activities such as chewing and tooth brushing.

- Bloodstream infection is a prerequisite for development.

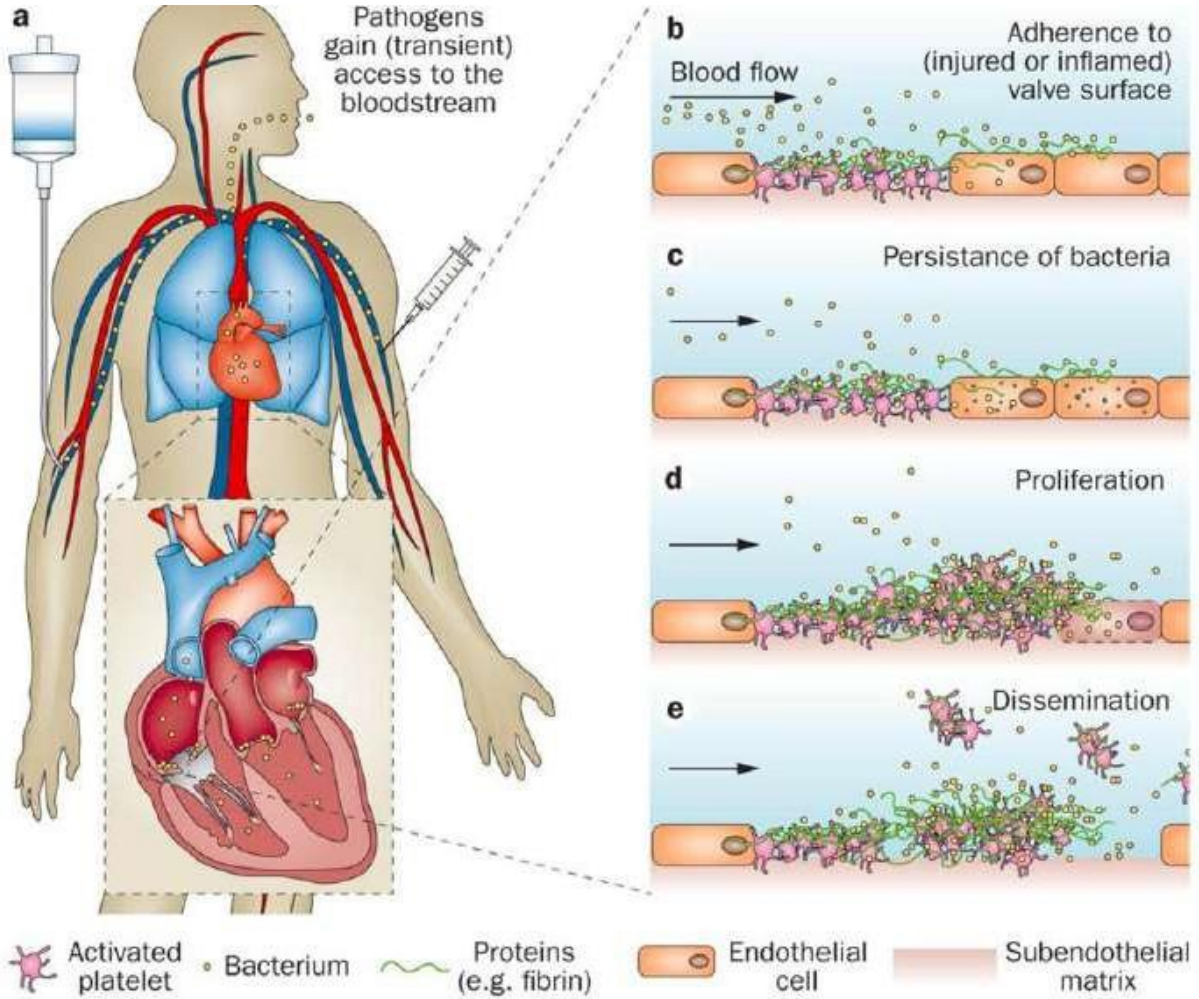
- The development of IE requires the simultaneous occurrence of several independent factors: alteration of the cardiac valve surface to produce a suitable site for bacterial attachment and colonization; bacteraemia with an organism capable of attaching to and colonizing valve tissue; and creation of the infected mass or 'vegetation' by 'burying' of the proliferating organism within a protective matrix of serum molecules (for example, fibrin) and platelet A **Biofilm ..** Accumulation Bacteria, platelets, fibrin and few leucocytes. If the vegetation dislodge may cause embolism, but if it stays this will cause fever for unknown origin and new onset murmur, and if the patient had an old murmur, it will be changed.

- We need two factors for IE to occur :

1. A way for the bacteria to enter the circulation in order to reach the blood.
2. A way for the bacteria to stick into the walls of the endothelial cells.

a: The most common pathogens that cause IE are already commensals in our body (specifically skin and oral cavity) so either by dental procedures oral commensals like viridans strep. or for skin commensals like staph. aureus through IV catheters or needles can enter the circulation.

- Just caving the pathogen enter the circulation and reaching the heart valve doesn't mean IE will develop, as the bacteria must stick into the endothelial lining which connot happen in normal healthy intact endothelium.

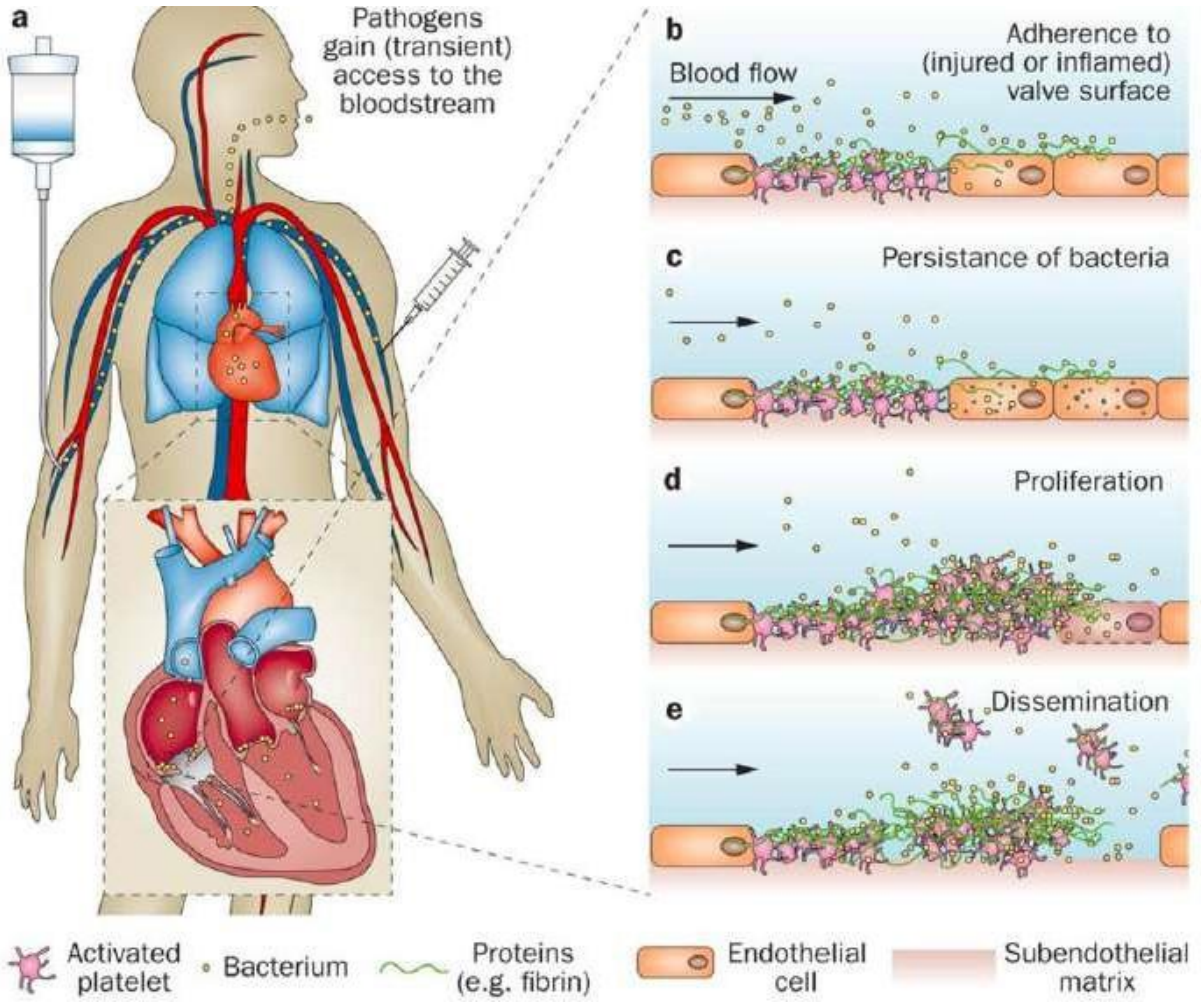


➢ We need to factors for IE to occur :

1. A way for the bacteria to enter the circulation in order to reach the blood
2. A way for the bacteria to stick into the walls of the endothelial cells

- There should already be damage in epithelium leading to part of it being exposed which will lead to coagulation cascade and forming of a blood clot between endothelial cells.
- Now if the bacteria has gained access to blood and reaches the chambers when there is already damage to the epithelium it can bind to a blood clot and colonize the damaged epithelium[2]. So the damage of the endothelial lining creates a 'sticky spot' for the bacteria.
- Some of the bacteria will gain entry to the underlying connective tissue and activating sentinel cells causing inflammation
- Then the bacteria can get buried under a protective clot, forming a mass called a **'vegetation'**
- This forms a big issue as the bacteria buried in the second coat of fibrin is now protected from WBCs (they can't penetrate the dense vegetation to reach them) and have the perfect environment to proliferate and part of will disseminate to the blood creating septic emboli leaving the WBCs fighting the CT which leads to extreme damage to heart valve.

Staph. aureus enters intracellularly then multiply and form a vegetative mass.

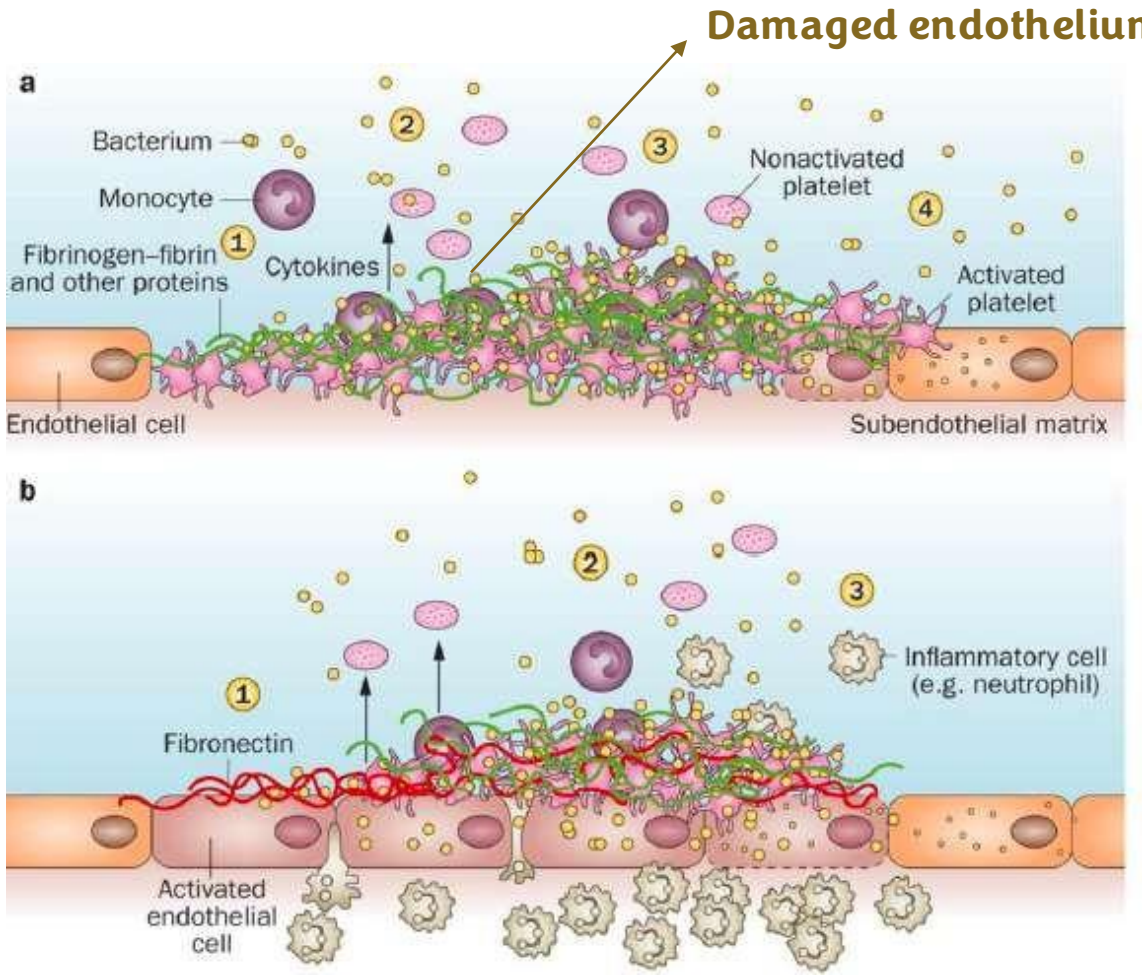


➢ Since bacteria is getting buried within the vegetation, recovery will take **longer** after taking the correct antibiotics.

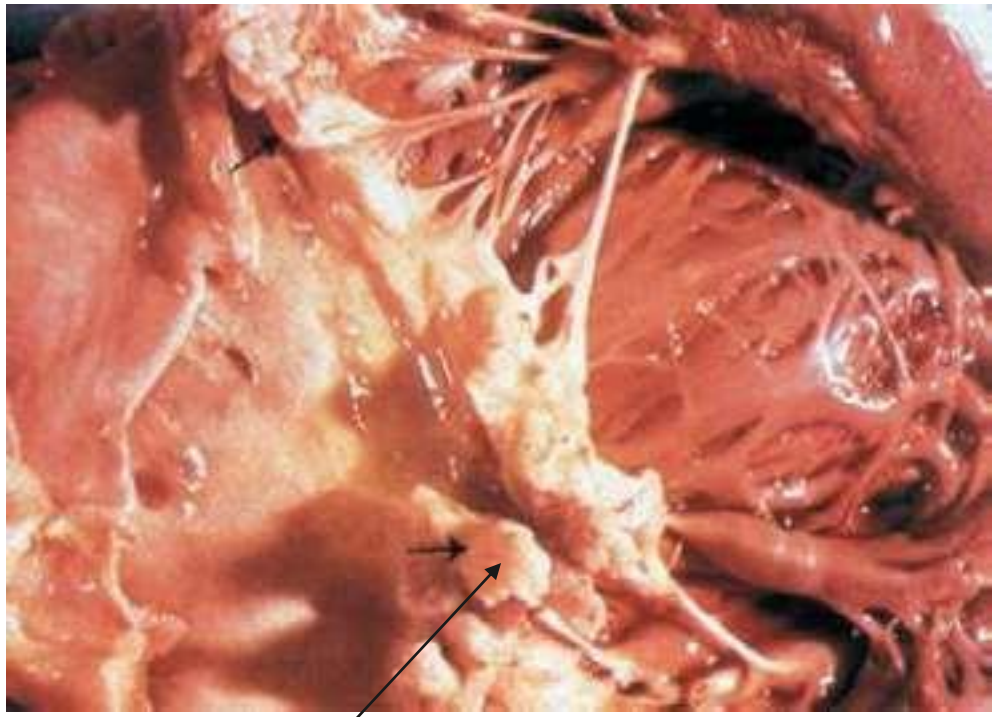
a. It can either start from a mechanical injury and ripping the endothelial cells off (space where the blood clot is) → dr mentioned this is an example on **prosthetic valve endocarditis.** These valves are more prone to bacteria adherence.

b. b. Or it can be from inflammation that leads to the activation of endothelial cells making them express specific adhesion molecules on their surface making it possible for bacteria to stick on its surface (dr mentioned this is an example of **native valve endocarditis**). The endothelial lining here is not damaged.

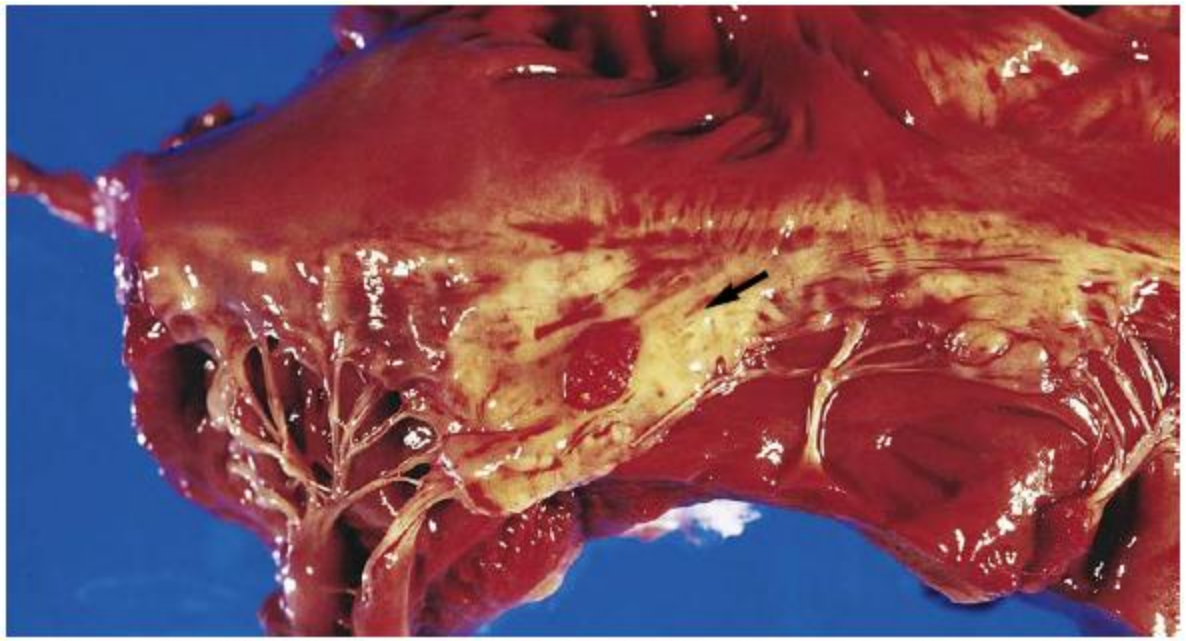
➢ Both will still have the same pathophysiology and formation of a vegetation.



EXTRA From baily and scott's microbiology



Autopsy showing vegetative mass



• **Fig. 67.1** Vegetations of bacterial endocarditis. *Arrow* indicates the vegetations. (Courtesy Celeste N. Powers, MD, PhD, Virginia Commonwealth University Medical Center, Medical College of Virginia Campus, Richmond, VA.)

Clinical features

- The most common presentation is unfocused fever.
- Second chief complain is the newly onset murmur OR changes in the already existing murmur.

- The clinical presentation of infective endocarditis is particularly diverse and non-specific.

- Acute endocarditis is a hectically febrile illness that rapidly damages cardiac structures, seeds extracardiac sites **causing extracardiac manifestations** , and, if untreated, progresses to death within weeks.

- Subacute endocarditis follows an indolent course; causes structural cardiac damage only slowly, if at all; rarely metastasizes; and is gradually progressive unless complicated by a major embolic event or a ruptured mycotic aneurysm

Cardiac Manifestations

- Although heart murmurs are usually indicative of the predisposing cardiac pathology rather than of endocarditis, valvular damage and ruptured chordae may result in new regurgitant murmurs.

- Congestive heart failure (CHF) develops in 30–40% of patients as a consequence of valvular dysfunction.

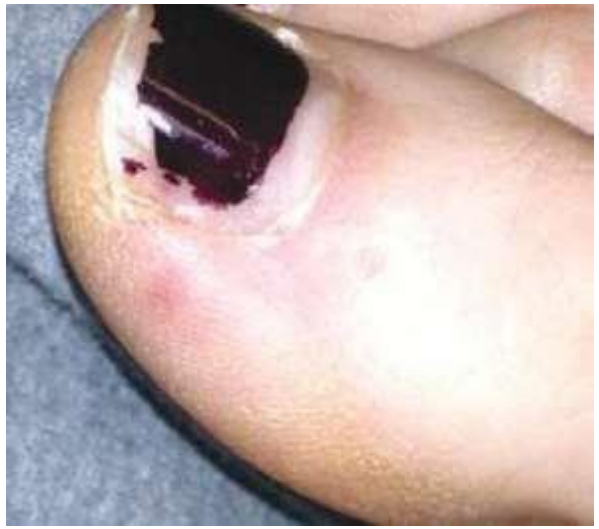
Noncardiac Manifestations

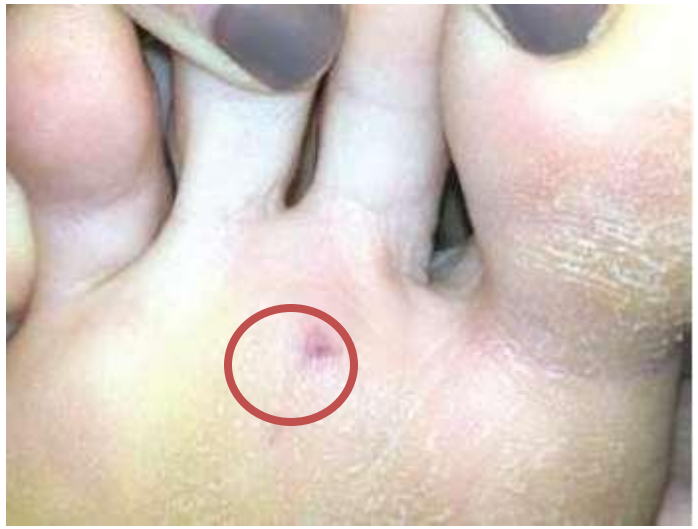
Minor events (major events may cause **stroke** or **mycotic aneurysm**).

- The classic nonsuppurative peripheral manifestations of subacute endocarditis (e.g., Janeway lesions are related to prolonged infection).
- In contrast, septic embolization mimicking some of these lesions (subungual hemorrhage, Osler's nodes) is common in patients with acute S. aureus endocarditis.

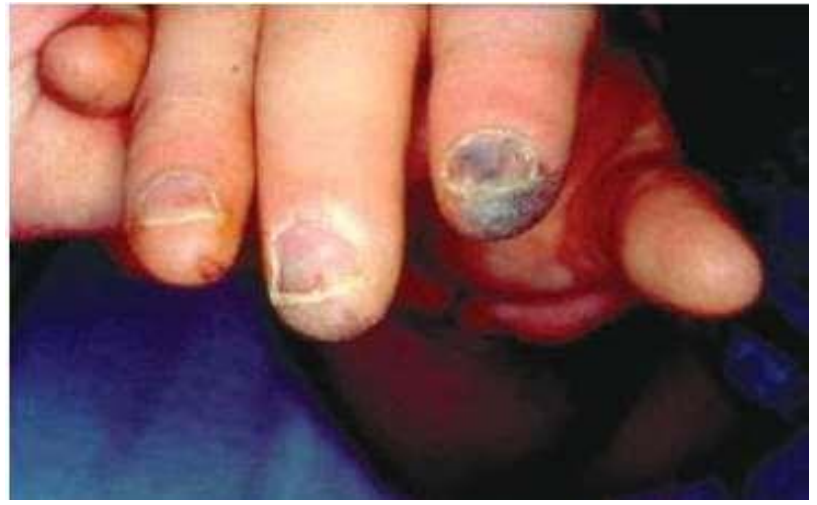
- 2 phenomena:
  - Vascular: lead to microemboli (**Janeway lesion** or splinter hemorrhage under nails). كل ما تكبر شوي بتروح عمكان تاني بالجسم
  - Immunological: caused by immune complex vasculitis and examples are **osler's node**, retinal hemorrhage and glomerular nephritis.

- Osler's phenomena occurs in soles and palms (same for Janeway lesion) except osler's is a nodule while Janeway is a papule or a macule. Osler nodes are tender (painful).





Osler's nodes (immunological)



Subangular hemorrhage (vascular)

➢ **2 Major criteria depend on:**

1. Microbiological culture: positive blood culture with one of the causative agents.
2. Echocardiography: structural deformity of the heart valves (echo showing structural changes and vegetative mass) .

➢ **5 Minor criteria:**

1. Predisposing factors.
2. Fever with unknown origin.
3. Culture negative, that can be conformed by serological tests.
4. Vascular phenomena.
5. Immunological phenomena.

- **we stopped using them for diagnosis because they can be seen in many other conditions other than EI.**

DIAGNOSIS

- The diagnosis of IE typically requires a combination of clinical, microbiological and echocardiography results .

- Blood culture is the most important initial laboratory test in the workup of IE. Bacteremia is usually continuous and the majority of patients with IE have positive blood cultures.

- Echocardiography is the second cornerstone of diagnostic efforts and should be performed in all patients in whom IE is suspected.

- A highly sensitive and specific diagnostic schema—known as the **modified Duke criteria**—is based on clinical, laboratory, and echocardiographic findings commonly encountered in patients with endocarditis

➢ To be diagnosed with IE, we use new sensitive criteria (**Duke criteria**) , and we need:

1. 2 major
2. OR 1 major + 3 minor
3. OR 5 minor

➢ Some causative agents will be hard to culture or fastidious bacteria so we will need to depend on serology.

- Non-Blood-Culture Tests : Serologic tests culture, microscopic examination with special stains, (i.e., the periodic acid–Schiff stain for T. whipplei), direct fluorescence antibody techniques and by the use of polymerase chain reaction to recover unique microbial DNA or DNA encoding the 16S or 28S ribosomal unit.

- Echocardiography

Management

- Blood should be collected multiple times from the patient through venous culture and before starting with drugs or it will alter the lab results (false negative) → usually venous culture is done about 4 times to reach 99% sensitivity.
- In cases with induction of skin flora during cannulation, the culture usually becomes positive within the first 4-5 hours. However, in true bacteremia, the culture tends to be heavily positive within the first 4-5 hours and remains positive for 12-24 hours.

❖ANTIMICROBIAL THERAPY

- **Vancomycin plus Gentamicin** initiated immediately after blood samples are taken for cultures.

- Extended courses of parenteral therapy with bactericidal (or fungicidal) agents are typically required.

→ **Amphotericin**

❖Surgical Treatment. For structural defects and predisposing factors.

PREVENTION

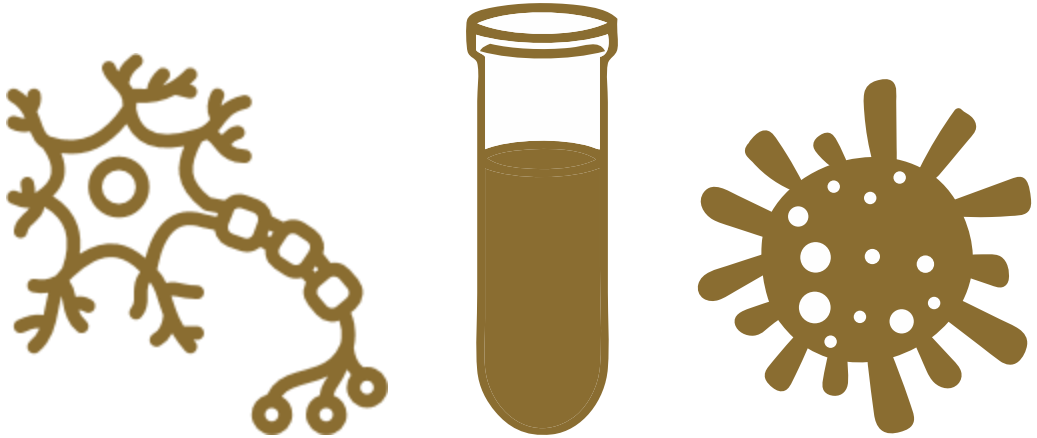
- There is a lot of controversy on prevention → if the patients has any predisposing factors and the procedure might introduce skin commensals then give antibiotics.

- To prevent endocarditis (long a goal in clinical practice), past expert committees have supported systemic antibiotic administration prior to many bacteremia-inducing procedures.

The End

الحمد لله

اللهم إني استودعتك ما قرأت وما حفظت وما تعلمت فردّه إلي عند حاجتي إليه، واجعل علمي هذا حجة لي لا علي.



MICROBIOLOGY QUIZ LECTURE 2

External Resources

رسالة من الفريق العلمي

References as cited:

1. chrome-extension://efaidnbmnnnibpcajpcglclefindmkaj/https://asm.org/getattachment/7ec0de2b-bb16-4f6e-ba07-2aea25a43e76
2. Baily and Scott's diagnostic microbiology 5th chapter 67 (pages 956-957)

click here and listen to it, you'll feel better

Scan the QR code or click it for FEEDBACK

Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1	13	Dental carriers	Dental tartars
V1 → V2			