CVS

MICROBIOLOGY

Modified NO: 1

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Welcome back! I know it might seem like the lecture's packed with much details and classifications, but don't worry, the questions in exam are usually clear and straightforward. Dr. focuses on certain points that will be clarified in the Modified. He's just trying to give you extra info to help you in clinical life to be a better doctor, so that's why he adds more details. But remember, the most important thing is to focus on the main points!

Infective endocarditis (IE)

Intro: In the past, it was called bacterial endocarditis, But because there are other than bacterial causes, which are fungal causes, so it became called infected endocarditis. also, the word "infective" may also tell you that there is endocarditis other than infectious causes, and they call it non-bacterial thrombotic endocarditis, but this forms a very minority of endocarditis. Anyways, we are talking **about Infective endocarditis** today, which is most commonly caused by bacterial organisms.

Assis. Prof. Nader Alaridah MD , PhD

- Infective endocarditis (IE) is an inflammation of the endocardium.. inner of the heart muscle & the epithelial lining of heart valves.
- Infective endocarditis is a rare, life-threatening disease that has long- lasting effects even among patients who survive and are cured

- 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.

Epidemiology

- The crude incidence in developed countries ranged from 1 to 10 cases per 100,000 person-years.
- Rheumatic heart disease remains the key risk factor for infective endocarditis in low-income countries 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. mortality rate, if untreated, is 100% .Even with best available therapy, contemporary mortality rates from IE are approximately 25% , So it's worse than most cancers.

There are two major points to emphasize in the epidemiology of infective endocarditis.

1.Age Shift in Presentation: In the past, infective endocarditis was commonly diagnosed in patients in their mid-30s. Now, it is more often seen in patients aged 50 and above. This shift is due to two main factors: *increased life expectancy and *improved healthcare facilities, which allow people to live longer, and consequently, they are older when diagnosed.

2.Change in Predisposing Factors: Previously, rheumatic heart disease was the most common underlying condition. Today, however, degenerative heart diseases, particularly valvular degenerative diseases, are the primary predisposing factors in developed countries. These include calcified aortic stenosis and valvular regurgitation.

What are the three independent factors involved in infective endocarditis?

1.Bacteremia: The first factor is the presence of bacteria circulating in the blood, known as bacteremia. Healthcare personnel, particularly doctors and dentists, can inadvertently introduce bacteria into the bloodstream during procedures. This is why prophylactic antibiotics are sometimes recommended before certain medical or dental procedures .

2.Structural Heart Defects: The second factor is the presence of a structural defect in the patient's heart. Bacteria in the bloodstream can adhere to abnormal or damaged areas of the heart, such as valve leaflets, which can lead to colonization and the establishment of an infection.

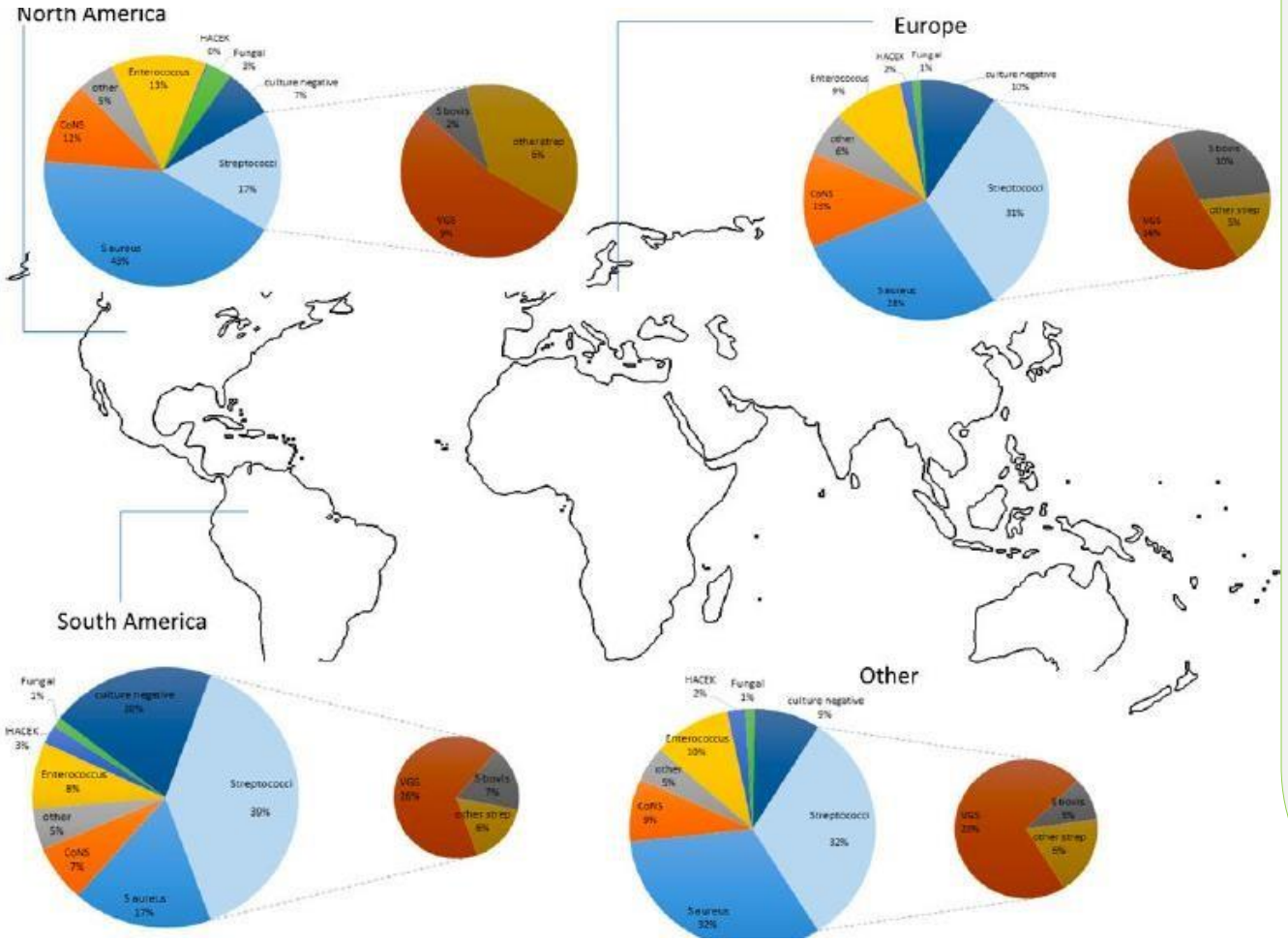
3.Vegetation Formation: The third factor is the formation of vegetations. This occurs when bacterial colonies become encased in a biofilm, creating a mass called "vegetation" on the heart valves. This vegetation can either stay in place, causing local damage to the heart, or it can dislodge and travel to other parts of the body, leading to complications.

These three factors : bacteremia, structural heart defects, and vegetation formation , are important to the pathogenesis of infective endocarditis.

Extra info: calling these factors "independent" means that each one plays a unique and crucial role in the process of developing the infection, but they do not necessarily need to happen at the same time or in a specific order. In practice, all three factors together (bacteremia, heart defect, and vegetation) are necessary for infective endocarditis to fully develop. But each factor independently increases the susceptibility or progression of the condition

The causative agents

✨يلي بدي اوصلك اياه انه:✨
The most common causative organism globally is ***Staph aureus***. In developing countries, ***Viridans streptococci*** remains a prevalent cause, reflecting differences in epidemiology



These graphs show the microbiology of infective endocarditis across different continents. In 90% of cases, infective endocarditis is caused by gram-positive cocci, with the majority being:
1.*Staphylococcus*,
2.Streptococcus, or
3. *Enterococcus* species

The remaining 10% of cases are typically caused by

gram-negative bacteria (HACEK), which are more commonly associated with healthcare-acquired (nosocomial) infective endocarditis

Non-bacterial causes, such as fungi (*Candida albicans*, *glabrata*, *auris*, *Aspergillus flavus*, and *Histoplasma capsulatum*)

Predisposing Factors for Endocarditis

The Dr read everything here

- Historically (in the past), **Rheumatic Disease ..caused by** Group A Streptococci was considered a frequent pre-disposing factor for endocarditis. it's still The most common predisposing factor in **developing** countries
- Congenital heart disorders, Prosthetic heart valves Pacemaker, following pneumonia and meningitis (most common in **developed** countries)
- Periodontal procedures/disease, Damaged gingival tissue due to plaque accumulation on teeth
- Dental extractions, Dental implants
- Hemodialysis Tonsillectomy , Esophageal dilation
- Skin infections.. Intravenous drug users
- Cystoscopy. Colonoscopy, Urethral dilation,

All these can introduce normal flora to blood then to the heart

- All these procedures.. associated with mucosal commensal flora.. May cause endogenous infections. Thus Antibiotic Prophylaxis is recommended.

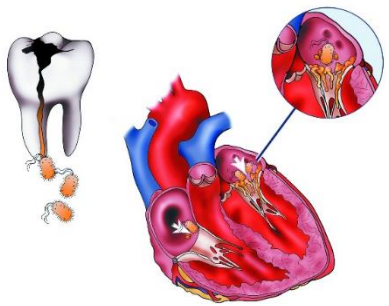
- rheumatic heart disease-linked to group A beta-hemolytic *Streptococcus* (also known as strep throat)-was a predisposing cause for EI, especially in developing countries.

Rheumatic fever and glomerulonephritis are post-streptococcal immune sequelae that may lead to heart complications.

- In developed countries, however, degenerative valvular disease has become the primary predisposing factor due to aging populations and improved management of infections. there has been a rise in cases related to prosthetic heart valves, pacemakers, and defibrillators, further increasing risk .

- Dentists are often referred to as potential culprits in cases of infective endocarditis. Any periodontal procedure, including tooth extractions, can introduce bacteria from the oral flora into the bloodstream, leading to bacteremia. This risk is particularly concerning for individuals with certain heart conditions, as transient bacteremia can potentially trigger infective endocarditis.

However, it’s important to note that bacteremia isn’t exclusive to dental procedures. Everyday activities, such as brushing teeth or chewing gum, can also introduce oral bacteria into the bloodstream, albeit in lower quantities.



- Medical procedures such as hemodialysis, dilation procedures, colonoscopy, and cystoscopy similarly carry a risk of bacteremia, which can lead to infective endocarditis in susceptible individuals.

For these high-risk groups, antibiotic prophylaxis is often recommended to prevent the development of infective endocarditis. We'll discuss more about prevention strategies at the last of the lec.

Microbiology Overview

- The microbiology of the disease has also changed, and staphylococci, most often associated with health-care contact and invasive procedures, 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. Enterococcus forms 5-10% from cases so all together 90% As we said before few slides :)

Types and Causes of Infective Endocarditis

1. **Native Valve Endocarditis**: This form affects the natural heart valves that a person is born with.

2. **Prosthetic Valve Endocarditis**: Occurs in patients with (prosthetic) heart valves, divided into 2 types
    1. **Early Prosthetic Valve Endocarditis**:
        *Develops within the first month after valve replacement surgery.
        *Commonly caused by coagulase-negative staphylococci.
    2. **Late Prosthetic Valve Endocarditis**:
        *Occurs more than a month after valve replacement.
        *Usually caused by *Staphylococcus aureus*.

    ***When asked about the most common causative agent of prosthetic valve endocarditis in general (without specifying early or late), the answer is *Staphylococcus aureus*.**

3. **IV Drug Abuser Endocarditis**:
    1. This form typically affects the right side of the heart.
    2. The most common causative organism is also *Staphylococcus aureus*.
        - In developed countries, *Staphylococcus aureus* is the most common species responsible for native valve endocarditis, prosthetic valve endocarditis, and endocarditis in IV drug users.

Microbiology outline

- 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 From the 90%
- 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.

- Infective Endocarditis in Developing Countries:

In these regions, the most common causative organisms are the **viridans** group streptococci, followed by *Staphylococcus aureus*.

Other Causative Agents:

Gram-Positive Cocci: *Enterococcus* species, accounting for about 5-10% from the 90% of cases. These enterococci are divided into enterococci and non-enterococci. They are classified as group D streptococci. The enterococci include *Enterococcus faecalis* and *Enterococcus* faecium

The non-enterococci are referred to as *Streptococcus bovis*.

Gram-Negative Bacteria:

-Represent approximately 10% of cases.

-Includes the HACEK group (Haemophilus species, Aggregatibacter, Cardiobacterium, Eikenella, and Kingella), which are more associated with subacute or nosocomial (healthcare-associated) endocarditis.

Non-Bacterial Causes (Fungal Endocarditis):

Fungi such as *Candida albicans*, *Aspergillus flavus*, and *Histoplasma capsulatum* can also cause endocarditis.

Other Gram-Negative Pathogens:

Rare pathogens like *Coxiella burnetii*, *Brucella*, and *Bartonella* can also be responsible in certain cases. They have chronic course and worse prognosis

الجدول مهم جدًا خاصة المعلومات في المربع الأحمر غالبًا بتيجي بالامتحان هاي الخصائص وبسأل عن نوع البكتيريا

To distinguish *Staph* species (+ve) from *Streps* and *Enterococcus* species(-ve)

helps differentiate between *Streptococcus* species

	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

Complete hemolysis

no hemolysis

Partial hemolysis

association with colon cancer in older patients

Microbial Causes-1

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

- Streptococci subdivided into groups according their hemolytic reaction on blood agar in vitro & by serotypes according to surface cell wall specific carbohydrate antigens.
- Remember that viridans group is **not groupable by the Lancefield classification**

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 which aids in adherence to surfaces, contributing to their role in dental plaque and caries formation..
- *St. mutans*, *St. mitis* *Streptococcus sanguinis*, *Streptococcus salivarius*, and *Streptococcus anginosus* ,accounted for many cases, and tend to be less susceptible to penicillins.

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

Although transient bacteremia can occur during daily activities like brushing teeth or chewing, not everyone develops infective endocarditis. **why**?
To cause infective endocarditis, bacteria need specific virulence factors that allow them to adhere to heart valve leaflets or heart tissue. These virulence factors are crucial for the development of infection in susceptible individuals.

Microbial Surface Components Recognizing Adhesion Matrix Molecules (MSCRAMMs):
•MSCRAMMs are surface proteins that enable bacteria to bind to host tissues, such as heart valves, enhancing their ability to attach and establish infection" virulence factor". حكاه الدكتور بس غالبا مو مهم

Microbial Causes-1B

❖***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.

❖*Enterococcus species* (*E. fecalis, E. faecium*) after urinary catheterization or colon cancer (bovis) are responsible for up to 5-10% of cases; some strains may be resistant to penicillin,vancomycin so we give teicoplanin

❖Because endocarditis can be life-threatening, empirical treatment is often started before culture results are available. Glycopeptides (like vancomycin) combined with an aminoglycoside (such as gentamicin), typically administered IV .. Treatment is prolonged due to the poor blood supply to the vegetation (infected mass) on heart valves.

Let's go into new classification at this lec, we have multiple ones but don't worry we're almost done!!

- **Acute vs Subacute Endocarditis**
  - Acute Endocarditis: Rapid progression within days to weeks; severe and can be fatal.
  - Typically caused by *Staphylococcus aureus*, *Group B Streptococci*, and *Streptococcus pneumoniae*
  - Subacute Endocarditis: Slower progression over weeks to months; may also lead to serious complications.
  - Often associated with viridans group streptococci, coagulase-negative staphylococci, gram-negative bacteria (HACEK group), and fungal infections.

Type of Endocarditis	**Acute Endocarditis**	**Subacute Endocarditis**
Progression	Rapid progression (days to weeks); severe and can be fatal.	Slower progression (weeks to months); may lead to serious complications.
Common Causes مهم	**Staphylococcus aureus, Group B Streptococci, Streptococcus pneumoniae**	**Viridans group streptococci**, coagulase-negative staphylococci, gram-negative bacteria (e.g HACEK group), fungal infections.

Additional important table to not get confused :)

Factor	Developing Countries	Developed Countries
Most common Predisposing Factor (There're other factors such as colonoscopy but we're talking about the most common only)	- **Rheumatic heart disease** (damage to heart valves from previous infection with sterp. type A)	- **Prosthetic heart valves**
Most important Microorganisms	**Streptococcus viridans**	- **Staphylococcus aureus**

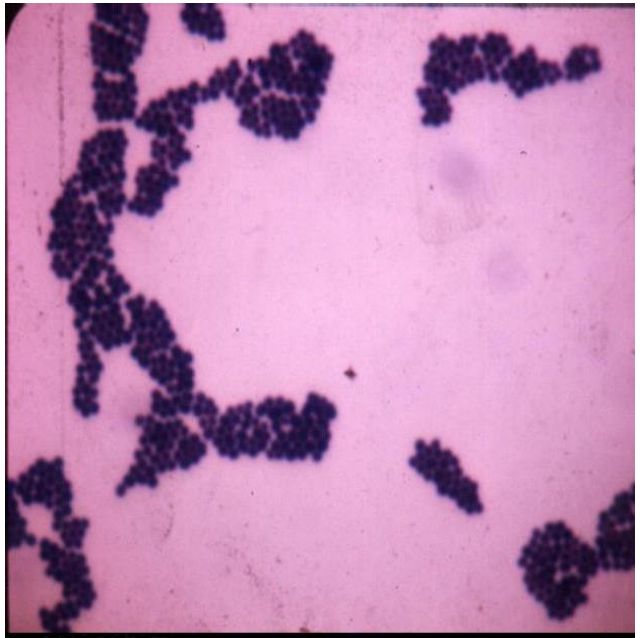
Predisposing factors describe the underlying conditions that make endocarditis more likely, while **microorganism classification** focuses on the specific bacteria causing the infection

Extra cascade for Rheumatic heart fever for further understanding :

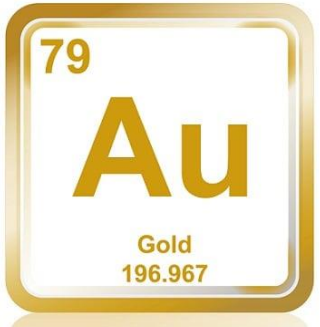
Streptococcus pyogenes infection (strep throat) → **Rheumatic fever** (immune reaction to strep pyogenes cuz of M protein) → **Rheumatic heart disease** (valve damage due to inflammation) → **Bacterial entry** (through dental procedures or bacteremia) → **Infective endocarditis** (caused mainly by **viridans group streptococci** or other bacteria on damaged heart valves)

Streptococci-Staphyloccoci

For Strep identification, it's important to note that this bacterium typically forms chains , it has alpha hemolysis and green sheen appearance



For Staph aureus identification, it's important to note that this bacterium typically forms clusters.
For its growth, a differential and selective agar called mannitol salt agar is used. The reason we call it *aureus* is because it turns the agar yellow (aureus in Latin means golden, aurum means gold.)



Microbial Causes-2

- 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 . these bacteria are often linked to nosocomial (hospital-acquired) infections ✨ المريض بلقطهم من المستشفى

- Clinically, these bacteria spp. causing subacute or chronic course(Dr didn't talk a lot about chronic), and often present with embolic lesions from large biofilm vegetations in heart valves .

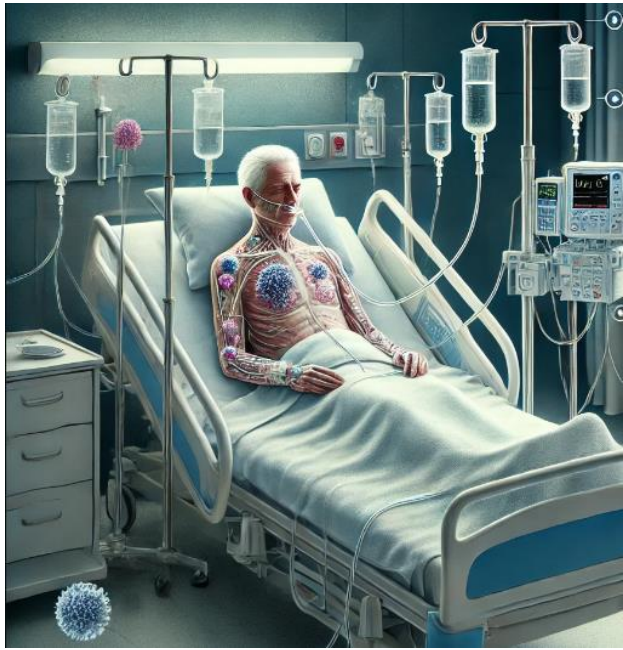
- Most cases of fungal endocarditis occur in patients who are receiving prolonged antibiotics or intravenous nutrition through central vascular catheters.. Immuno-compromised patients. Come with elderly ICU patients on total parenteral nutrition and broad-spectrum antibiotics. خلي هاي الصورة الذهنية ببالك

- (prognosis of fungal infx is much worse than bacterial)

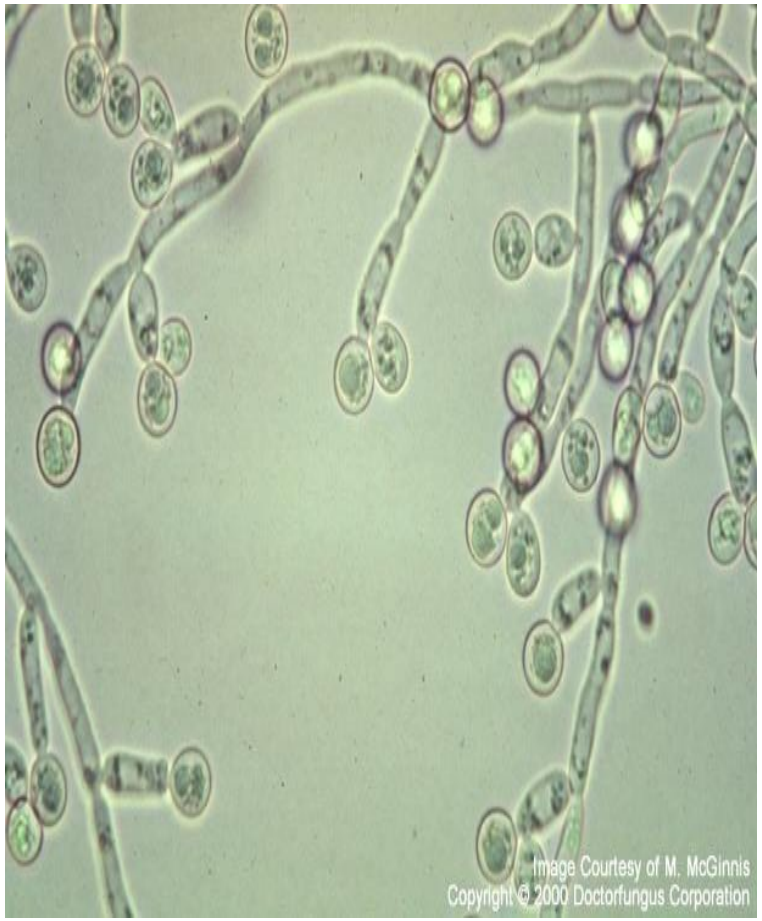
Yeast & Filamentous Fungi

- The most common species is ***Candida albicans***, followed by other less common *Candida spp.* (C. glabrata, C. krusei, C. Tropicals قرأهم ع السريع).
- 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(2nd most common fungal infx after Candida) .. Immuno-suppressed patients -Remember the stereotype which we talked about in prev. slide-

Extra pic for the stereotype of fungal infx pts.



Candida albicans Pseudohyphae



Candida albicans is a common yeast that grows on universal agar. It forms white, creamy, waxy, smooth colonies that are glabrous and smooth on various dextrose agars.

If I take these colonies and incubate them with serum from individual at 37°C, it will form **germ tube**, which is a test used to distinguish **Candida albicans**.

Alternatively, we can inoculate a different agar, called cornmeal agar. After incubation at room temperature, we examine the cultures under the microscope, where we observe the formation of chlamydospores. This includes **pseudohyphae** with **chlamydospore** formation. (pic on the **left**

-Candida are yeasts, but they also exhibit pseudohyphae and true hyphae in pathological conditions. This is why we refer to it as pseudohyphal formation with chlamydospore formation as well

•**All here is additional for ur better understanding**

•Candida species are typically **yeasts**, which means they usually exist as single-celled organisms that reproduce by **budding**.

Pseudohyphae:

•Under certain conditions (such as in infections or in response to environmental stress), Candida can form pseudohyphae.

•**Pseudohyphae** are chains of elongated **yeast cells** that remain attached to each other, resembling hyphae (the branching filaments seen in molds) but lacking the complete separation seen in true hyphae. The cells in pseudohyphae appear **elongated** and **connected**, but they do not have the full structural integrity of true hyphae.

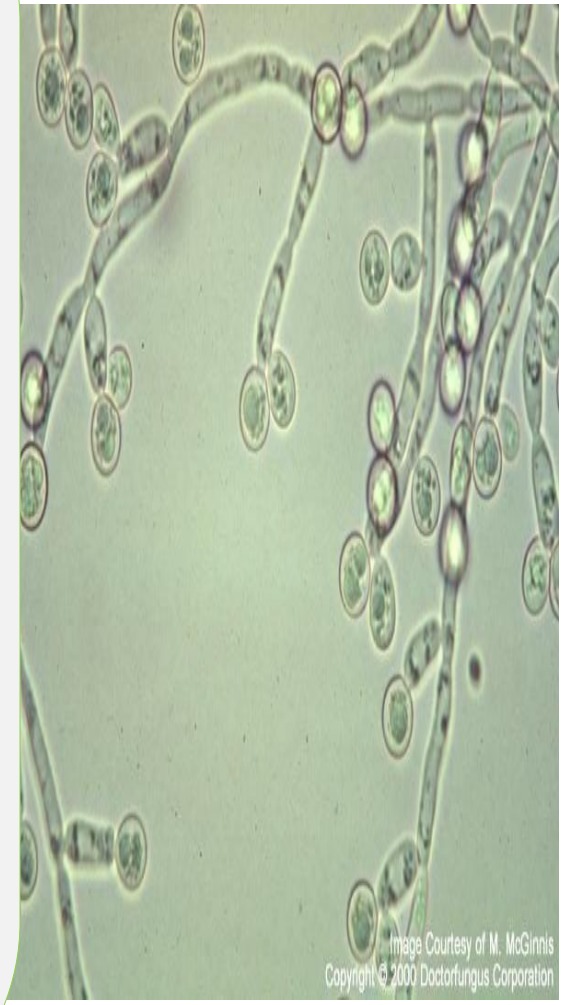
•**Pseudohyphal formation** typically happens when Candida switches from a **single-cell yeast form** to a more **invasive form** (like pseudohyphae) in response to changes in the environment (e.g nutrient depletion or in tissue invasion).

Chlamydospore Formation:

•**Chlamydospores** are a **specialized type of spore** that certain fungi, including **Candida**, form under **stressful conditions**.

•These are thick-walled, **resting spores** that allow the organism to survive in unfavorable environments.

•In **Candida**, the chlamydospores form at the tips of the **pseudohyphae**, where the cells appear swollen and **round**. This gives the appearance of **chlamydospore formation** under the microscope.

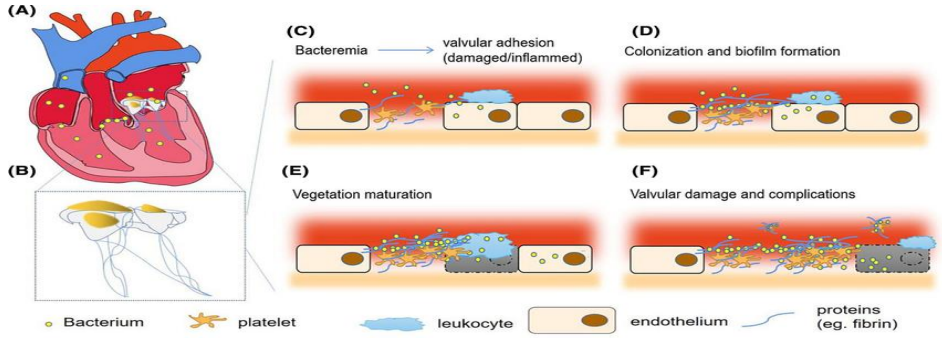


Pathophysiology

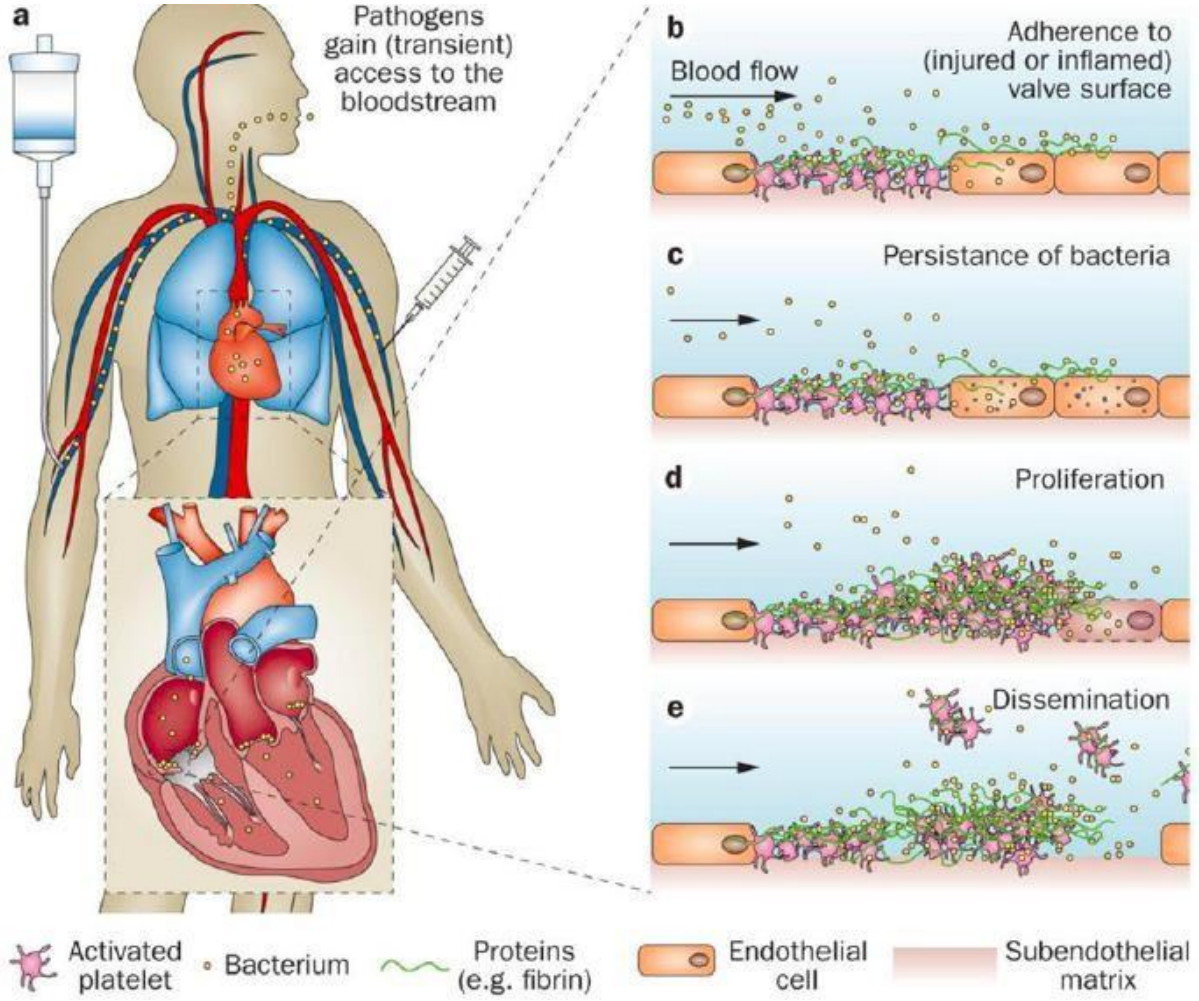
- The healthy cardiac endothelium is resistant to frequent bacteremia caused by daily activities such as chewing and tooth brushing.

- Bloodstream infection is a prerequisite for development.
- The development of IE requires the simultaneous occurrence of several independent factors we've talked about all of them previously: 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 زي كانها مدفونة' 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. It can have a dislodgement so It can move from one place to another and cause vascular complications even in non-cardiac sites.

Extra image



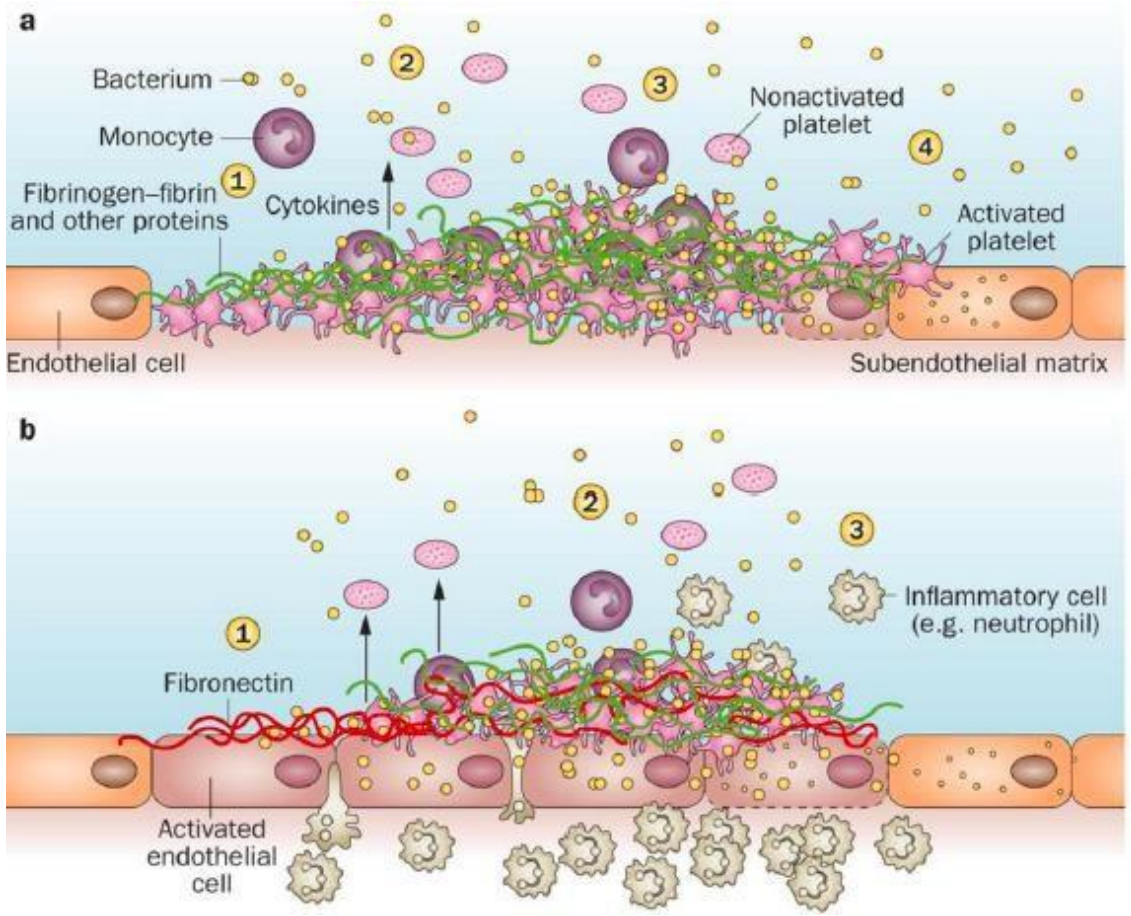
Transient bacteremia occurs when bacteria gain access to the systemic circulation, either through a catheter, IV drug abusers, or a dental procedure. Once in the bloodstream, bacteria can colonize, attach, and adhere to tissues. They can proliferate, and in the case of **Staphylococcus aureus**, they may even become intracellular. This can lead to the development of infective endocarditis in the patient.

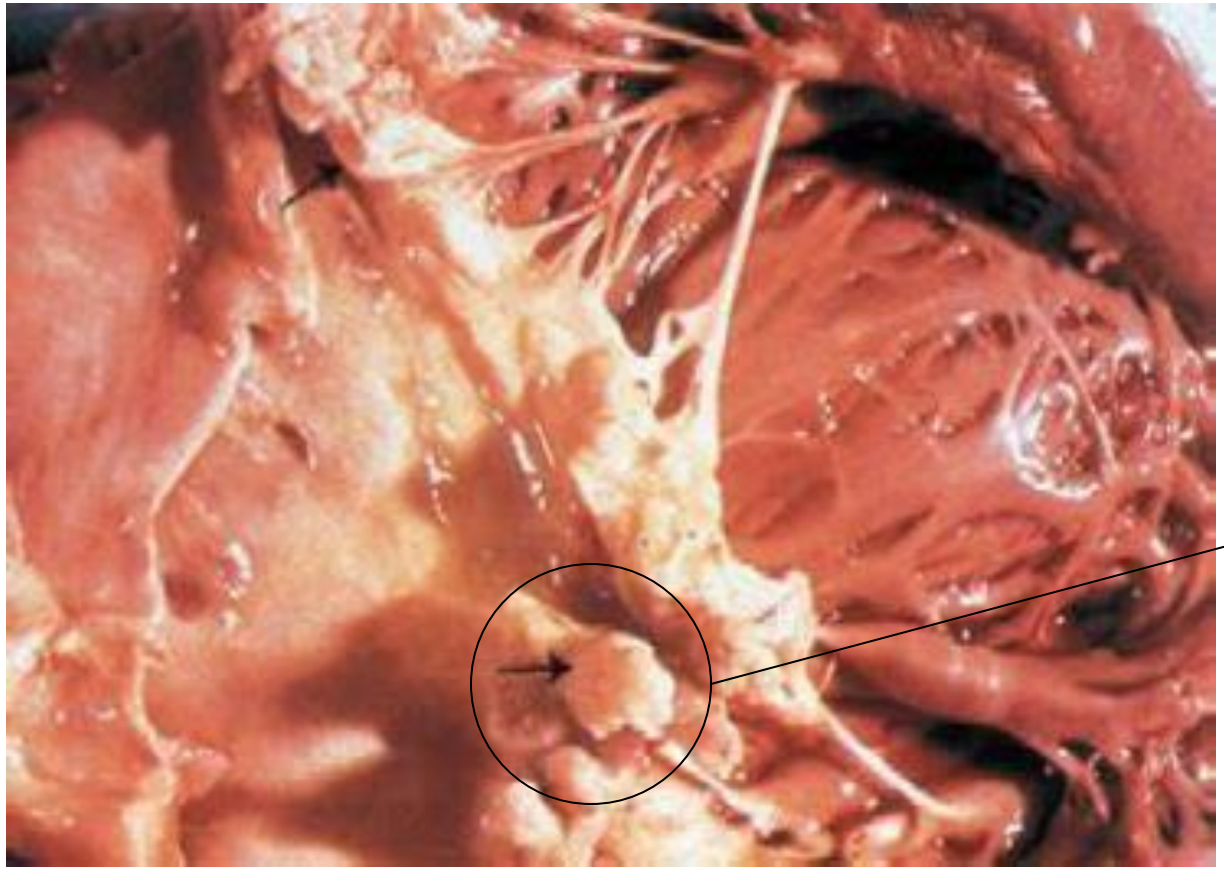


- What is the diff between two pics?
- In endocarditis, there is both a native valve and a prosthetic valve. When a prosthetic valve is present, it can induce mechanical injury.

- 1st pic show prosthetic valve and mechanical injury. There is no continuous epithelial lining

- While in 2nd pic there is native valve endocarditis





This is an autopsy of the heart.

This is the vegetative mass associated with endocarditis.

It can become dislodged, or part of it may break off.

Even when it remains in place, it can lead to valve damage.

- The most commonly involved valve in endocarditis is the mitral valve, but In IV drug abusers, the tricuspid valve is more commonly affected (the reason if u're interested in the next slide)

•All here is extra so u can skip :))

•**Mitral Valve Involvement (Non-IV Drug Users)**: In most cases of infective endocarditis, bacteria enter the bloodstream through routes like dental work or skin infections. The mitral valve is often affected because it experiences high-pressure blood flow. Any structural abnormalities or turbulent blood flow can make it easier for bacteria to attach and form vegetations on this valve.

•**Tricuspid Valve Involvement (IV Drug Users)**: For intravenous drug users, bacteria often enter directly into veins and travel through the right side of the heart first. The tricuspid valve is the first valve that bacteria encounter in the bloodstream. As a result, it becomes the most common site of infection in IV drug users.

- **Additional Story لتغيير جو المحاضرة which u can also skip ;)**

كيف تسببت حبة فشار علقت بين اسنان مريض في دخوله لعملية تبديل صمامات قلب لمدة 7 ساعات 🧐؟

علق جزء من الفشار بين أسنانه لمدة ٣ ايام وحاول اخراجها باستخدام أدوات غير معقمة (قلم اوخيط غير معقم ..الخ) وبعدما نجح في ازالتها بأدواته المبتكرة بعد شهر ظهر معه ألم في الأسنان، بعدها بدأت تظهر عليه أعراض شبيهة بالانفلونزا وإرهاق وصداع فقرر الذهاب لطبيب وتم تشخيصه بالتهاب البطانه الداخليه للقلب "Endocarditis" وبعدها فورا أجري له عملية تبديل صمامات القلب بعد الضرر بسبب العدوى
ومن حسن حظه دخل عملية مدتها ٧ ساعات انقذت حياته ، كان سبب الالتهاب دخول بكتيريا لمجرى الدم ومن طرق دخول البكتيريا عن طرق الفم بسبب جرح يكون سببه عود أسنان خشبي او اقلام او أداة حادة غير معقمة وهذا ما قد استخدمه ليخرجها من بين أسنانه...

Clinical features

- 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, and, if untreated, progresses to death within weeks. Again, often caused by **Staphylococcus aureus**, **Streptococcus pneumoniae**, or **Group B streptococci**.

- Subacute endocarditis follows an indolent very slow 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 (an abnormal heart sound.) are usually indicative of the predisposing cardiac pathology rather than of endocarditis, valvular damage and ruptured chordae may result in new regurgitant murmurs.

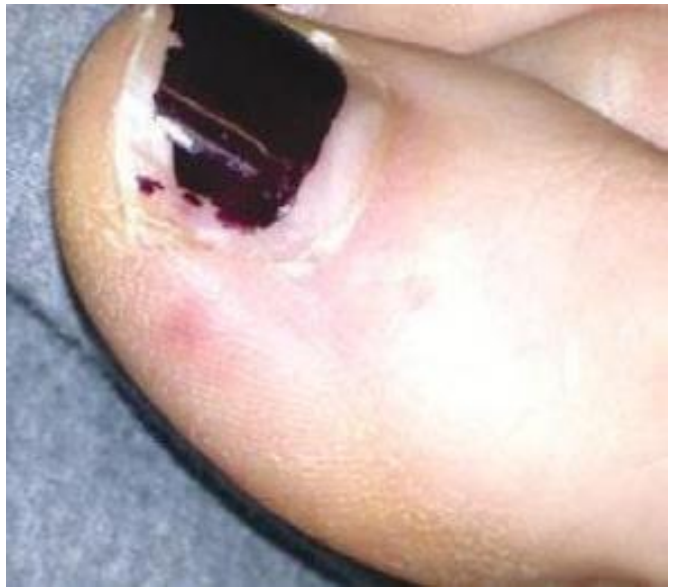
- Congestive heart failure (CHF) Cardiac manifestations can lead to congestive heart failure, we have Non-Cardiac ones which are the topic of next slide
- develops in 30–40% of patients as a consequence of valvular dysfunction.

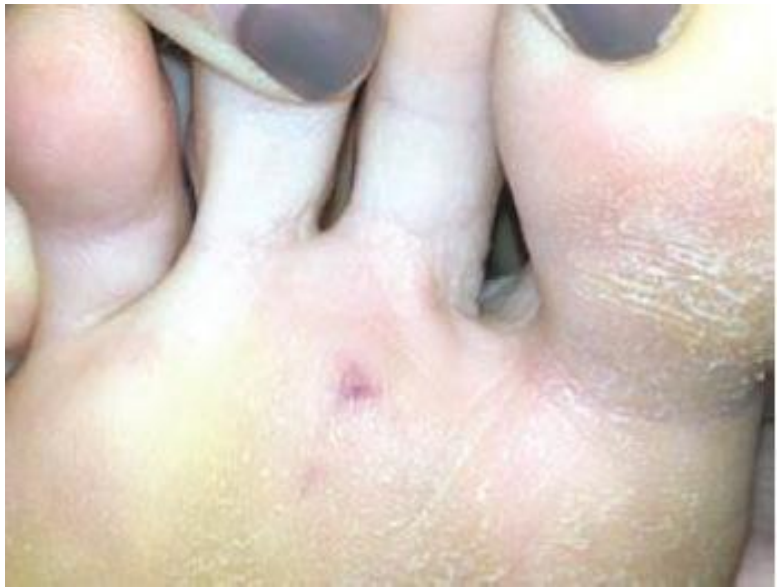
If a patient presents with a **fever** of unknown origin – meaning there is no obvious focus of infection, such as in the respiratory system, GI system, or extremities- When fever of unknown origin is combined with a newly onset **murmur**, it suggests infective endocarditis until proven otherwise. Similarly, if there is fever of unknown origin along with a change in a pre-existing murmur, it is also indicative of infective endocarditis..

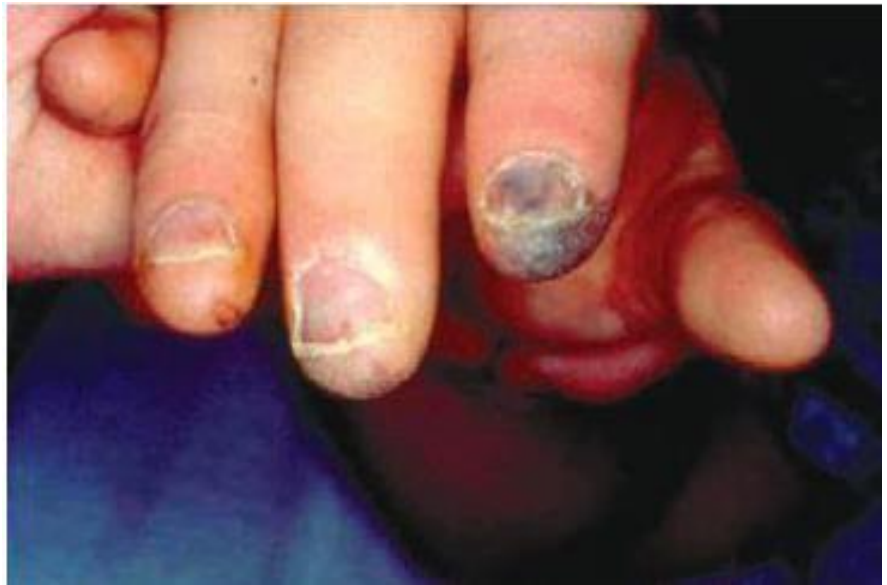
Noncardiac Manifestations

- 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.



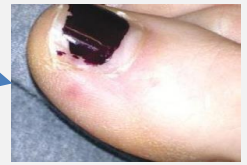




- **In infective endocarditis, there are both vascular and immunological phenomena.**

Vascular Phenomena it causes Emboli: Fragments or masses from the heart can travel to other organs, causing ischemia or infarction. This can affect: Brain , Kidneys , Distant organs

1 Janeway Lesions

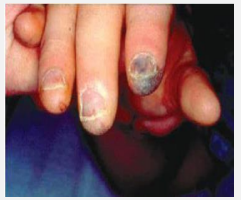


*Painless erythematous lesions typically found on the palms or soles.(the little red spot)

2 Conjunctival Hemorrhages

*Small hemorrhages in the eye

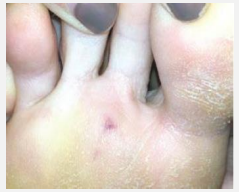
3 Subungual Hemorrhages



*Small blood spots under the nails

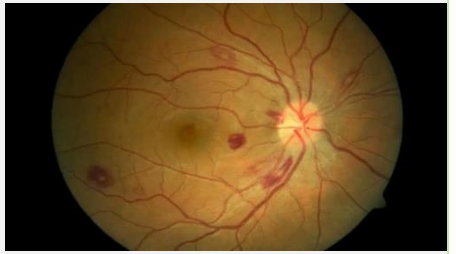
immunological phenomena

1 **Osler Nodes**:



*Painful, raised lesions on the palms or soles, associated with immunological responses.

2 **Roth Spots**



*Retinal hemorrhages with a clear center. These are distinct from conjunctival hemorrhages and are an immunological phenomenon.

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

Modified Duke Criteria for Infective Endocarditis:

*Major Criteria:

1.Positive Blood Cultures:

1. The presence of typical organisms for infective endocarditis, such as *Staphylococcus aureus*, *Strep viridans*, etc.

2.echocardiography :

1. **Transesophageal echocardiography** or **transtracheal echocardiography** is the most recommended.
2. The presence of a **vegetative mass** (a mass of infected tissue) seen oscillating in the heart is a key sign.

*Minor Criteria:

1.Fever: A temperature of >38°C.

2.Culture of Non-Typical Organisms: Organisms that are not typically associated with infective endocarditis or situations where the organism cannot be cultured.

3.Vascular and Immunological Phenomena: which we talked about before few slides

As a result , we'll have three possibilities (the following is from Dr, study it but actually it has not been in past papers ever)

Definite Infective Endocarditis(IE)
A diagnosis of definite infective endocarditis requires:
- **Two major criteria**, or
- **One major and three minor criteria**, or
- **Five minor criteria**.

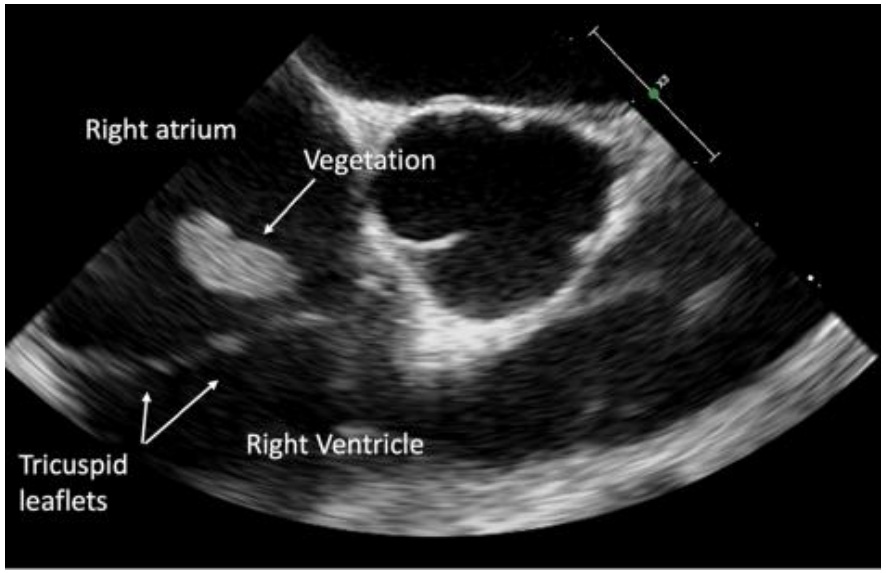
Possible IE
When the criteria don't fully meet the requirements for definite endocarditis but still suggest it.

Rejected IE:
If the clinical findings do not meet any of the above criteria, the diagnosis is rejected.

- 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

Extra pic for Echocardiography result



•Some organisms that cause infective endocarditis are **difficult to culture** and may require **serological testing**. In such cases, these organisms contribute to the **minor criteria**.

•Echocardiography showing an oscillating mass or turbulent blood flow across the heart valves are considered **major criteria**.

Management

❖ ANTIMICROBIAL THERAPY

- Vancomycin plus Gentamicin initiated immediately after blood samples are taken for cultures, they're given IV

- Extended courses of parenteral therapy with bactericidal (or fungicidal) agents are typically required. These drugs are used to address the **metabolically inactive vegetative mass** in the heart, which is difficult to treat due to lack of blood supply.

❖ Surgical Treatment. In some cases, a **cardiothoracic surgeon** may be needed to address structural defects that create a suitable environment for bacteria to colonize the inner parts of the heart and the heart valves.

A common **mistake** is Not to start appropriate antibiotics immediately, before receiving the sensitivity test results. The **correct** approach is to start with vancomycin and gentamicin to cover likely pathogens.

Blood samples should be taken **before** initiating antibiotics (✨ خطوة مهمة رح تتبهدلوا عليها بالكلينيكال ✨).

Three blood cultures should be taken from different venous sites, every 6 to 8 hours on the same day to improve the chances of detecting the bacteria. Timing is crucial in ensuring that cultures reflect the true bacterial presence, not a negative result caused by premature initiation of antibiotics.(again, these details – which are no. of blood cultures and interval between samples- probably for your medical info not for exam purposes, but study it to be on the safe side)

PREVENTION

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

Should Antibiotics Be Given?

•The decision to administer **antibiotics** depends on the risk and presence of predisposing factors.

Dr Nader's Advice: It's always better to **be on the safe side** and start antibiotics if there's any doubt, especially in patients with a higher risk for infective endocarditis.

PAST PAPERS

ـ نُقِل من ملف مالك ابو رحمة

•Catalase negative, coagulase negative alpha hemolysis and causes subacute endocarditis

ANSWER: Streptococcus viridans

•Rheumatic fever is a complication of?
A) Group B strep
B) Group A strep
C) Viridans group
ANSWER: B

•Which of the following is the most common cause of infective endocarditis in developing countries:
A) Staphylococcus aureus
B) Streptococcus viridans
C) Fungi
D) Haemophilus influenzae

ANSWER: B

•Infection with subacute infective endocarditis is associated with the following:
A) Abnormal valves
B) Congenital deformities
C) Rheumatic lesions
D) A & B
E) All A, B, and C

ANSWER: E

• **Most common cause of acute endocarditis :**
A) S.aurues
B) S.epidermidis
C) Aggregatibacter

ANSWER: A

•The main mechanism of rheumatic fever:
A) Bacterial or infectious cause
B) Autoimmune by antibodies against M protein
ANSWER: B

11. All of the following statements regarding viridans streptococcal group infective endocarditis are correct except the causative agents are correct except:
A) Causative agents are gram +, catalase -, a Hemolytic optochin resistant and bile and insoluble
B) pathogenesis involve dextran (biofilm)-mediated adherence onto both normal or damaged heart valve and to each other (vegetation)
C) St. Mutans, st. mitis Account for many IE cases, and tend to be less susceptible to penicillin
D) VGS causes hectically febrile illness that rapidly damages cardiac structures, seeds extra cardiac sites, and, if untreated, progresses to death within weeks
E) Common cause of dental carries hence prophylactic antibiotics prior to dental work for individuals with damaged heart valve is recommended

ANSWER : D

12. Given the diagnosis of infective endocarditis which of the following is incorrect:

A) IE is a multisystem disease that results from infection, usually bacterial, of the endocardial surface of the heart and the epithelial lining of heart valves
B) Rheumatic heart disease remains the key risk factor for infective endocarditis in low income countries
C) Gram positive cocci are the most frequent causative agents of IE
D) A gram positive, catalase positive, mannitol fermenter is the most common cause in IE in low-income countries
E) Vancomycin plus gentamycin initiated immediately after blood samples are taken for cultures

ANSWER : D

لا يألفنَّ أحدكم مُصابَ أخيه -وإن طال-، ولا يغفلنَّ بالدعاءِ عنه متى بقيَ البلاءُ أو زال

Additional sources

1. PAST PAPERS
2. المصدر الأهم

VERSIONS	SLIDE #	BEFORE CORRECTION	AFTER CORRECTION
V1→ V2	19,31 _____ 20	Acute IE is caused by **Group A Streptococci** ---------- **Predisposing Factors (in the table)**	Acute IE is caused by **Group B Streptococci** ---------- **Most common Predisposing Factor (There're other factors such as colonoscopy but we're talking about the most common)**

امسح الرمز و شاركنا بأفكارك لتحسين أدائنا!!