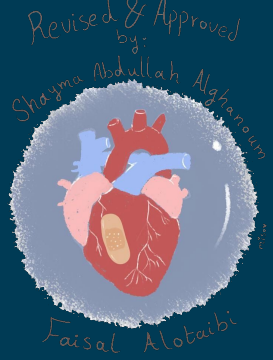
Infective Endocarditis

TEAM 439
MICROBIOLOGY



VERSION 1

Objectives

- Define infective endocarditis
- Discuss the pathogenesis, epidemiology and classification of infective endocarditis
- List important risk factors
- Describe the clinical presentation and complications
- Discuss the clinical and laboratory diagnosis
- Discuss the causes and diagnosis of culture negative endocarditis
- Discuss the management and prophylaxis

Colour index:

Red: Important & Notes.

Grey: Extra info & explanation.

Dark Blue: Original Text

Any future corrections will be in the editing file, so please check it frequently.

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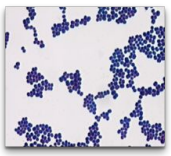
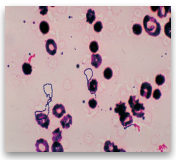
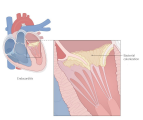
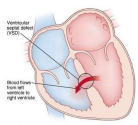
Introduction & Overview

Endocarditis usually refers to infection of the endocardium. The term can also include non-infective endocarditis, in which sterile vegetations (platelet and fibrin thrombi) form on cardiac valves and adjacent endocardium in response to factors such as trauma, circulating immune complexes, vasculitis, or hypercoagulable state such as pregnancy.. Noninfective endocarditis sometimes leads to infective endocarditis. Both can result in embolization and impaired cardiac function. Noninfective endocarditis is much less common than infective endocarditis.

Staphylococcus Aureus	Organism	Streptococcus Viridans
Gram + cocci in clusters 	**Morphology and Description**	 Gram + cocci in chains
Catalase +ve 		 Catalase -ve
Coagulase +ve 		 Alpha-hemolytic Green discoloration on blood agar
High Virulence So it can cause endocarditis even if the heart is healthy.	**Virulence**	**Low virulence** So it can only cause endocarditis if the heart is already damaged.
Skin flora	**Source**	**Oral flora**
اوكي هنا مثلا يجيك IV Drug user ماعنده أي مشكلة في الهارت . staph aureus = skin flora وعارفين اكيد ان وبرضو عارفين أن في وضعه الستير لايزيشن والانفكشن كنترول بيكون بالزاوية Not to mention that IV drug users usually share the same needles الزبدة ستاف اوريس بتدخل ال blood stream Bacteriemia → it will go to the heart and multiply there Result is: Fast destruction & breakdown of the endothelium + ↑fibrin, ↑platelets, ↑microbes, ↑lymphocytes, ↑destructive products. = Vegetation, which is a mass of platelets, fibrin, microcolonies of microorganisms, and scant inflammatory cells.   الveggitations في هالحالة تكون مرره fragile وتتفكك بسهولة So it might breakdown, metastisize, embolize other places e.g. brain, kidney, skin, and anywhere in the body	**Pathophysiology**	يجيك شخص عنده أي مشكلة في القلب تسبب له abnormal blood flow المسببات كثيرة لكن نفترض عنده Ventricular septal defect،  اللي بيحصل هو أن الدم بيجي بسرعة من LV ويروح لـRV ومن قوة الflow الجدار حق RVبيصير damaged وطبعًا زي أي repair mechanism بيتكون فيه microthrombi والخ. ليه؟ Because when endocardium is injured, it loses it anticoagulant activity and becomes procoagulant. Note that microthrombi is a very welcoming & protective place for bacteria :) الزبدة بعدين مثلا يروح يخلع سن او يسوي أي dental procedure . وزي مو عارفين ان strept viridans فلورا بالفم. بتصير bacteremiaوتروح البكتيريا تلقى damaged heart tissue وبتفرز glucans and polysaccharides عشان تساعدها بالadherence Result is: Colonization + slow destruction of the endothelium + Fibrosis = **fibrotic** vegetation (mass or clump)
Fast & Stormy Since its develops over a short period, usually the patient will present with leukocytosis	**Onset**	**Slow** Since its develops slowly and for a long period, usually the patient will develop leukopenia anemia, because the bone marrow is not making enough WBC on the long term.

Diagnosis of Endocarditis

1- Microbiology (Positive blood cultures).
2- Additional laboratory non-specific tests. (ESR, CRP).
3- Radiology (Echocardiography).
4- Urinalysis (Blood, protein, and pus in urine)
5-ECG

Infective Endocarditis (IE)

An infection of the heart's endocardial surface (which involves heart valves).

Epidemiology:

- Incidence: 1.7— 6.2 / 100 000 person years.
- Male:Female 1.7.
- Becoming a disease of the elderly.
- Median age:
  - Pre antibiotics era —35y. It used to occur in younger people.
  - Now —58y.
- Due to two factors:
  - The decline of rheumatic heart disease.
  - The increasing proportion of elderly.
    (Mainly intravenous drug abusers in young)

★Classified into four groups:

Native Valve IE

★ **Staphylococcus aureus** (~30%, especially in IV drug users)
★ Strept., mostly **S. viridans.** (~25%) Especially post dental procedures.
- Enterococci (5-10%).
- Gram negative bacteria = HACEK (5%).
- Fungi (rarely).

Prosthetic Valve IE

Prosthetic Valve:

- 7 -25 % of cases of infective endocarditis.
- 0.94 per 100,000 bioprosthetic.
- Initially mechanical valves at greater risk for **first 3 months**, then risk same at 5 years:
  - 1-3.1% risk at 1 year.
  - 2-5.7% at 5 year.

"الرسك بشكل عام موجود لكنه أكثر شي في الثلاث شهور الأولى"

Early (≤12 months): first year after operation	**Late (≥12 months):** after first year
- 1-3.1% Staph aureus. ★ **Staph epidermidis.** Coagulase -ve bacteria, skin flore, usually comes from the patient or surgeon's skin, has a tendency to infect **prosthetics,** as it almost never infect native/normal tissue.	Same organisms of early in addition to Strept viridans & enterococcus. - 2 - 5.7% Staph aureus. ★ **Staph epidermidis.** ★ **Strept. Viridans.** oral flora - Enterococcus.

Intravenous drug abuse (IVDA) IE

★ **Staph. Aureus** (50- 60%). Any organism can be seen but generally staph. Aureus.

Intravenous drug users tend to get their right-sided heart valves infected because the veins that are injected drain into the right side of the heart.

Nosocomial IE

A rare complication of nosocomial bacteraemia; however, it is an infection of great importance because of its high mortality and because in many cases it is potentially preventable.

Infective Endocarditis (IE)

Further Classification:

	Acute **Few days**, Very sick + comatose	Subacute **1-2 weeks**, mildly sick
Predisposing condition	Affects **normal** heart valves	Often affects **damaged** heart valves
Onset	**Rapidly** destructive Stormy onset	**Indolent** خامل nature
Fatality	Usually **fatal within 6 weeks (if not treated)**	Usually **fatal by one year (if not treated)**
Causative organism	**Staphylococcus** → Metastatic foci (Specially with **staph. Aureus**)	**Strep. Viridans** Coagulase -ve, usually after dental procedures.
Symptoms	- **High grade fever** and chills. - Shortness of breath. - Arthralgia (Joints pain) - Myalgias (Muscle pain). - Abdominal pain. - Pleuritic chest pain - Back pain. Due to spread of infection, the patient may have more symptoms depending on the other infected areas.	- **Low grade fever.** - Anorexia. Eating disorder, rarely associated with IE. - Weight loss. - Fatigue. - Arthralgia (Joints pain) - Myalgias (Muscle pain). - Abdominal pain. - Nausea / vomiting..
Notes	Both can infect native and prosthetic valve.	

❖ The onset of symptoms is usually **~2 weeks or less** from the initiating bacteremia.

★ Pathophysiology:

01 **Injury of endothelium**
Turbulent blood flow **disrupts the endocardium** making it "sticky".

02 **Bacteria access to bloodstream**
Bacteremia delivers the organisms to the endocardial surface. From normal flora.

03 **Adhere to endocardium**
Adherence of the organisms to the damaged endocardial surface.
If there was no adherence, the blood flow will take away the bacteria.

04 **Invasion and cause disease**
Eventual **invasion** of the valvular leaflets.

★ **In short: Injury & damage→ Bacteremia → Adherence to endocardium → Invasion & disease**

Infective Endocarditis (IE)

Risk Factors:

1. Cardiac Abnormality/Anomalies

High Risk:	Moderate Risk: (Other valves)	Low/No Risk:
- Previous IE 4.5(2.5 to 9)%. - Aortic valve disease 12 to 30%. - Rheumatic valve disease. - Prosthetic valve. - Coarctation. Congenital narrowing of a short section of the aorta. - Complex cyanotic congenital HD.	- Mitral valve prolapse - Mitral valve thickened leaflets - 5 to 8 times (100/100 000 person years). - Mitral Stenosis. - tricuspid valve. - Pulmonary Stenosis. - Hypertrophic Obstructive Cardiomyopathy (HOCM).	- Atrial septal defect ASD (secundum). - CABG (coronary artery congenital bypass graft). قسطرة

- 75% of patients with IE will have a pre-existing structural cardiac abnormality.
- 10-20% have congenital heart disease.
- People with one of these factors need prophylaxis when they undergo dental procedures

2. Rheumatic valve disease

- Childhood disease
- Predisposition for young in some countries 37%-76% of cases.
- Usually affects mitral valve 85%, secondly, it affects aortic valve 50%.
- Degenerative valvular lesions.
- Mitral valve prolapse and associated mitral regurgitation - 5 to 8 times higher IE risk.
- Aortic valve disease (stenosis or/and regurgitation) is present in 12 to 30% of cases.

3. Injection Drug use & IV drug use:

- 100x ↑ Risk in young people: usually **Staph. aureus.**
- Rates 150-2000/ 100 000 person years.
- Higher among patients with known valvular heart disease.

4. HIV infection:

- A number of cases of IE have been reported in patients with HIV infection.
- It has been suggested that HIV infection is an independent risk factor for IE in IDU.

5. Other risks:

- Poor dental hygiene.
- Hemodialysis. غسيل كلوي because it involves a lot of IV procedures.
- Diabetes mellitus.
- HIV

Infective Endocarditis (IE)

History of prior cardiac lesions

You should ask the patient about history of cardiac injury or damage by any disease. E.g. RHD or atherosclerosis.

Diagnostic Approach

A recent source of bacteremia.

You should ask the patient about recent dental procedures or endoscopy etc..

Clinical Presentation & Physical Examination

- Look for small and large emboli with special attention to the fundi, conjunctivae, skin, and digits.
- ★ Cardiac examination may reveal signs of **new regurgitation murmurs and signs of CHF (worsening murmur)**.
- Neurologic evaluation may detect evidence of focal neurologic impairment.

Signs:

- Continuous fever. (You cannot say a patient has endocarditis unless there is fever, low or high grade)
- Heart murmur.
- Nonspecific signs: **petechiae**, subungual or **"splinter" hemorrhages**, clubbing, splenomegaly, neurologic changes.
- More specific signs: **Osler's Nodes, Janeway lesions**, and **Roth Spots** (seen at the back of the eye with fundoscopy).

الـmainfestations ذي كلها ياتكون ناتجة عن الـemboli والـ detached veggitations غالبًا في حالة الـacute

أو ممكن تكون ناتجة عن vasculitis: غالبًا في حالة الـsubacute

Just like any immune response, immune complexes will form and circulate the body causing antigenemia
This will lead to activation of immune complement → damaging the tissue & Type 3 hypersensitivity reaction.

وأحيانًا الـimmune complexes ذي تتجمع بأماكن زي في الـ capillaries اللي تحت الأظافر وتسوي لك vasculitis ويطلع لك حاجة زي splinter hemorrhages

Other Aspects Clinical Diagnosis

★ Know which valve is involved, is **right or left heart**, and where would emboli go.

The site of infection determine where it will spread in any system organ:

- **Right valves> cause a complication inside the lung (e.g pulmonary embolism)**.
- Left valves> cause a complication in other parts of the body ,e.g back (vertebral osteomyelitis), liver, brain(stoke)...

- Evaluate heart function
- Pump, acute valve dysfunction conduction.
- Look for evidence emboli.
- Look for ant Bleeding (intracranial, elsewhere mycotic aneurysm).

لازم نشوف اذا عنده أعراض ثانية زي headache , back pain and neurological defects لأنه تدلنا على أن العدوى يمكن راحت مكان ثاني.

Diagnostic Approach

1. Positive blood culture results:

★ **A minimum of <u>three</u> blood cultures** should be obtained over a time period based upon the severity of the illness.

معلومة خارجية بس حلوة: اذا عندك مريض endocarditis وسويت كلتشر وطلع لك streptococcus bovis
معناه احتمال كبير جدًا جدًا ان عنده colon carcinoma أو أي GIT Malignancy.
Strpt. Bovis is a normal flora of the gut :) #justsaying

2. Additional laboratory "Nonspecific test":

- An elevated ESR and/or an elevated level of CRP is usually present.
- Most patients quickly develop a normochromic normocytic anemia.
- The WBC count may be normal or elevated.

Additional Tests

- **Abnormal urinalysis:** The combination of RBC casts on urinalysis and a low serum complement level may be an indicator of immune-mediated glomerular disease

Urinalysis may show evidence of gross or microscopic hematuria (blood in urine), proteinuria (abnormal quantities of protein in urine) , or pyuria (pus in urine) caused by the immunologic effects of **endocarditis** on the kidneys.

- **Abnormal ECG:** New AV, fascicular, or bundle branch block. perivalvular invasion monitoring? Pacing?
- **Echocardiogram (more important & Sensitive than ECG)**

Specific signs for endocarditis

Osler's Nodes	Janeway Lesions	Roth spots
Painful erythematous nodules Located on: pulp of fingers and toes. More common in subacute IE	**Painless** and erythematous, blanching macules. Located on palms and soles.	Retinal hemorrhage You can see it in the back of the eye with fundoscopy
		

Non-Specific signs for endocarditis

Petechiae	Splinter Hemorrhages
Often located on extremities or mucous membranes. Some related to vasculitis (immune complex related)	Non blanching Linear reddish-brown lesions found under the nail bed. (Usually do not extend the entire length of the nail)
 	

manifestations are important, and what is seen in hands can be seen in feet as well

Echocardiographic Findings

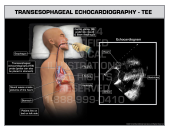
- Presence of **vegetations** defined as oscillating intracardiac mass:
  - On valve or supporting structure
  - In the path of regurgitation jets
  - On implanted material, in the absence of an alternate anatomic explanation
- Abscess:
  - New partial dehiscence of prosthetic valve
  - New valvular regurgitation (increase or change in pre-existing murmur not sufficient)

Improved diagnostic value of echocardiography in patients with infective endocarditis by transesophageal approach (A prospective study):

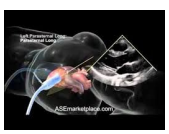
- Eur Heart J, 1988 Jan;9(i): 43.5396 patients were studied consecutively with TEE and TTE
- TEE sensitivity 100 percent for vegetations as compared to 63 percent with TTE
- Both TTE and TEE had specificity of 98%
- 25% of vegetations less than 5 mm, 69% of vegetations 6-10 mm, and 100% of vegetations greater than 11 mm detected by TEE were also observed with TTE

بإختصار اعرفوا أنه إذا سويت الـ echo من الـ esophagus، بيكون sensitive أكثر من لو سويته transthoracic
لكنه more invasive and less comfortable for the patient

TEE (transesophageal echocardiogram) يدخلون المنظار من الـesophagus



TTE (transthoracic echocardiogram) من خارج الجسم



Negative Blood Culture

Why is The Blood Culture Negative?

Previous antibiotics intake
in 50% of the cases of false negative cultures.
(However, if the cause was S.aureus it's usually not affected by antibiotics because it takes 1-2 days to clear it from the body, but less virulent organisms such as s.viridans will be easily killed by antibiotics and the culture will be negative)

Fastidious Bacteria
Bacteria with special nutritional requirements

How hard did you look?

- Negative cultures are usually associated with subacute, valve destruction, CHF.
- Another reason is that it could be non-infectious endocarditis (caused by tumors)

How will you detect the organism if the culture came negative?

Laboratory test	Etiology These organisms need special media & different techniques
PCR of vegetation/emboli	Tropheryma whippelii, bartonella
Histology/stain /culture of vegetation/emboli	Fungus, it grows on regular media but ofc itt will be less sensitive
Prolonged, enriched cultures	HACEK
Lysis centrifugation system (Isolator)	Bartonella, legionella (BCYE), fungal Legionella rarely causes endocarditis & it doesn't grow on regular media
Serology	Endemic fungi, bartonella, **Q fever, brucella** (needs longer incubation period), legionella, chlamydia
Thioglycolate or cysteine supplemented media.	S.aureus satellitism: Abiotrophia (NVS)

Poor Prognostic Factors

- Female (Probably due to hormones etc..)
- S. aureus (more virulent)
- Vegetation size
- Aortic valve problems
- Prosthetic valve
- Older age
- Diabetes mellitus
- Low serum albumin
- Apache II score (Acute Physiologic Assessment and Chronic Health Evaluation II) Is a severity of disease classification system
- Heart failure
- Paravalvular abscess
- Embolic events

Infective Endocarditis (IE)

Complications:

common in patient with IE but it depends on the size of the veggitiation & the organism itself (more virulent).

1. Embolic complications.
2. Local spread of the infection. E.g. abscess around the heart.
3. Metastatic spread of the infection. Lung, back, or kidney.
4. Formation of immune complexes leading to:

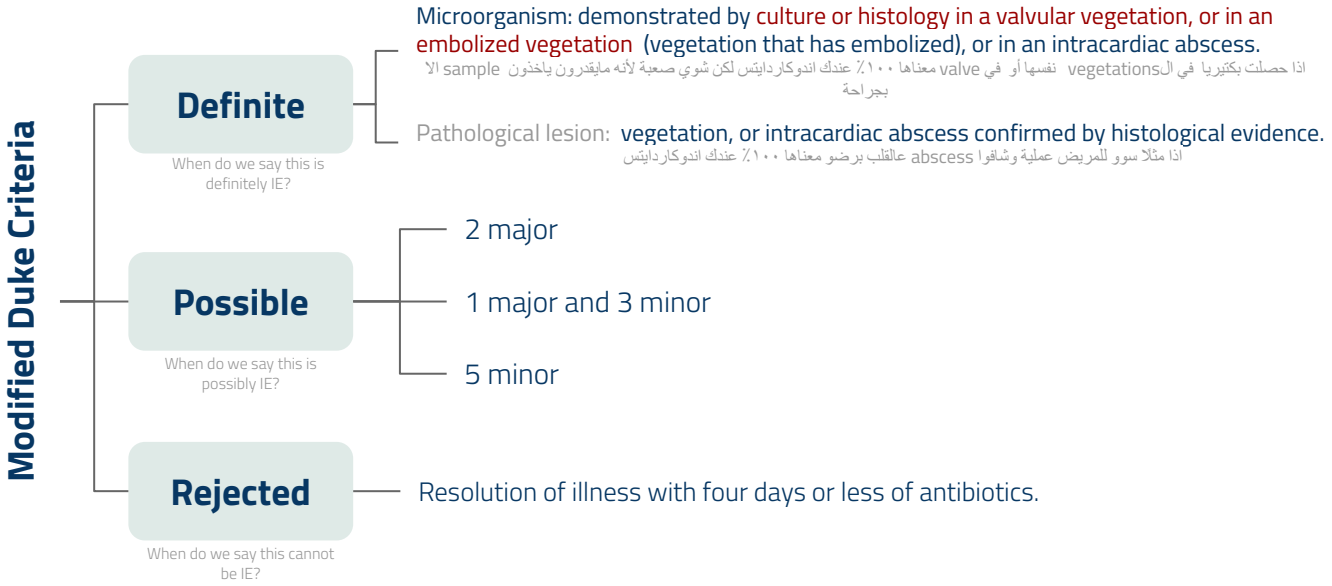
   Glomerulonephritis, arthritis, and vasculitis.

Embolic Complications The vegetation breaks down and travels somewhere else in the body and emboliz or causes infection.	❖ Occur in up to 40% of patients with IE ❖ Predictors of embolization : - Size of vegetation. - Left-sided vegetations. - Virulent organisms (Fungal pathogens, S. aureus, and Strep. Bovis). ❖ Incidence decreases significantly after initiation of effective antibiotics. Includes: 1-Brain: Stroke 2-Heart : Myocardial infarction (Fragments of valvular vegetation or vegetation-induced stenosis of coronary ostia). 3- Lung / Pulmonary circulation: Hypoxia from septic pulmonary emboli. 4-Abdomen: Abdominal pain (splenic or renal infarction) 5-Extremities: Ischemic limbs 6- Eye: septic retinal embolus .
Local spread of infection (inside the heart)	1- Heart failure due to extensive valvular damage. 2- Paravalvular abscess (30-40%): ❖ Most common in aortic valve, IVDU, and S. aureus ❖ May extend into adjacent conduction tissue causing arrhythmias. ❖ Higher rates of embolization and mortality. 3-Pericarditis. 4- Fistulous intracardiac connection Abnormal connection between one of the coronary arteries and a heart chamber or another blood vessel. .
Metastatic spread of infection The infection spreads through bacteremia, not involving the vegetation	1-Metastatic abscess: Kidneys, spleen, brain, soft tissues 2-Meningitis and/or encephalitis 3-Vertebral osteomyelitis (especially staph aureus in older people) 4-Septic arthritis

Put in your mind that Gram -ve bacteria IE is way more severe due to the production of endotoxins.

Modified Duke Criteria

- In 1994 investigators from Duke University modified the previous criteria to include the role of **echocardiography** in diagnosis.
  Here is when they found out that echocardiography is the most sensitive test for endocarditis.
- They also expanded the category of predisposing heart conditions to include intravenous drug use.



"مارح ندقق عليها بشكل كبير بس اقروها بشكل عام"

Major Criteria	**1- Microbiological evidecne: Positive blood culture (BC)** - Typical organism from two separate blood cultures. - Two blood cultures positive for organisms typically found in patient with IE (e.g.: s.aureus,s.viridans, HACEK organisms) - Persistently positive blood cultures. - Single positive blood culture for for Coxiella Burnetii, or titer greater than 1:800. If the organism was typical or common within IE patients, u need 2 positive blood cultures to meet a major criteria. However, if the organism was atypical such as coxiella, 1 single blood culture is enough to meet a major criteria.
	2-Examination evidence / endocardial Involvement : New (not changed) murmur of Regurgitation
	3- Positive Echo: (Transesophageal echocardiography if prosthetic valve, complicated, or pretest probability possible IE)

Minor Criteria	**1-Predisposition:** Predisposing to heart condition or IV drug use.
	2- Fever greater than 38C.
	3- Vascular phenomena: **Includes: major arterial emboli** (emboli travelling within the arterial circulation) causing: Mycotic aneurysm, Intracranial or conjunctival hemorrhages, Janeway lesions. **Excludes :** Petechiae, and Splinter Hemorrhages.
	4-Immunologic phenomena: -Rheumatoid factor (RF) -Roth's spots (retinal hemorrhage with pale center) -Glomerulonephritis -Osler's nodes
	5-Microbiological evidence: - Positive blood culture but NOT meeting major criteria. مثلا مايطلع لك الا كلتشر وحده بوستف والمفروض ٢ أو ٣ - Serology test

Treatment of I.E

General Considerations:

- Antimicrobial therapy should be administered in a dose designed to give sustained **bactericidal** serum concentrations throughout much or all of the dosing interval.
- In vitro determination of the minimum inhibitory concentration of the etiologic cause of the endocarditis should be performed in all patients. (Enough dosage)
- The duration of therapy has to be sufficient to eradicate microorganisms growing within the valvular vegetations.
- The need for prolonged therapy in treating endocarditis has stimulated interest in using combination therapy to treat endocarditis

Valve	**Native**	**Prosthetic**
MSSA / MRSA	Cloxacillin (or vancomycin in case of MRSA)	Cloxacillin (or vancomycin in case of MRSA) In addition to Gentamicin & Rifampin
Streptococcus (strept.viridans)	Treatment depends on MIC (Minimum inhibitory concentration) Lower MIC means less antibiotic is needed. So: If MIC is low (<0.1), we need to use one antibiotic. (Penicillin alone or Cephalosporin alone) If MIC is intermediate (>0.1-0.5), we need to use 2 antibiotics (Penicillin in addition to Gentamicin) If MIC is high (>0.5), we need to use 2 antibiotics but for longer time (Ampicillin in addition to Gentamicin)	
HACEK	Cephalosporin (ceftriaxone)	
Bartonella	Doxycycline + gentamicin	-
Q-fever	Doxycycline +or- hydroxychloroquine	35% surgical

❖ If you want the original table from The slides with specific durations, click here.

Indications for Surgery:

the general idea is that it is needed in some cases involving significant valvular damage or if the valve is prosthetic or in case of medically failing thearapy)

1. Refractory CHF
2. Perivalvular invasive disease
3. Recurrent systemic emboli, particularly in the presence of large vegetations
4. Uncontrolled infection on maximal medical therapy
5. Some pathogens : Pseudomonas, brucella, coxiella, fungi, enterococci
   (these always need surgery especially fungi)
6. Usually advised with combined therapy

Treatment of I.E, contd..

In case of prosthetic valve endocarditis,

When do we need surgery? And when medical therapy alone is enough?

Surgical intervention is needed	- Perivalvular infection valve - Dehiscence, a surgical complication where the edges of a wound no longer meet - Excessively mobile prosthesis on echo results in hemodynamic instability - Prosthetic infection with S. aureus usually needs surgery. - Relapse and recurrence after surgery about 7% in 6 years. S. aureus risk rate of death is 0.18 in (surgery + antibiotics) vs (antibiotics alone). ??!!!!
Medical treatment may be sufficient (No surgery)	- 12 months or more post surgery - Viridans group streptococci (VGS) or HACEK or Enterococci. - No perivalvular extension (the valve extend to the adjacent periannular areas and erode into nearby cardiac chambers)

Prophylaxis

Recommended for	- For High or Moderate cardiac risk conditions (previous list of risk factors) - For dental procedures, rigid bronchoscopy, esophageal procedures, GI mucosal procedures, cystoscopy, prostate surgery . - Antibiotic Prophylaxis (American Heart Assoc). JAMA
Timing	One hour prior to procedure: - 2mg Amoxicillin orally - 600 mg Clindamycin orally - 2mg Cephalexin orally - 500mg Clarithromycin orally - 2mg Ampicillin intramuscularly
Dental procedures where endocarditis prophylaxis indicated	- Extraction - Periodontal procedures - Implants - Root canal - Subgingival antibiotics fiber/strips - Initial orthodontic bands (not brackets) - Intraligamentary local anesthetic - Cleaning of teeth/implants if bleeding anticipated
Dental procedures where endocarditis prophylaxis NOT indicated	- Filling cavity or local anesthetic - Placement of rubber dam - Suture removal - Orthodontic removal - Orthodontic adjustments - Dental X-rays - Shedding of primary teeth

SAQ

SAQ1: A 63-year-old gentleman with a history of mitral valve prolapse presents to his internist with a 2-week history of fever, night sweats, and general malaise. Three weeks ago, he underwent periodontal surgery for gingival hyperplasia, for which he did not receive antibiotic prophylaxis. He denies a history of drug abuse. His physical exam is notable for a temperature of 39 deg C and a faint pansystolic murmur loudest at the cardiac apex. Splinter hemorrhages are seen under his nails.

A) Your diagnosis? **B)** What is the most likely causative organism in this case? **C)** What are the specific signs that you will be looking for to confirm your diagnosis? **D)** Your treatment plan? **E)** What is the predisposing risk factor in this case? **F)** Briefly explain the pathogenesis of his condition. **G)** Beside the physical examination, what are the test that you should ask for to confirm your diagnosis?

SAQ2: A 37-year-old man with a history of IV drug use presents to the ED with complaints of fevers, chills, and malaise for two days. He admits to recently using IV and intramuscular heroin. Vital signs are as follows: T 40.0 C, HR 120 bpm, BP 110/68 mmHg, RR 14, O2Sat 98%. Examination reveals a new systolic murmur that is loudest at the lower left sternal border.

A) Your diagnosis? **B)** What is the most likely causative organism in this case? **C)** Mention two non-specific signs that might be seen in similar cases **D)** Your treatment plan? **E)** What is the risk factor in his condition? **F)** List some possible complications of this condition.

SAQ3: A 70-year-old woman, who underwent biological aortic valve replacement surgery 3 months before, was admitted to hospital with fever and cough for more than 1 week. Two sets of blood cultures were taken. Physical examination showed a temperature of 37.6° with otherwise unremarkable vital parameters, a 3/6 holosystolic cardiac murmur and splinter haemorrhages of her right thumb. Laboratory tests revealed an elevated C reactive protein (CRP) of 208 mg/l (standard value <5). Transesophageal echocardiography (TEE) showed multiple vegetations on the biological aortic valve up to 15 mm and a perivalvular abscess. Microbiology lab reported that Gram positive cocci in clusters was growing, and coagulase test came negative.

A) What is your diagnosis? **B)** What is the organism?

SAQ1: A) Subacute endocarditis B) Strept. viridans C) osler's nodes, janeway lesions, and roth spots.
D) depending on the MIC, Penicillin alone, Gentamicin will be added if the bacteria is less sensitive. E) Mitral valve prolapse
F) endothelial injury (his mitral valve prolapse) followed by bacteremia, then adherence and finally invasion.
G) Minimum of 3 blood cultures, Echocardiography, Non-specific lab tests such as ESR, urinalysis.

SAQ2: A) Acute endocarditis B) Staph aureus C) Splinter hemorrhages and petechiae .
D) depending on the microbiology lab results, if MSSA, cloxacillin. If MRSA, vancomycin. E) IV Drug use F) See page 10 :)

SAQ3: A) Early prosthetic valve endocarditis. B) Staph. epidermidis.

MCQs

Q1: The condition associated with the highest risk of developing infective endocarditis (IE) in young people is:			
A- IV drug use	B- Mitral valve thickened leaflets	C- Mitral Stenosis.	D- Mitral valve prolapse
Q2: A 64-year-old man presents to the emergency department with chest pain, fever, fatigue, and arthralgias. His past medical history is significant for rheumatic heart disease and a dental procedure a few weeks before admission. Blood culture came negative. Probable cause is:			
A- Fastidious bacteria	B- Previous antibiotics intake	C- Embolic events	D- A & B
Q3: a 65-year-old woman who has developed endocarditis with viridans streptococci (MIC ≤ 0.1 ug/mL) on a native heart valve. The patient has no known drug allergies and normal renal function. Which of the following intravenous regimens is most appropriate?			
A- Penicillin alone	B- Cephalosporin alone	C- A & B are correct	D- Penicillin in addition to gentamicin
Q4: A 35-year-old intravenous drug abuser has been diagnosed with native tricuspid valve endocarditis due to methicillin-sensitive Staphylococcus aureus. Other than intravenous drug abuse, his past medical history is noncontributory. He has no known drug allergies, normal renal function, and appears in no apparent distress. Which of the following intravenous regimens would be most appropriate?			
A- Cloxacillin	B- Vancomycin	C- Vancomycin in addition to Gentamicin & Rifampin	D- Penicillin
Q5: All are true about echocardiographic findings regarding infective endocarditis except:			
A- Vegetations are seen on the valve	B- Abscess might be revealed	C- TEE is better than TTE	D- TTE is better than TEE
Q6: Non specific sign of endocarditis:			
A- Osler's nodes	B- Petechiae	C- Roth spots	D- Janeway lesions
Q7: Infective endocarditis is becoming a disease of the elderly, this mainly due to:			
A- The decline of rheumatic heart disease.	B- The increasing proportion of younger generations.	C- The decreasing proportion of elderly.	D- The increasing incidence of IV drug use.

Q1	Q2	Q3	Q4	Q5	Q6	Q7
A	D	C	A	D	B	A

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