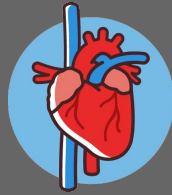




# pericarditis, myocarditis & infective endocarditis

Editing file



# Objectives

## Color index:

- Important, ☀
- Doctor Notes
- Extra, TN
- NOT important

## Endocarditis

- Definition, Pathogenesis, Risk factors, Clinical presentation, Diagnosis, Culture negative endocarditis, Management and Prophylaxis

## Myocarditis & Pericarditis

- Describe the epidemiology, risk factor for myocarditis, Explain the pathogenesis of myopericarditis, Differential between the various types of myocarditis and pericarditis, Name various etiological agents causing myocarditis and pericarditis, Describe the clinical presentation and differential diagnosis of myocarditis and pericarditis, Discuss the microbiological and non microbiological methods for diagnosis of myocarditis and pericarditis and Explain the management , complication and prognosis of patient with myocarditis and/or pericarditis.



## Dr. Khalifa overview

- **Myocarditis**: inflammation of the heart

### Infectious:

Viral : **Coxsackie B**. (most common cause) Bacterial: **corynebacterium, syphilis**

### Non infectious:

SLE, autoimmune, drugs, chemotherapy, radiation

**Giant cells** (very serious cause and can be fatal)

**Symptoms** (more non specific): **chest pain, arrhythmia, fever**

**Diagnosis**: **cardiac enzymes are high, ECG (nonspecific ST-T changes), heart biopsy, radiological studies**

**Treatment**: **Mainly supportive (mild and self limiting , mostly viral)**

Most patients are young (20's-30's)

- **Pericarditis**: inflammation of the pericardium

### Infectious:

Viral : **Coxsackie B & A**. Bacterial: **same as acquired pneumonia (acute) and TB (chronic)**

(not the most common)

### Non infectious:

SLE, autoimmune, drugs, chemotherapy, radiation, **myocardial infarction**

**Uremic carditis** (related to kidney problems)

**5 Main types**: **Caseous** (TB), fibrinous, hemorrhagic (TB), **serous** (viral), **purulent/suppurative** (bacterial because bacterial infections come with pus especially S.aureus)

**Symptoms** (more specific): **chest pain (positional, can hear a pericardial friction rub, related to breathing), ECG (specific changes: ST elevation, RP depression, T-wave inversion)**

**Diagnosis**: Cardiac enzymes are high if the pericarditis extends to the myocardium, Echo is important, **has to check the amount of fluid around the heart**, radiological studies, **blood culture**, TB lab for AFB culture, biopsy, sample taken either diagnostic or therapeutic

**Treatment**: **supportive but you have to look for the cause** (is it bacteria or virus?, signs of pneumonia or TB, is there any Renal issues, previous myocardial infarction? Then decide on the treatment)

In case of Tamponade, use **Pericardiocentesis**

- Both disease are potentially infectious and non infectious
- Both need general blood work and have high ESR

## Prof. Hanan

- It's important to know the cause of each one and how to manage. **clinical presentations are SO IMPORTANT**
- blood culture usually negative “ because commonly it's a virus or bacteria like: TB, chlamydia or mycoplasma pneumonia which need a special culture to grow. Rarely, blood culture will be positive if it started from the lung like: klebsiella and S.auros.”
- **Myocarditis**: can be sudden or not, symptoms similar to fever, can be infectious or noninfectious, if “troponin and CK-MB” are evaluated → we know that there is a problem with heart muscle (myocardium). chest X-ray will show cardiomegaly. risk of sudden death.
- **Pericarditis**: sudden, acute or chronic(TB). usually infectious and rarely noninfectious. chest pain relieved by sitting forward. pericardial rub and thickening more than 5mm. chest X-ray will show enlarged cardiac shadow. the best way to reduce the pressure is Pericardiocentesis.
- both diseases are self limiting when it caused by a virus. we use antiviral only in severe cases.



Dr's  
notes:

## Prof. Somily

- Numbers are **NOT** important
- Important to Know:
  - Risk Factors
  - Identifying causative agents.
  - methods of diagnosis
  - Treatment
- Infection of endocardial is very critical because it involve the valves.
- Acute endocarditis:
  - Patient will deteriorate within 2-3 days or 1 week, high fever, palpitation, myalgias, and leads to high mortality.
  - Staph aureus is the most common cause.
- Subacute endocarditis:
  - gradual onset, mild -low fever & gradual fever, myalgias, rash, & less mortality.
  - Strept. viridans is the most common cause.
- Pathophysiology:
  - Usually it happens before abnormal valves of the heart, due to distributes in blood flow & can lead to damage of endothelium → which leads to accumulation of blood & coagulation factors.
- Risk factors:
  - the most imp one is prosthetic valves.
  - then abnormal valves, and aortic valve is the most common valve associate with endocarditis.
- The most important in laboratory findings is blood culture.
- When you have repeated culture growing +ve organism with valvular vegetation → endocarditis.
- Culture negative:
  - some patients have vegetation but culture is **negative**, means that the organism difficult to grow (usually fastidious bacteria), or may be the organism cannot grow because the patient had antibiotics & kills the organisms.

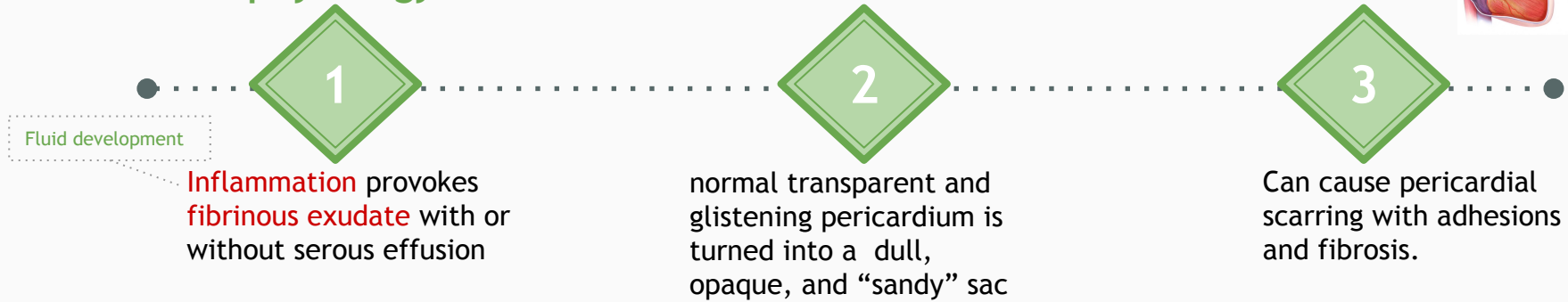
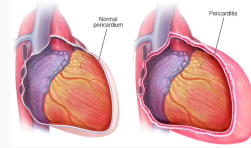
## Dr's notes:

## Dr. Fawzia

- heart infections are not common, because 1- heart is sterile 2- It's the site of the secondary immune system, its so strong and power.
- **Types:** Acute → S.aureus - normal valve  
Subacute → S.viridans - damaged valve
- **prosthetic valves** infections divided into 1-early (during a year after valve transplant surgery)-S.epidermidis \ 2- late (after one year of the surgery)
- Organisms (normal flora) enter blood circulation through some ways: tooth extraction - bleeding during teeth brushing - procedures involves gut - catheterization- colonoscopy .so, in history we need to consider any procedure that patient did.
- **Physical examination** is very important and critical: Fever, petechiae (check skin rash/ mucus membrane in mouth), nail bending (splinter hemorrhage)
- **Laboratory tests** in endocarditis: 3 sets of blood culture + echocardiography (ultrasounds of heart)
- **Treatment** of IE must follow 4 things: 1- IV administration. 2- bactericidal antibiotic. 3- combination (B-lactam+aminoglycoside). 4- 4 weeks of duration at least ( MIC should be done).

# Pericarditis

- It is an inflammation of the pericardium, Usually by infectious etiology.
- **Pathophysiology:**



## Spread of Infection:

Contiguous

-Spread from near organ; **Lungs**, pleura, mediastinal lymph nodes, myocardium, aorta, esophagus, liver.

Lymphangetic

Hematogenous

Through blood  
septicemia, toxin,  
neoplasm, metabolic

Traumatic or irradiation

# Etiology

## Infectious etiology

### → Viral Pericarditis:(Typical cause)

- ◆ Most common:
  - **Coxsackie virus A & B**, and Echovirus.
- ◆ other viruses:
  - Herpes, Hepatitis B, HIV, Mumps, Influenza, Adenovirus, Varicella viruses.

### → Disseminated Fungal Infection:

- ◆ Histoplasma, and Coccidioides.

### → Parasitic infections: (rare)

- ◆ e.g. disseminated toxoplasmosis,
- ◆ contagious spread of **Entamoeba histolytica**.

Cats at home are the source of it

### → Bacteria Pericarditis:

- ◆ Usually it's a **complication of pulmonary infections** (e.g. pneumonia, empyema, TB).
- ◆ So **organisms** are similar to those in lung infections:
  - **S.pneumonia, M.tuberculosis,**
  - **S.aureus, H.influenzae,**
  - **Klebsiella pneumonia, & Legionella.**
  - M.catarrhails, Chlamydia pnemonia, Mycoplasma pneumonia.
- ◆ HIV patients may develop pericardial effusions by;
  - M.tuberculosis, M.avium.

## Non-Infectious etiology

→ Immune-mediated: as in **rheumatic fever & SLE**.

→ **Miscellaneous:** e.g. due to **myocardial infarction**, **malignancy** & **uremia**.

NOT important

# Classifications

1. Can be divided into 5 types according to the composition of the fluid that accumulates around the heart:

## 1- Caseous Pericarditis:

→ commonly **tuberculosis** in origin.

## 2- Serous Pericarditis:

→ due to **autoimmune diseases** (e.g. Rheumatoid arthritis, SLE), & **viral infection**.  
◆ Transudative serous fluid (yellow clear fluid).

## 3- Fibrinous Pericarditis:

→ due to acute Myocardial infarction (MI), uremia, radiation.  
◆ Fibrinous exudative fluid.

## 4- Purulent/Suppurative Pericarditis:

→ due to **Acute bacterial infection**, fungi or parasites infections.  
◆ Purulent exudative fluid (formation of pus).

## 5- Hemorrhagic Pericarditis:

→ usually caused by infection (e.g. **TB**) or malignancy.  
◆ blood mixed with a fibrinous or suppurative effusion.

2. According to the clinical presentation and the duration:

1- Acute pericarditis.

2- Chronic pericarditis.

# Pericarditis

## Acute Pericarditis

- CA Pneumonia organisms usually.
- **Clinical presentation:** ✨
  - ◆ **Sudden pleuritic chest pain**<sup>1</sup>, which is **positional restroterna** (relieved by setting forward).
  - ◆ Dyspnea & fever.
  - ◆ On examination:
    - **Pericardial friction rub**<sup>2</sup>, exaggerated pulses, paradoxus JVP & tachycardia.
    - As the pericardial pressure increase → palpitations, presyncope or syncope may occur.
- **Differential diagnosis:**
  - ◆ Acute myocardial infarction.
  - ◆ Pulmonary embolism.
  - ◆ Pneumonia.
  - ◆ Aortic dissection.

## Chronic Pericarditis

- **Tuberculous Pericarditis:**
  - ◆ Has insidious onset.
  - ◆ Incidence of pericarditis in patients with pulmonary TB ranges from 1-8%. (secondary)
  - ◆ Clinical presentation:
    - Fever, Pericardial friction rub<sup>2</sup>, Hepatomegaly, If it's secondary to PTB, could mention its symptoms (e.g. night sweats, weight loss).
  - ◆ Tuberculin skin test usually positive.
  - ◆ Fluid smear for acid fast bacilli (AFB) often negative.
  - ◆ Pericardial **biopsy more definitive**. biopsy is better than fluid in cases of extrapulmonary TB because it specify the type of pericarditis ( caseous necrosis → TB ).
- **Constrictive Pericarditis:**
  - ◆ In extreme cases, the heart is so completely encased by dense fibrosis that it cannot expand normally during diastole resulting in this condition.
  - ◆ Causes:
    - Idiopathic, Radiotherapy, Cardiac surgery, Connective tissue disorders, Dialysis, Bacterial (TB), viral & fungal infections.

<sup>1</sup>it's affected by position and breathing might make it worse

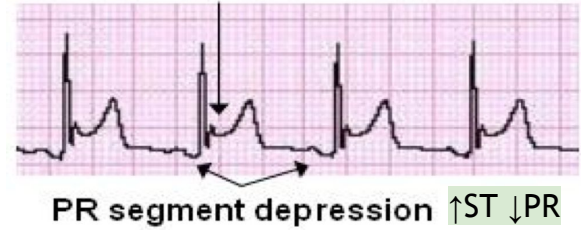
<sup>2</sup>diagnostic for pericarditis - [Listen](#).



# Diagnosis of pericarditis

- ECG (specific ST elevation and PR depression and T-wave inversion may occur later)
- Blood culture → most of the time negative
- Leukocytosis and an elevated ESR are typical
- Other routine testing: urea and creatinine.
- Tuberculin skin test is usually positive in tuberculous pericarditis cases. (Not ideal for diagnosis)
- Chest x-ray may show enlarged cardiac shadow or calcified pericardium and CT scan show pericardial thickening >5mm.
- Pericardial fluid or pericardial biopsy specimens for fungi.
- Immunology/Serology: Antinuclear antibody tests and Histoplasmosis complement fixation indicated in endemic area.

Concave-up ST elevation



Chest X-ray show enlarged cardiac shadow

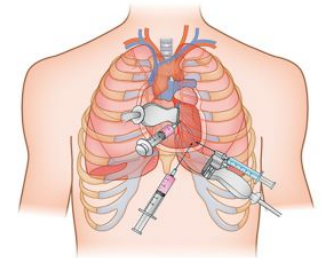
# Management of pericarditis

Management of pericarditis depend on its cause. Usually supportive  
 - uremic pericarditis → dialysis/ TB → antituberculous  
 .. look for the cause and treat it

Depend on the cause:

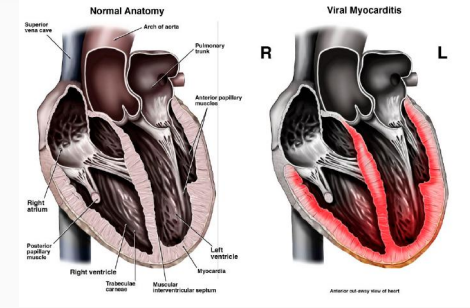
		Management				
Cause	→ idiopathic → viral	→ controversial → anticoagulants	→ S. aureus → respiratory bacteria	→ viral	→ <b>Cardiac Tamponade</b> (severe pericarditis, blood or fluid in the pericardial sac which defect the pumping)	
	<b>Supportive</b> • bed rest • NSAIDS • Colchicine	Corticosteroid usually contraindicated.	Specific antibiotics	Antiviral • Acyclovir (Herpes simplex or Varicella) • Ganciclovir (CMV)	<b>Pericardiocentesis</b>	
Management						

- **Pericardiocentesis:** a therapeutic procedure to remove fluid from the pericardium (**to relieve Tamponade**) in severe cases with pericardial effusion. **To reduce the pressure**
- Patients who recovered should be observed for recurrence.
- Symptoms due to viral pericarditis usually subsided within one month.



# Myocarditis

→ inflammatory disease of the heart muscle



- ★ Mild & self-limited with few symptoms OR severe with progression to congestive heart failure & dilated cardiac muscle.
- ★ localized or diffuse
- ★ **Viral infection is the most common cause → (Coxsackie virus B)**
- ★ no accurate estimate of incidence as many cases are mild & brief and diagnosis is not made.

# Etiology

## Infectious myocarditis

## Non-infectious myocarditis

### Viruses:

Most common : \* **Coxsackie virus B** (typical cause)  
**HIV**, Coxsackie virus A, Echoviruses,  
Adenoviruses , **Influenza**, **EBV**, Rubella,  
Varicella, Mumps, Rabies, Hepatitis viruses

### Systemic diseases:

- SLE ( systemic lupus erythromatosis )(cause Giant cell endocarditis *rare but important for mortality rate*).
- sarcoidosis
- vasculitis ( Wegener's disease )
- celiac disease

### Bacterial:

**Corynebacterium diphtheriae**, **Syphilis** ,Lyme disease, as a complication of bacterial endocarditis, Rickettsiae, Chlamydia, Legionella and Mycobacterium tuberculosis.

### Neoplastic infiltration:

- Thymoma (cause Giant cell myocarditis).

### Protozoan / parasitic:

Trypanosomacruzi (Chagas disease), Trichinella spiralis, Taxoplasma gondii and Echinococcus.

### Drugs & toxins:

Ethanol, cocaine, radiation, chemotherapeutic agents - Doxorubicin

**Spirochete:** Borrelia burgdorferi (Lyme disease)

**Others:** Fungi, enteric pathogen

**Others:** Thyrotoxicosis (cause Giant cell myocarditis).

Most common

NOT important Rare

# ★ Clinical presentation of myocarditis

Highly variable· May occur:

**Not sudden** → days to weeks after onset of acute febrile illness ( flu-like symptoms )

OR

**Sudden** → with heart failure without any known antecedent symptoms

## General symptoms :

**Fever**, headache, muscle aches, diarrhea, **sore throat** and rashes similar to most viral infections

## ★ Specific symptoms :

**Chest pain, arrhythmias** ,sweating , fatigue and may present with **congestive heart failure**. (very severe infection to the point the heart doesn't function properly which will lead to fluid in the lungs)

## Differential diagnosis

Acute Myocarditis

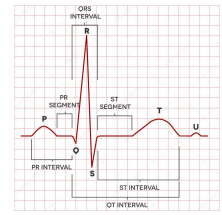
Vasculitis

Cardiomyopathy

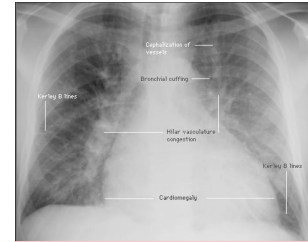
due to drugs or radiation

# Diagnosis of myocarditis

- CBC, WBCs, ESR, **Troponin and CK-MB\*** usually elevated
- ECG (**nonspecific** ST-T changes and conduction delays are common)
- Blood culture → **usually negative**
- **Viral serology** and other specific tests for Lyme disease, diphtheria and Chagas disease may be indicated on a case by case basis.
- Chest X-rays: show **cardiomegaly\*** (not seen in self limited)
- Radiology: MRI and Echocardiogram **very helpful in myocarditis**
- Endomyocardial diagnosis (heart muscle biopsy): Pathologic examination is not sensitive. It may reveal lymphocytic inflammatory response with necrosis. “Giant cells” may be seen.  
**is not that helpful in case of myocarditis**

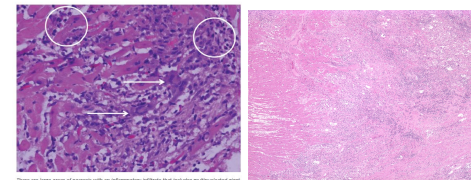


Normal ECG



Chest X-ray show cardiomegaly

Figure 2: Endomyocardial Biopsy showing Giant Cell Myocarditis



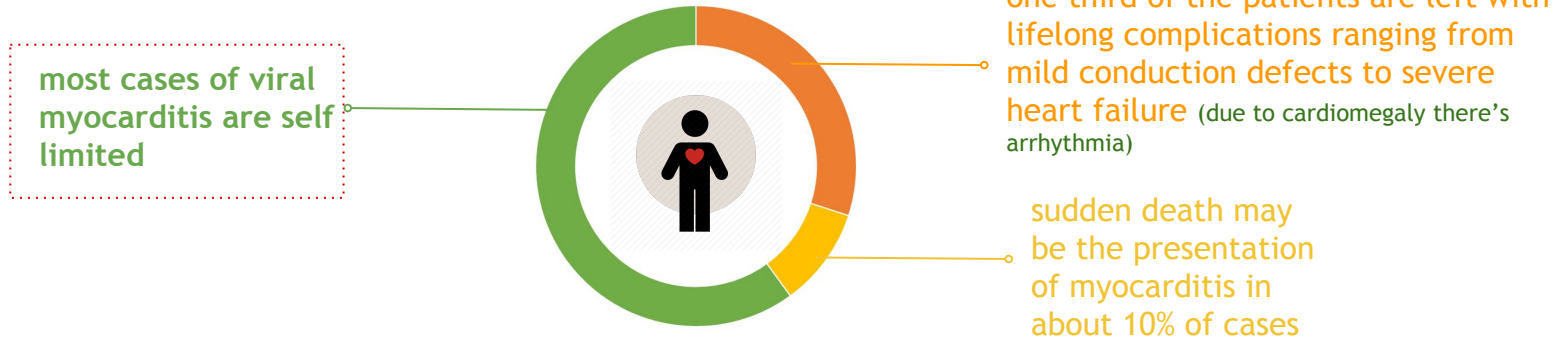
There are large areas of necrosis with an inflammatory infiltrate that includes multinucleated giant cells (circles) as well as mononuclear and macrophage (arrows) (hematoxylin and eosin, 20x original magnification).

Endomyocardial biopsy show giant cell

\*Troponin and CK-MB are helpful tests to detect any problem with heart muscle  
\*Cardiomegaly : enlarged heart

# Management of myocarditis

- ▶ Often **supportive** : restricted physical activity in heart failure “advice the patient to take rest”
- ▶ Giant cell myocarditis potentially does not have a treatment, just supportive care.
- ▶ Specific antimicrobial therapy (sometimes) is indicated when an infecting agent is identified
- ▶ Treatment of heart failure arrhythmia
- ▶ Other drugs indicated in special situations like anticoagulant, NSAID (non-steroidal anti inflammatory drugs)(depending on the cause) , steroid or immunosuppressive immunomodulatory agents.
- ▶ Heart transplant
- ▶ Patient should be followed regularly every 1-3 months.



# Infective Endocarditis

→ an infection of the heart's endocardial surface (heart valves)

## Epidemiology:

Incidence: 1.7– 6.2 / 100, 000 person years

→ More in male cardiac diseases are common in male in general.

Becoming a disease of the Median age and elderly

→ Pre ABx era –35y and Now –58y

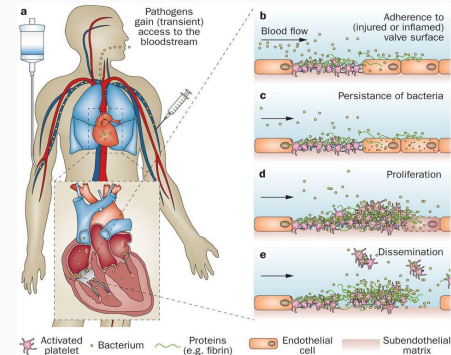
Due to two factors

1-The decline of rheumatic heart disease    2-The increasing proportion of elderly

## Pathophysiology:

→ Usually it happens before abnormal valves of the heart, due to distributes in blood flow & can lead to damage of endothelium → which leads to accumulation of blood & coagulation factors.

1. Turbulent blood flow disrupts the endocardium making it “sticky”
2. Bacteremia delivers the organisms to the endocardial surface
3. Adherence of the organisms to the endocardial surface
4. Eventual invasion of the valvular leaflets





# Classifications

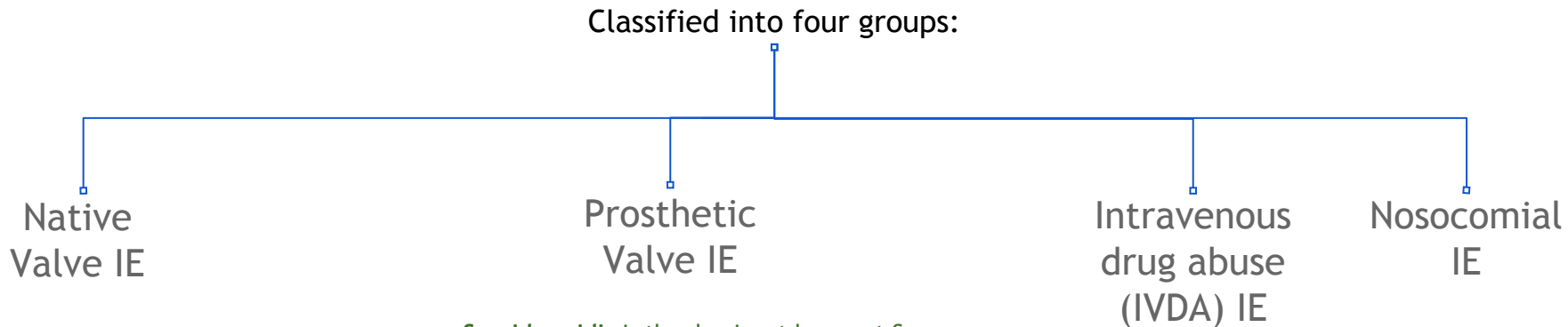
→ 1- It can divide to **Acute & Subacute**, according to clinical presentations, duration, & Causative organisms:

Acute	Subacute
★ Affects <b>normal</b> heart valves	★ Often affects <b>damaged</b> heart valves
★ Rapidly destructive	★ Indolent nature
★ If not treated, usually fatal within 6 weeks	★ If not treated, usually fatal by one year
★ <b>Commonly Staph ( S.aureus )</b> Metastatic foci	★ Commonly ( <b>Strepto viridans</b> )
Symptoms :	
★ High grade fever and chills	★ Low grade fever
★ Shortness of breath	★ Anorexia and weight loss
★ Arthralgias/ myalgias / Abdominal pain	
★ Back pain and Pleuritic chest pain	★ Nausea / Vomiting and Fatigue

*S.viridans* is an alpha-hemolytic streptococci, normally found in oral cavity, no capsule or toxins only production of polysaccharide which give it the stickiness nature, cannot affect normal valves.

- The onset of symptoms is usually ~2 weeks or less from the initiating bacteremia **for acute, and gradually for subacute.**

→ 2- It can divide to 4 groups, according to the valve involved :



→ It's normal valve, so it's strong valve & will damage by strong organism:

- Mainly Subacute: S. viridans
- Acute: Staph. aureus
- Enterococci (5-10%), GNB=HACEK (5%), Fungi are rare.

**S.epidermidis** is the dominant here not S.aureus

→ It's replacement valve :

- Early :(after surgery by 0-2 mo): Staphylococcus epidermidis → Gram +ve, in clusters, catalase +ve, coagulase -ve **It's skin flora.**
- Late (after 1 year) they might have **Staph aureus**, GNB, & enterococci.

→ Staph. aureus mostly

\*GNB = Gram Negative Bacteria

\*HACEK = Fastidious group of g -ve bacteria that are an unusual cause IE ( Haemophilus, Aggregatibacter , Cardiobacterium, Eikenella, Kingella )

# Risk Factors

## ★ Prosthetic Valve:

The most important risk factor, because it's a good surface for organisms.

infective endocarditis	7 -25 %	Early <12 mons after surgery	Late >12 mons after surgery
Effect	0.94 per 100,000 bioprosthetic		
greater risk of mechanical valves	for first 3 mons, then risk same at 5y	1-3.1% risk at 1 yr	2-5.7% at 5 yr

### ★ Cardiac Abnormality

High risk	Moderate risk	Low/no risk
<ul style="list-style-type: none"> <li>• <b>Prosthetic valve</b> • Coarctation</li> <li>• <b>Previous IE</b> 4.5(2.5 to 9)%</li> <li>• Aortic valve disease 12 to 30%</li> <li>• Rheumatic valve disease</li> <li>• Complex cyanotic congenital</li> </ul>	<ul style="list-style-type: none"> <li>• MVP w/ MR/thickened leaflets- 5 to 8 times (100/100 000 person years)</li> <li>• Mitral Stenosis • tricuspid valve</li> <li>• Pulmonary Stenosis • Hypertrophic Obstructive Cardiomyopathy (HOCM)</li> </ul>	<ul style="list-style-type: none"> <li>• ASD "Atrial Septal Defect" (secundum)</li> <li>• CABG "Coronary Artery Bypass Grafting"</li> </ul>

## Risk Factors continued ..

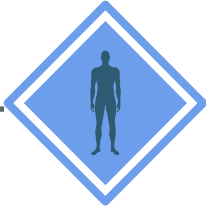
★ Injection drug use - IVDU		★ Structural cardiac abnormality
<ul style="list-style-type: none"> <li>• 100X increase risk in young</li> <li>-<b>Staphylococcus aureus</b></li> </ul>	<ul style="list-style-type: none"> <li>• Rates 150- 2000/100 000 person years</li> <li>• Higher among patients with known <b>valvular heart disease</b></li> </ul>	<ul style="list-style-type: none"> <li>• 75% of pts will have a preexisting structural cardiac abnormality</li> <li>• 10-20% have congenital heart disease</li> </ul>
★ HIV infection:	★ Rheumatic valve disease:	
<ul style="list-style-type: none"> <li>• A number of cases of IE have been reported in patients with HIV infection</li> <li>• It has been suggested that HIV infection is an independent risk factor for IE in IDU</li> </ul>	<ul style="list-style-type: none"> <li>• Predisposition for young in some countries 37%-76% of cases</li> <li>• Mitral 85%, Aortic 50%</li> <li>• Degenerative valvular lesions</li> <li>• MV Prolapse and associated mitral regurgitation - 5 to 8 times higher IE risk</li> <li>• Aortic valve disease (stenosis or/and regurgitation) is present in 12 to 30 % of cases</li> </ul>	

## Diagnostic approach

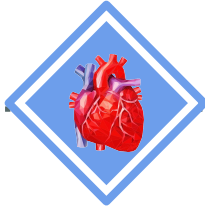
- History of prior cardiac lesions

- A recent source of bacteremia  
Like: tooth extraction - colonoscopy ...

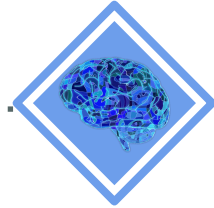
# Physical examination



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



Neurologic evaluation may detect evidence of focal neurologic impairment

## Signs: Fever & Heart murmur

Nonspecific signs	More specific signs
★ petechiae, subungual/“splinter” hemorrhages, clubbing, splenomegaly, neurologic changes	★ Osler’s Nodes, Janeway lesions, and Roth Spots More explanation in next slides

## Other aspects clinical diagnosis

- Important to know which valve involved R or L.
- And the function of heart.
- Pump, acute valve dysfunction conduction
- Look for evidence emboli
- Bleed (intracranial, elsewhere mycotic aneurysm )

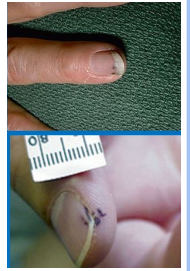
- 1- **Nonspecific**
- 2- Often located on extremities or mucous membrane



### Petechiae

### Splinter Hemorrhages

- 1- **Nonspecific**
- 2- Non Blanching
- 3- Linear reddish-brown lesions found under the nail bed
- 4- Usually do NOT extend the entire length of the nail



- 1- **More specific**
- 2- **Painful** and erythematous nodules
- 3- Located on pulp of fingers and toes
- 4- More common in subacute IE



### Osler's Nodes

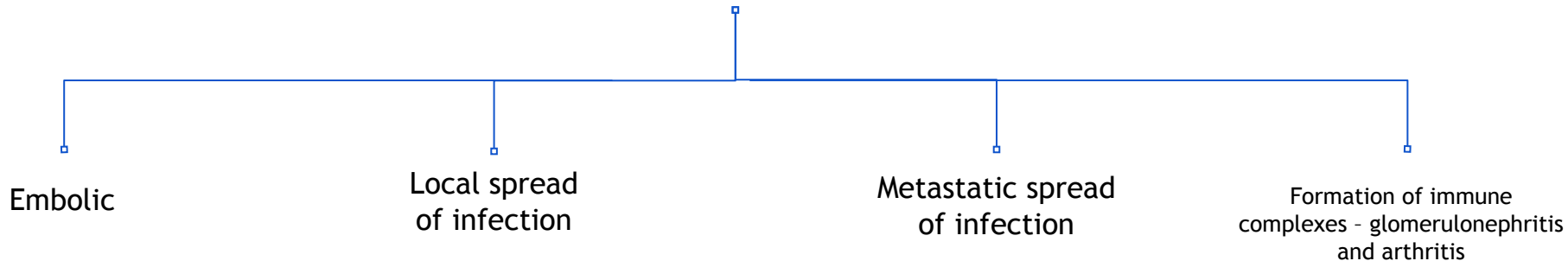
### Janeway Lesions

- 1- **More specific**
- 2- Erythematous, blanching macules
- 3- **Nonpainful**
- 4- Located on palms and soles



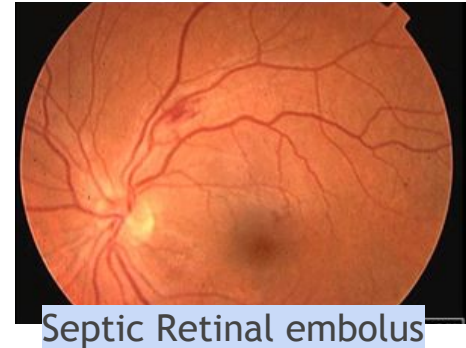
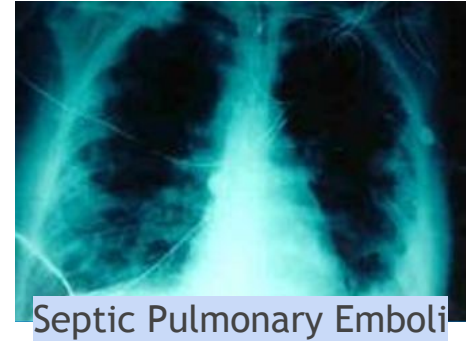
## Complication

Four etiologies:



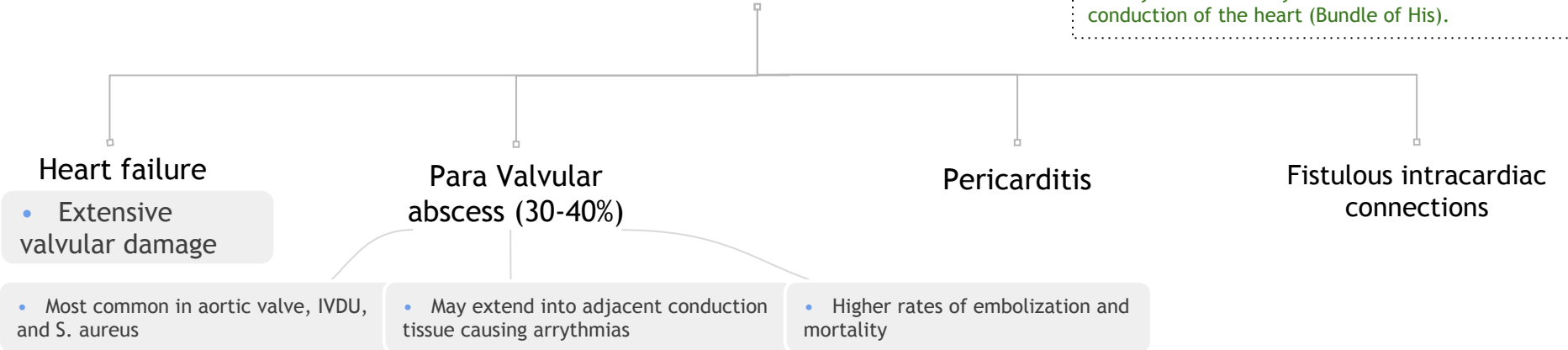
# Emboic Complications

- They have Emboli due to the damage heart and vegetation, The clots can be sent to any place and can cause an infection, surgery might be needed. it's the most dangerous complication.
- Occur in up to 40% of patients with IE
- Predictors of embolization
  1. Size of vegetation
  2. Left-sided vegetations
  3. Fungal pathogens, *S. aureus*, and *Strep. Bovis*
- Incidence decreases significantly after initiation of effective antibiotics
- Stroke
- Myocardial Infarction
- ★ Fragments of valvular vegetation or vegetation-induced stenosis of coronary ostia
- Ischemic limbs
- Hypoxia from pulmonary emboli
- Abdominal pain (splenic or renal infarction)

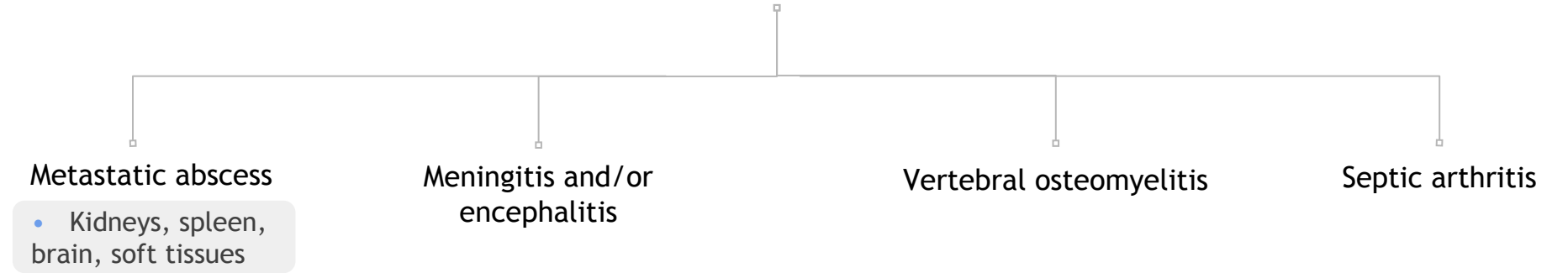


# Local Spread of Infection

It's in the endocardium which can damage the myocardium and pericardium which leads to effusion and sometimes they can lead to rupture of the myocardium. They can affect the electrical conduction of the heart (Bundle of His).



# Metastatic Spread of Infection





# Laboratory tests

## Microbiology

### 1- Positive blood culture results

- A minimum of three blood cultures should be obtained over a time period based upon the severity of the illness

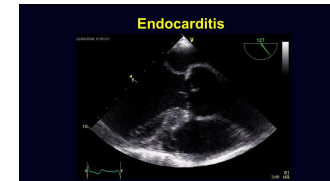
### 2- Additional laboratory Nonspecific test

- 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

## Radiology

### - Echocardiography ( ultrasounds of heart )

-What we are looking for in echo ? **Thrombi**  
We use transesophageal echo because it's more accurate



### Echocardiographic findings :

NOT important

#### 1- 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

#### 2- abscess

- New partial dehiscence of prosthetic valve
- New valvular regurgitation (increase or change in pre-existing murmur not sufficient)

## Additional laboratory tests

Dr.note:  
"ECG is Not imp or helpful  
in diagnosis of IE"



### 1- abnormal urinalysis

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

### 2- ECG

- New AV, fascicular, or bundle branch block.  
Perivalvular Invasion monitoring + pacing



- ★ Extra: The changes on II lead ECG and demonstrates first degree atrioventricular block (a delay or interruption in the transmission of an impulse from the atria to the ventricles owing to an anatomical or functional impairment in the conduction system that results in a PR interval longer or greater at slow heart rates).

NOT important

## Improved diagnostic value of echocardiography in patients with infective endocarditis by transoesophageal 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

# Case Definition: IE

- Duke criteria
- In 1994 investigators from Duke University modified the previous criteria to include the role of echocardiography in diagnosis
- They also expanded the category of predisposing heart conditions to include intravenous drug use

## Modified Duke criteria

- Proposed: 2000, Addresses TEE, Broad “possible categories.
- *S. aureus* risks (13-25% *S. aureus* bacteremia have IE )

Definite IE	★ Microorganism (via <u>repeated +ve culture</u> or histology) in a valvular vegetation, embolized vegetation, or intracardiac abscess
	★ Histologic evidence of <u>vegetation</u> or intracardiac abscess
Possible IE Next slide	★ 1 major and 3 minor
	★ 2 major
	★ 5 minor
Rejected IE	★ Resolution of illness with four days or less of antibiotics

# Modified Duke criteria

Major criteria	Minor criteria: Predisposition (heart condition or IV drug use)
<p>1- MICROBIOLOGY</p> <ul style="list-style-type: none"> <li>• Typical organism from 2 separate cultures OR</li> <li>• <u>Microorganism from persistently positive BC</u> OR</li> <li>• Single BC + for Coxiella burnetii, or titer &gt;1:800</li> </ul>	<p>1- Fever <math>\geq 38</math> C</p> <p>2- Vascular phenomenon (excludes petechiae, splinter hemorrhage)</p> <p>3- major arterial emboli</p> <ul style="list-style-type: none"> <li>• Mycotic aneurysm, intracranial or conjunctival hemorrhages. Janeway lesions</li> </ul>
<p>2- ENDOCARDIAL INVOLVEMENT</p> <ul style="list-style-type: none"> <li>• New (not changed) murmur of regurgitation</li> </ul>	<p>4- Immunologic phenomena</p> <ul style="list-style-type: none"> <li>• RF, Roth's spots glomerulonephritis, Osler's nodes</li> </ul>
<p>3- POSITIVE ECHO</p> <ul style="list-style-type: none"> <li>• (TEE if prosthetic valve, complicated, or pretest probability possible IE)</li> </ul>	<p>5- Microbiologic evidence</p> <ul style="list-style-type: none"> <li>• Not meeting major criteria single BC not CNS, serology</li> </ul>

# Poor Prognostic Factors

-Female	-S.aureus	-Prosthetic valve	-Diabetes mellitus	-Apache II score	-Para Valvular abscess
-Vegetation size	-Aortic valve	-Older age	-Low serum albumin	-Embolic events	-Heart failure

★ **Culture negative” IE** If the patient show negative blood culture !

## Why blood culture show negative results ?

★ Previous antibiotic intake  
- 50% of cases

★ **Fastidious bacteria\***

★ Long incubation subculturing ( eg. HACEK ~ 2-3 weeks )

- Tend to see subacute w/ valve destruction/CHF

-Hemophilus  
-paraphrophilus,  
-aphrophilus.  
-Parainfluenzae  
-Aggregatibacter (Actinob acillus)

-actinomycetemcomitans  
-Cardiobacterium hominis  
-Eikenella corrodens  
- Kingella spp.

NOT important

A **fastidious** organism is any organism that has a complex nutritional requirement. In other words, a fastidious organism will not grow in BC

# Lab Diagnosis! Etiologies “Culture Negative” IE Based on clinical setting

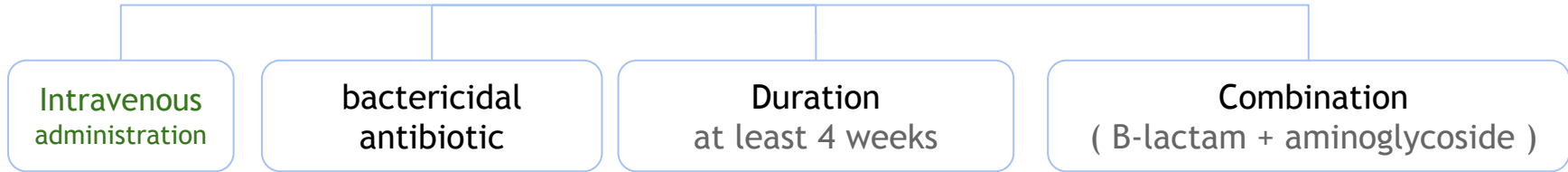
How to detect the etiology if the culture is negative ?

Laboratory test	Etiology
<ul style="list-style-type: none"> <li>● PCR of vegetation/emboli</li> </ul>	Tropherymawhippelei, bartonella
<ul style="list-style-type: none"> <li>● Histology/stain /culture of vegetation/emboli</li> </ul>	Fungus
<ul style="list-style-type: none"> <li>● Prolonged, enriched cultures</li> </ul>	“HACEK”
<ul style="list-style-type: none"> <li>● Lysis centrifugation system (Isolator)</li> </ul>	Bartonella, legionella (BCYE), fungal
<ul style="list-style-type: none"> <li>● Serology</li> </ul>	Endemic fungi, bartonella, Q fever, brucella, legionella, chlamydia
<ul style="list-style-type: none"> <li>● Thioglycolate or cysteine supplemented media</li> </ul>	S.aureus satellitism: Abiotrophia (NVS)

# Treatment of infective endocarditis

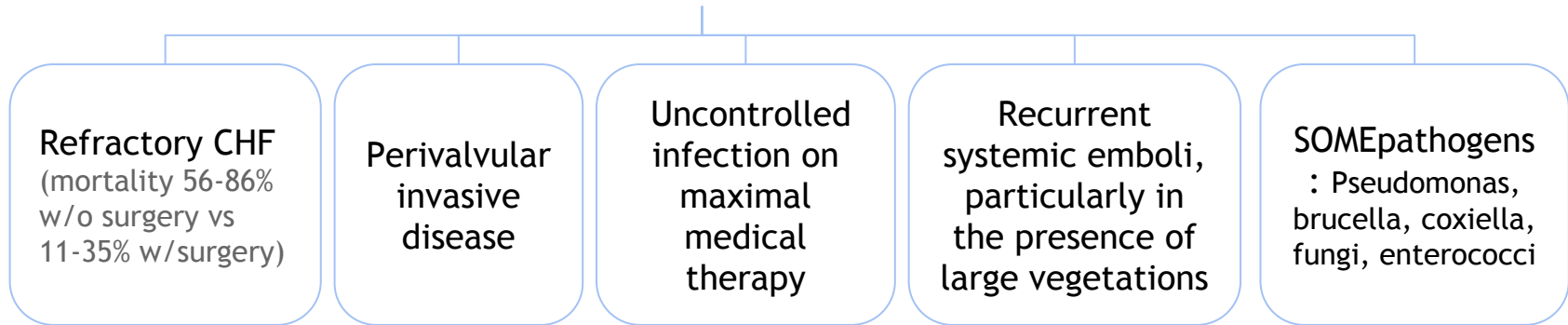
Example of the combination therapy : (gentamicin + ceftriaxone) the first work in the cell wall and the second work in the ribosomes of the bacteria, So they enhance each other.

## General consideration :



+ do minimum inhibitory concentration for patient to know the suitable dose to eradicate the organism

## Indications for surgery in IE NOT important



- usually advised with combined therapy

# Treatment of prosthetic valve endocarditis

## Medical treatment alone

1. >12 months post surgical

2. VGS or HACEK or enterococci

3. No perivalvular extension

## Medical treatment + surgery

Same as normal valve surgery indications in the past slide

+

Dehiscence

S.aureus

Perivalvular infection valve

Excessively mobile prosthesis on echo results in hemodynamic instability

- Recurrence after surgery about 7% / 6 years
- Relapse : S. aureus-RR death 0.18 in surgery plus AB vs ABx alone



# Antibiotics

→ Duration of treatment usually 4 weeks, but sometimes we can treat shorter with sensitive organism

Streptococci <sup>1</sup>	<u>Penicillin G + Gentamicin</u> (Gentamicin used in case of prosthetic valve)
Enterococci <sup>2</sup>	Ampicillin
Staph. aureus <sup>3</sup>	<u>Cloxacillin</u> (MSSA), sometimes 1st generation of Cephalosporins
MRSA <sup>4</sup>	<u>Vancomycin</u>
HACEK	<u>3rd generation of Cephalosporins</u> (e.g. cefotaxime, cefixime)

<sup>1</sup> S.viridans : Gram +ve in chains (catalase -ve), alpha hemolytic.

<sup>2</sup> Enterococci : Gram +ve in chains (catalase -ve), gamma (non-hemolytic).

<sup>3</sup> S.aureus : Gram +ve in clusters (catalase +ve), coagulase +ve.

<sup>4</sup> MRSA : Methicillin Resistant Staphylococcus Aureus.

# Prophylaxis of IE

- ★ For High or Moderate cardiac risk conditions (previous list)
- ★ For Dental, rigid bronchoscopy, esophageal procedures, G I mucosal procedures, cystoscopy, prostate surgery
- ★ Antibiotic Prophylaxis (American Heart Assoc. JAMA)

NOT important

**Timing** : One hour prior to procedure:

-2gm Amoxicillin orally

or

- 600 mg Clindamycin orally

or

-2gm Cephalexin orally

or

- 500mg Clarithromycin orally

or

-2 gm Ampicillin intramuscularly

Dental procedures where endocarditis prophylaxis indicated:	Dental procedures where endocarditis prophylaxis NOT indicated: NOT important
<ol style="list-style-type: none"><li>1. Extraction</li><li>2. Periodontal procedures</li><li>3. Implants</li><li>4. Root canal</li><li>5. Subgingival antibiotics fiber/strips</li><li>6. Initial orthodontic bands (not brackets)</li><li>7. Intraligamentary local anesthetic</li><li>8. Cleaning of teeth/implants if bleeding anticipated</li></ol>	<ol style="list-style-type: none"><li>1. Filling cavity or local anesthetic</li><li>2. Placement of rubber dam</li><li>3. Suture removal</li><li>4. Orthodontic removal</li><li>5. Orthodontic adjustments</li><li>6. Dental X-rays</li><li>7. Shedding of primary teeth</li></ol>

# Quiz

1-Which of the following patients are MOST at risk for developing endocarditis? “Select-all-that-apply”

- A. A 25 year old male who reports using intravenous drugs on a daily basis.
- B. A 55 year old male who is post-opt from aortic valve replacement.
- C. A 63 year old female who is newly diagnosed with hyperparathyroidism and is taking Aspirin.
- D. A 66 year old female who recently had an invasive dental procedure performed 1 month ago and is having a fever.

2- A patient is receiving treatment for infective endocarditis. The patient has a history of intravenous drug use and underwent mitral valve replacement a year ago. The patient is scheduled for a transesophageal echocardiogram tomorrow. On assessment, you find tender, red lesions on the patient's hands and feet & it's painful. You know that this is a common finding in patients with infective endocarditis and is known as?

- A. Janeway Lesions
- B. Roth Spots
- C. Osler's Nodes
- D. Trousseau Sign

3-A patient with severe pericarditis has developed a large pericardial effusion. The patient is symptomatic. The physician orders what type of procedure to help treat this condition?

- A. Pericardiectomy
- B. Heart catheterization
- C. Thoracotomy
- D. Pericardiocentesis

4-A 17-year old "street kid" is brought in from a free clinic because he had been sick with a fever and malaise for several days and when he presented to the free clinic, he was febrile and complaining of exertional fatigue. On exam had a heart murmur which he doesn't think he had when he ran away from his upper middle class home. He uses intravenous drugs. Imaging revealed a tricuspid vegetation. What is the most likely causative agent?

- A. Candida albicans
- B. Enterococcus faecalis
- C. Staphylococcus aureus
- D. Streptococcus pyogenes

## SAQ

1- 35 years old man who was drunk at night and had a fight with another man in the bar the other man bit him. A few days later he came to the ER suffering from fever, chest pain and joint pain culture was negative for the first few days. Days later the culture showed Eikenella corrodens.

2- 30 year old presented in the ER with few days of fever, sore throat and the ECG showed ST-T changes and potential arrhythmia

Answers

1- "Septic Arthritis" and "Endocarditis"

2- Myocarditis

**Key answers:**

1-A,B and D 2-C 3-D 4-C

# Thank you



**Give us your FEEDBACK !!**

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Special thanks to our amazing designer and member

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