# Infective endocarditis

# Notes:

•damage in endocardium after a microbial colonization.

•its rare, highly fatal and has long lasting effects even among patients who survive it.

•it becomes systemic

It has 3 factors that occur simultaneously:

1-microbes in systemic circulation

2-structural defect in heart/ valves

3-vegetations(bacteria+leukocytes+platelets+fibrin), they can cause embolus and infarction.

# Notes:

•it affects prosthetic heart valves & native valves(when superimposed with inflammation)

•1-10 cases per 100000 person-year.



•mean age of patients was <30 now its >50.

### •mortality rates are 25%



gram +ve 90%

1-streptococci A (mostly) developing

2-staphylococcus aureus (developed)

3-enterococcus (10%)

•staphylococci is associated with healthcare contact and invasive procedures more than streptococci.+ (it causes 30% of developed country El cases)

# **Predisposing factors of endocarditis**

- 1- Rheumatic disease (caused by group A streptococci)
- 2- Congenital heart disorders , prosthetic heart valves
- 3- Periodontal (surrounding teeth) procedures/diseases , damaged gingival tissues due to plaque accumulation
- 4- Dental extractions or implants
- 5- Hemodialysis, tonsillectomy, esophageal dilation
- 6- Skin infections, iv drug abusers
- 7- Cystoscopy, colonoscopy, urethral dilation

•Basically anything that can allow commensal bacteria to enter circulation. That's why antibiotic prophylaxis are always recommended during these procedures.

•Note: antibiotic prophylaxis aren't proven to protect people who are at risk of IE, & they also can cause antibiotic resistance.

•IV drug abusers have 2 distinctive characteristics :

1-organism coagulase -ve staph (staphepidermis)2-most common valve involved is tricuspid valve

## Valve Notes:

•the most common affected value is the mitral value (expect for iv drug abusers its tricuspid value)

•most common cause that affects native valves is group A beta hemolytic streptococci and streptococci viridians.

• prosthetic valves are mostly affected by staphylcocci.

# **General notes:**

- 1- Streptococcal IE caused by oral viridians remain most common in low income countries
- 2- The.HACEK.bacteria(haemophilius,aggregatibacter,Cardiobacte rium,Eikenella corrrodens ,Kingella) cause 3% of cases (G-ve)
- 3- Fungal endocarditis (usually :Candida, Aspergillus & histoplasma) is rare but fatal, it affects prosthetic heart valves in immunosuppressed patients or after cardiac surgery.
- 4- Always remember that in fungal IE the patient is usually in ICU with a central catheter & they are given IV nutrition with a broad spectrum antibiotic
- 5- Most endocarditis cases occur within 2 months-1year following a vascular catheter / surgical wounds/ skin injury/invasive dental

bacteria	hemolysis	Disease presentations
Staphylococcus aureus	Beta hemolysis	Acute IE
Staphylococcus epidermis (viridians group)	No hemolysis	-Endocarditis in iv drug abusers -Dental carries
Streptococci bovis (resistant to vancomycin)	No hemolysis	Colon cancer & endocarditis



# 1-Streptococci :

Remember : they're g+ve ,catalase -ve

## 1-viridians streptococci group:

Non hemolytic

•They are normal oral-intestinal flora

•They cause dental caries, oral abscesses gingivitis

•when they get access to circulation they deposit: dextran , adhesins & fibronectin binding proteins (these help in their adhesion to valves so they could start forming vegetations and causing bacteremia).

•they include: St.mutants & St.mitis

•they are less susceptible to penicillins.

# 2-group A streptococci:

Beta hemolytic

•they include S.pyogenes (strep throat)

•they can cause sore throat infection(pharyngitis mainly in children) and less commonly skin infection (impetigo).

# •a few weeks later some patients may develop post-streptococcal disease syndromes and they are:

1-rheumatic heart disease (cuz of their ability of molecular mimicry)2-post streptococcal glomerulonephritis

## 2-S.aureus:

Remember they are +ve gram +coagulase

• a common cause of acute endocarditis

•in developed countries may result in severe sepsis syndrome with a fatal outcome (progression happens from 2weeks to months)

### **3-enterococcus:**

Remember; they are gram +ve , -ve coagulase.

•Some strains are resistant to penicillin & vancomycin, so its treated with teicoplanin



# Staphyloccoci



# 4-gram-ve bacteria:

•brucella,salmonella,haemophilus,cardiobacterium,eikenella & gram+ve actinobacillus.(part of normal flora)

•they don't respond to vancomycin and gentamycin, so we prescribe Ceftriaxone or 3<sup>rd</sup> generation cephalosporins.

•they cause subacute or chronic endocarditis and are often present with embolic lesions from large biofilm vegetations in heart valves.

•they mostly occur in elderly patients, patients receiving prolonged antibiotics or iv nutrition through central vascular catheters or immune compromised patients

# 5-yeast & filamentous fungi:

Most common: candida albicans

Least: candida spp

Candida is a normal oral-intestinal-urinary tract-vagina flora, infection follows catheters or respiratory intubation

# Notes:

1-endocarditis due to Histoplasma capsulatum/aspergillus species Is very rare (only in immunocompromised)

2-there is an increase in the likelihood of developing fungal endocarditis in immunocompromised patients especially ones with HIV.

# **Candida albicans Pseudohyphae**



# Pathophysiology:

•A healthy cardiac endothelium is resistant to frequent bacteremia caused by daily activities.

•Blood stream infection is prerequisite for IE development.

•The development of IE requires simultaneous occurrence of:

1-bacteremia with an organism capable of attaching and colonizing valve tissue.

2-alteration of cardiac valve endothelium (to produce a suitable site for bacterial colonization and attachment).

3-creation of vegetations (platelets, fibrin & neutrophils burry they bacteria which appears as vegetations)

Note:1) vegetations can disseminate and result in systemic emboli.

2)patients are given a long term antibiotic because the bacterial colonization can multiply (but are metabolically inactive)

There are favorable conditions for microorganisms to colonize and they are :

1-absence of continuous endothelial cells (ex ;mechanical injuries)

2-inflammed continuous endothelial cells (ex; in rheumatic fever)

In both cases its easier for microorganisms to attach and colonize

inflammatory endocarditis

#### acute

1-fast onset of action (few weeks to months)

2-caused by staph.aureus & group A beta hemolytic streptococcus &streptococcus pneumoniae

3- febrile

 4- rapidly damages cardiac strictures & seeds extracardiac sites

5-if untreated can cause death within weeks

subacute (indolent) 1-needs months to a year

2-caused by G-ve & viridians

3-rarely damages cardiac structure & slowely if it does

4- rarely metastasizes

5-gradually progressive unless complicated by a major embolic event or a ruptured mycotic aneurysm

**Note:** the main differences between acute and subacute endocarditis are: level of toxicity ,onset of action & disease progression.

# Modified duke criteria:

It's a criteria that helps us identify IE, by giving us major and minor points that if present can determine the possibility of IE existence.

# major criteria:

1-+ve blood culture of an IE causing organism

2-transthoracic echocardiography showing any evidence of heart vegetations (or structural damage)

Minor criteria:

1-fever

2-predisposition factors

3-IV drug abuse

4-vascular phenomena (Janeway lesions)

5-immunological phenomena (Osler's node)

•depending on the patients findings or risk factors we classify IE diagnosis to : definite , possible or rejected diagnosis

1-if 2 major criterias were found =definite

2-1 major criterion & 3 minor = definite

3- 5 or more minor criterion =definite

Note: the most common presentations are :

- 1- Fever (90-95% of cases)
- 2- Murmur (90-95% of cases) : it could be newly onset or a verification in an already existing murmur.

# manifestations cardiac manifestations:

1-vulvar damage & ruptured chordae may result in regurgitant murmurs.

2-congestive heart failure : develops in (30-40%) of patients as a result of vulvar dysfunction.

#### non-cardiac manifestations (minor criteria):

1-non suppurative peripheral manifestations of subacute endocarditis (ex; janeway lesions, non tender and painless (vascular phenomena))

2-septic embolization mimicking some of these lesions (ex; Oslers node in acute S.aureus endocarditis its tender and painful (immunological phenomena))

•subungual hemorrhages are a painless vascular phenomena, sometimes the emboli can reach to other places in the body and cause infarctions ex; in lungs or brain or periphery.



Subungual hemorrhage

**Janeway lesions** 

**Osler's nodes** 

**Note :** 1-fever & murmurs for the first time : IE until proven otherwise

2-fever & change in existing murmurs : IE until proven otherwise

3-IE patients will have a good amount of medical history

# Diagnosis:

## We mainly use duke criteria

- 1- Blood culture (most imp): +ve for IE causing organisms
- 2- Echocardiography showing evidence of structural heart defect
- 3- Non-blood culture tests (if we got a -ve blood culture we use them):
  - a- Serologic test culture
  - b- Microscopic examination with special stains
  - c- Direct fluorescence antibody techniques
  - d- Pcr encoding 16s or 28s ribosomal units

## Management:

1- Antimicrobial therapy : ( we use it because 90% of IE cases are caused by G+ve bacteria , however if patient didn't respond then it might be a fungal IE).

We use IV vancomycin +Gentamicin (initiated immediately after blood samples are taken for cultures)

**Note:** extended courses of parenteral therapy with bactericidal or fungicidal agents are typically required.

2- Surgical treatment : for vegetations

# **Remember:** G+ve = vancomycin+gentamycin

Enterococci= teicoplanin

G-ve= 3<sup>rd</sup> generation cephalosporins

Fungi=amphotericin B + sometimes surgery

# **Prevention:**

•Systemic antibiotic administration prior to many bacteremia -inducing procedures.

•you have to give patients with a high risk factor (IHD, structural defect in heart & in invasive surgeries) prophylaxis!