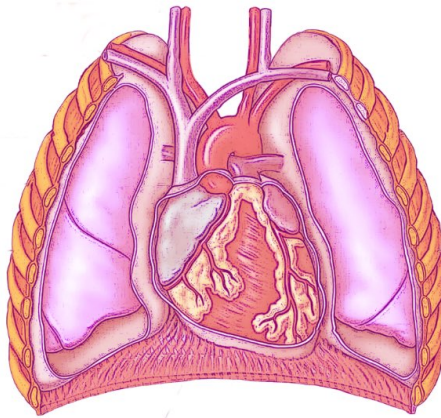




Internal Medicine



Topic : Infective Endocarditis Notes
Done by : Johainah Taha

Infective Endocarditis

Definition

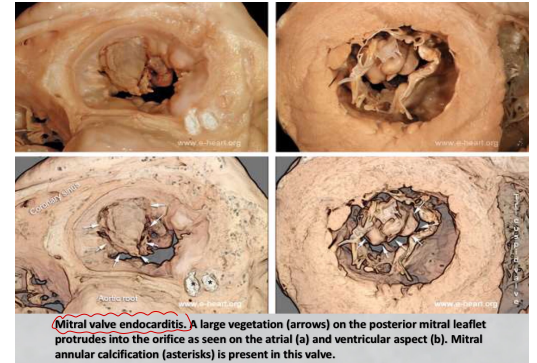
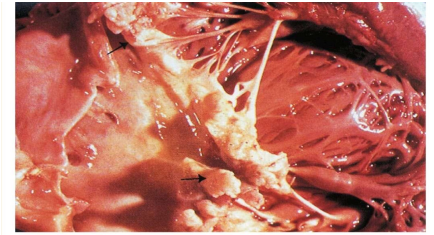
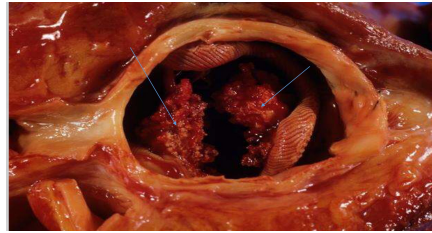
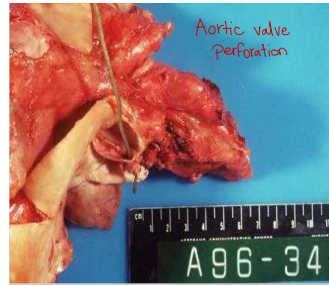
Infective endocarditis is a microbial infection that affects:

- A heart valve (native or prosthetic)
- The lining of a cardiac chamber or blood vessel
- A congenital anomaly (e.g., septal defect)

Causative Organisms

Type	Examples
Bacteria	<i>Staphylococcus aureus</i> , <i>Streptococcus</i> spp.
Rickettsia	<i>Coxiella burnetii</i> (Q fever endocarditis)
Chlamydia	<i>Chlamydia psittaci</i> (rare)
Fungi	<i>Candida</i> , <i>Aspergillus</i> spp. (mainly in immunocompromised patients)

- Both native and prosthetic valves can be affected.



Illustrations

- Mitral valve endocarditis: Large vegetation on the posterior mitral leaflet, visible on atrial and ventricular aspects.
- Acute bacterial endocarditis: Caused by *Staphylococcus aureus*, leading to aortic valve perforation and vegetations.

Epidemiology

Statistic	Details
Incidence	5–15 cases per 100,000 annually
Age Distribution	>50% of patients are over 60 years old
Underlying Cardiac Conditions (large british study)	Prevalence
Rheumatic heart disease	24%
Congenital heart disease	19%
Other abnormalities (e.g., calcified aortic valve, floppy mitral valve)	25%
No pre-existing cardiac abnormality	32%
Case Fatality Rate	~20% even with treatment (higher in prosthetic valve endocarditis & antibiotic-resistant infections)

Pathogenesis: How the Infection Develops

Stage	Process
1. Endothelial Damage	Damage occurs, attracting platelets and fibrin (deposits)
2. Colonization	Blood-borne organisms adhere to the site
3. Vegetation Formation	Bacteria, fibrin, and platelets form vegetations
4. Tissue Destruction	Nearby tissues are damaged , and abscesses may form
5. Complications	Valve regurgitation, embolization, extracardiac manifestations

Pathophysiology

pre-existing endocardial damage + risk factor + micro organism → Vegetation → Stasis → Complications

Key Factor	Details
Site of Infection	Occurs at sites of pre-existing endocardial damage
Virulent Organisms	<i>Staphylococcus aureus</i> can infect normal hearts
IV Drug Use	Staphylococcal endocarditis commonly affects the tricuspid valve
High-Risk Lesions	Ventricular septal defect, mitral regurgitation, aortic regurgitation
Low-Risk Lesions	Large atrial septal defects (due to low-pressure flow)

Table 8 Cardiac and non-cardiac risk factors

Cardiac risk factors
Previous infective endocarditis
Valvular heart disease
Prosthetic heart valve
Central venous or arterial catheter
Transvenous cardiac implantable electronic device
Congenital heart disease
Non-cardiac risk factors
Central venous catheter
People who inject drugs
Immunosuppression
Recent dental or surgical procedures
Recent hospitalization
Haemodialysis

Important

- * The most common cause of IE → *S. aureus* → Gme with acute endocarditis.
- * The most common cause of IE with dental procedure → *Viridans* → Gme with Subacute.
- * The most common cause of IE with Colon Cancer → *Strep. gallolyticus*.

Effects on the Heart and Body

Complication	Description
Valve Regurgitation	Tissue distortion, cusp perforation, chordae rupture
Embolization	Fragments of vegetations travel to distant organs
Vasculitis & Skin Lesions	Immune response or embolization effects
Mycotic Aneurysms	Arterial wall infection due to emboli
Organ Infarctions	Spleen, kidneys, and brain infarctions in severe cases
Glomerulonephritis	Immune complex deposition in kidneys

Fetal Cases

Other Definition

• Microbial infection of a heart valve (native or prosthetic), the lining of a cardiac chamber or blood vessel, or a congenital anomaly (e.g., septal defect).

• Causative organisms:

- Usually bacteria (most commonly streptococci or staphylococci). 3/4 of Cases.
- Can also include rickettsia, chlamydia, or fungi.
- Valves affected: Both native and prosthetic valves

Pathophysiology

Mechanism	Details
Site of infection	Usually occurs at sites of pre-existing endocardial damage .
Virulent infections	Staphylococcus aureus can cause endocarditis in a previously normal heart.
Risk factors	- Intravenous drug use → Tricuspid valve endocarditis - High-pressure jets of blood → Ventricular septal defect, mitral regurgitation, aortic regurgitation (even if hemodynamically insignificant). - Low-pressure lesions (e.g., large atrial septal defect) → Minimal risk .

Pathogenesis

- **Endothelial damage** → Platelet & fibrin deposits → **Bacterial colonization**. - **Avascular valve tissue** and **fibrin aggregates** protect bacteria from immune defenses. - **Vegetations** (bacteria, fibrin, platelets) form → Possible **obstruction or embolism**. - **Valve damage**: Cusp perforation, chordae disruption → **Valve regurgitation**. - **Extracardiac effects**: Embolization → **Vasculitis, skin lesions, mycotic aneurysms**.

Microbiology

Pathogen	Source & Clinical Significance
Streptococci (Viridans group)	- Oral commensals → Enter bloodstream via chewing, tooth brushing, or dental procedures. - Common cause of subacute endocarditis .
Enterococcus spp. (<i>E. faecalis</i> , <i>E. faecium</i> , <i>Strep. gallolyticus</i>)	- Bowel/urinary tract source. - Strep. gallolyticus → Associated with colon cancer → Colonoscopy recommended .
Staphylococcus aureus	- Most common cause of acute endocarditis . - Originates from skin infections, abscesses, IV catheters, or IV drug use . - Highly virulent → Rapid valve destruction & abscess formation. (produces vegetations)

Other streptococci (<i>S. pneumoniae</i> , <i>S. pyogenes</i>)	- Less common but can cause acute endocarditis.
HACEK organisms * 3-4% of Cases.	- Slow-growing Gram-negative bacteria : - <i>Haemophilus aphrophilus</i> (<i>Aggregatibacter aphrophilus</i>) - <i>Aggregatibacter actinomycetemcomitans</i> - <i>Cardiobacterium hominis</i> - <i>Eikenella corrodens</i> - <i>Kingella kingae</i> - Oral commensals → Require prolonged culture . - May be penicillin-resistant .
Coxiella burnetii (Q fever endocarditis)	- Farm animal exposure . - Aortic valve is typically affected. - Can present with hepatitis, pneumonia, purpura .

Brucella spp.	- Exposure to goats or cattle . - Affects aortic valve .
Fungi (<i>Candida</i> , <i>Aspergillus</i>)	- Immunocompromised patients or prosthetic valves . or with pt with intravenous catheters.

منشرة في مناطق المراهقين المتوسط وجنوبي أوروبا والبلد الشام

له العلاج يجب ويمكن ختاج لـ Surgical intervention

Brucella

- Characteristics
 - Gram negative coccobacillus**
 - Facultative intracellular
- Transmitted via ingestion of **contaminated animal products** (e.g. unpasteurized milk)
 - May also be transmitted by direct contact with infected sheep/goats
 - Survives in macrophages in the reticuloendothelial system
 - Can form non-caseating granulomas
- Presentation
 - Undulant fever**
 - Night sweats may be seen
 - Lymphadenopathy**
 - Hepatosplenomegaly**
 - Arthralgia
- Diagnosis
 - Blood or bone marrow culture
- Treatment
 - Doxycycline and rifampin**
 - Streptomycin
 - indicated for relapse of disease



A 55 year old hypertensive male, presents with fever with rigor and nightsweats since 2 months. Patient also complains of worsening dyspnea over the last month. Patient has past medical history of endocarditis and past surgical history of aortic valve repair 20 years back. He is ill looking, and febrile. Blood cultures are obtained and ECG taken. (lead 2 given below)



prolonged PR interval → infective endoca.

What is the most likely complication to have developed?

- Severe valvular incompetence
- Aortic root abscess**
- Ventricular aneurysm
- Valvular stenosis
- Pericardial effusion

← مهم جداً و منوفع



Figure 12.1 Janeway lesions are hemorrhagic macules or nodules that may appear on the palms of the hands or soles of the feet in cases of infective endocarditis.

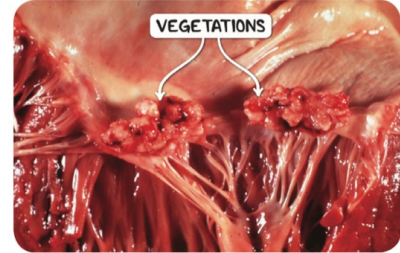


Figure 12.2 Bacterial vegetations on the mitral valve in endocarditis.

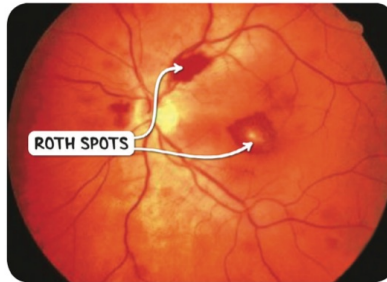


Figure 12.3 Roth spots seen in the retina.

Clinical Features

Type	Characteristics
Subacute endocarditis <i>* it may develop acute life threatening complications, such as valve disruption or emboli.</i> <i>بتكون أصعب وأخطر</i> <i>ويجي بعدة attacks</i> <i>وتكون بدأت مع ال</i> <i>Complications</i>	- Low-virulence organisms (Viridans streptococci). - Affects previously abnormal valves (left > right) . - Symptoms: Persistent fever, fatigue, night sweats, weight loss . - Can lead to embolic stroke or peripheral arterial embolism . - Signs: Purpura, petechiae, splinter hemorrhages, Osler's nodes, clubbing (late sign).
Acute endocarditis	- Highly virulent organisms (Staph. aureus) . - Can affect previously normal valves . - Right-sided involvement in IV drug users . - Symptoms: High fever, changing murmurs, petechiae . - Rapid progression: Embolism, cardiac/renal failure, abscesses.

**No splinter hemorrhage*
**No Osler's or janeway*
**No clubbing*

Post-operative endocarditis	- Can present as unexplained fever after heart valve surgery. - Infection of valve ring → Subacute or acute form . - High morbidity & mortality → Often requires surgical revision . <i>فترتيب الأحداث بالأسابيع الأولى بعد العملية</i>
Post-operative (prosthetic valve) endocarditis	- Coagulase-negative staphylococci (Staph. epidermidis) from surgical contamination . - Frequently associated with wound infection . - Can be mistaken for blood culture contamination . - Highly destructive → Normal valves & multiple emboli .

Clinical manifestation
80-90% → Fever
80-85% → Murmur
30% → Leukocytosis

Key Points

1. Streptococci & Staphylococci cause most cases.
2. Viridans streptococci → Subacute endocarditis; Staph. aureus → Acute, aggressive disease.
3. Embolization risk is high, leading to strokes, infarctions, and aneurysms.
4. Post-operative endocarditis can involve prosthetic materials & valve rings.
5. Fungal endocarditis is rare but occurs in immunocompromised patients

Pathophysiology of Osler's and Janeway Nodules

Feature	Osler's Nodes	Janeway Lesions
Cause	Deposition of immune complexes	Septic emboli depositing bacteria
Pathophysiology	Immune complex deposition triggers an inflammatory response, leading to swelling, redness, and pain.	Microabscesses form due to septic emboli, causing small-vessel thrombosis without vasculitis .
Appearance	Painful, tender, red nodules	Painless, flat, red or hemorrhagic macules
Location	Fingers and toes	Palms and soles
Associated Condition	Subacute bacterial endocarditis	Acute infective endocarditis

Investigations To confirm the diagnosis

1. **Blood Culture:** The most critical investigation for identifying the causative organism and guiding treatment.

• Procedure:

- Before starting antibiotics, **take 3-6 sets of blood cultures**.
- For suspected native valve endocarditis (NVE), prosthetic valve endocarditis (PVE), or cardiac implantable electronic device (CIED) endocarditis, obtain **three 2-bottle blood culture** sets from different venipuncture sites, spaced at least 2 hours apart over 24 hours.
أخذ 3 مجموعات 2 bottle لكل Culture
- If cultures remain negative after 48-72 hours, obtain two or three additional blood culture sets and consult the lab for specialized culture techniques.

** if I suspect a fungal infection; I can call the lab to stay for 1 month.*

ممكن ان media غير مناسبة.
 . Sets لزيت بنوعه

• Key points:

- First two specimens detect 90% of culture-positive cases.
- Cultures should be taken from peripheral sites at ≥ 6 -hour intervals to minimize the risk of skin contamination.
- Isolation of the same organism in multiple cultures supports the diagnosis.
- Do not use in-dwelling lines for blood cultures.
- Both aerobic and anaerobic cultures are required.

2. Echocardiography

→ Detecting
 → Following
 → Assess
 → Valve damage.
 → abscess formation.
 → See Complications.

- Essential for detecting vegetations, assessing valve damage, and identifying abscess formation.
- Helps monitor intracardiac complications and evaluate cardiac function.
- Two main types:

Type	Key Features	Sensitivity	
Transthoracic Echocardiography (TTE)	- Non-invasive and highly specific. - Cannot detect vegetations < 2 mm. - Technically inadequate in ~20% of cases (e.g., due to emphysema or obesity).	65–80%	
Transesophageal Echocardiography (TEE)			→ Safe but invasive. - More sensitive ($> 90\%$ detection rate). - Recommended for prosthetic valve endocarditis (PVE), myocardial abscess, valve perforation, intracardiac fistulae, and patients with CIED. - If TEE is negative but suspicion is high, repeat in 7–10 days.

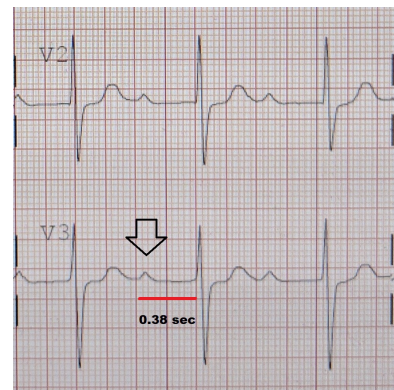
→ maybe the vegetations are small.

3. Laboratory Tests

Test	Findings	Clinical Relevance
Erythrocyte Sedimentation Rate (ESR)	Elevated	Non-specific but supportive evidence
C-Reactive Protein (CRP)	Elevated	More reliable than ESR for monitoring disease progression
Complete Blood Count (CBC)	Normocytic normochromic anemia, leukocytosis	Common but not always present
Urinalysis	Proteinuria, non-visible hematuria	Suggests immune-mediated glomerulonephritis

4. Other Investigations

Test	Findings	Significance
Electrocardiogram (ECG)	Atrioventricular (AV) block → prolonged PR interval	Suggests aortic root abscess
Chest X-ray (CXR)	Cardiomegaly, pulmonary congestion	Indicates heart failure or embolization



Modified Duke Criteria for Infective Endocarditis (IE)

Diagnosis Classification

Diagnosis	Criteria
Definite Endocarditis	- 2 major criteria, OR - 1 major + 3 minor criteria, OR - 5 minor criteria
Possible Endocarditis	- 1 major + 1 minor criteria, OR - 3 minor criteria

سؤال متوقع

①

Major Criteria.

Category	Criteria
Positive Blood Culture	- Typical organism from two separate blood cultures - Persistent bacteremia with two positive cultures >12 hours apart - Three or more positive cultures taken over >1 hour
Endocardial Involvement	- Echocardiographic evidence of vegetations , abscess, or prosthetic valve dehiscence - New valvular regurgitation (not pre-existing or worsening murmur)

Typical Microorganisms in Infective Endocarditis

Organism	Notes
Staphylococcus aureus	Most common cause, associated with IV drug use and prosthetic valves
Viridans streptococci	Associated with dental procedures and subacute IE
Streptococcus gallolyticus (formerly <i>S. bovis</i>)	Linked to colon cancer and GI disease
HACEK group	<i>Haemophilus spp</i> , <i>Aggregatibacter spp</i> , <i>Cardiobacterium hominis</i> , <i>Eikenella spp</i> , <i>Kingella kingae</i> – slow-growing, culture-negative IE
Enterococci (community-acquired)	Common in elderly patients, associated with GI or urinary tract infections

Persistently Positive Blood Cultures

Organism Type	Blood Culture Requirements
Typical IE Organisms	At least two positive blood cultures drawn >12 hours apart
Skin Contaminant Organisms	Three or majority of ≥4 positive cultures , with first and last drawn ≥1 hour apart
Coxiella burnetii	Single positive blood culture or phase I IgG antibody titer >1:800

②

Minor Criteria

Category	Criteria
Predisposition	- Pre-existing valvular or cardiac abnormality - Intravenous drug use
Clinical Signs	Fever ≥38°C - Embolic phenomena (e.g., stroke, Janeway lesions, splenic infarcts) - Vasculitic phenomena (e.g., Osler's nodes, Roth spots, glomerulonephritis)
Microbiological Evidence	- Positive blood culture not meeting major criteria
Imaging Findings	- Echocardiographic abnormalities suggestive but not definitive for IE

◆ **Major Criteria:**

- ✓ **Positive blood cultures** for typical organisms.
- ✓ **Vegetations seen on echo** (or new valve regurgitation).

◆ **Minor Criteria:**

- ☑ Fever ≥38°C.
- ☑ Predisposing condition (valve disease, IV drug use).
- ☑ Vascular signs (Janeway lesions, emboli).
- ☑ Immunologic signs (Osler's nodes, Roth spots).
- ☑ Positive but non-specific blood culture.

Left-Sided vs. Right-Sided Infective Endocarditis (IE)

Feature	Left-Sided IE	Right-Sided IE
Valves Affected	Mitral or Aortic Valve	Tricuspid or Pulmonic Valve
Prevalence	Most common form of IE	Accounts for 10–20% of cases
Common Causes	Associated with structural heart disease, prosthetic valves, or prior endocarditis	Common in IV drug users and patients with indwelling catheters
Complications	- Systemic embolization (e.g., stroke, renal infarcts, splenic infarcts) - Heart failure due to valve destruction	- Septic pulmonary emboli leading to cough, pleuritic chest pain, and hemoptysis - Tricuspid regurgitation murmur

Management of Infective Endocarditis

General Principles

Management Approach	Details
Multidisciplinary Care	Involves cardiologists, infectious disease specialists, and cardiac surgeons.
Source Control	Remove any source of infection, such as infected catheters, dental abscesses, or prosthetic material.
Empirical Therapy Considerations	Depends on: - Mode of presentation (acute vs. subacute) - Suspected organism - Presence of prosthetic valves - Penicillin allergy

Subacute Presentation (Hemodynamically Stable)	- If cultures are pending, empirical antibiotics should be withheld, especially if the patient has received antibiotics in the past 2 weeks. - This allows for accurate identification of the causative organism.
Acute or Deteriorating Patients he's unstable	- Empirical treatment should begin immediately after collecting three sets of blood cultures over several hours.

لحق لوما لحاجت النتيجة.

Example

* a patient who came to you with fever with unknown origin

Search and ask for subacute complications of IE.

then took blood sample → culture → result → Decision.

((لا داعي للاستعجال هونت))

* if a patient come with acute presentation + unstable → start antibiotic as soon as possible.

Empirical Antibiotic Therapy

((الدكتور مارج يركز عليهم))

Clinical Scenario	Empirical Treatment
Subacute Presentation (Hemodynamically Stable)	Withhold antibiotics initially unless clinically necessary.
Acute Presentation	Vancomycin (1 g IV twice daily) + Gentamicin (1 mg/kg IV twice daily; adjust based on levels).
Native Valve IE (Empirical Therapy Needed)	Amoxicillin (2 g IV every 6 hours) ± Gentamicin غائب بدون especially in patients with renal impairment.
Prosthetic Valve IE	Vancomycin (1 g IV twice daily) + Gentamicin (1 mg/kg IV twice daily) + Rifampicin (300–600 mg PO twice daily). → to prevent biofilms occurrence

Definitive Therapy

Step	Details
Pathogen Identification	Once the causative organism is identified, determine its minimum inhibitory concentration (MIC) to guide therapy.
Treatment Duration	- Fully sensitive streptococcal strains may require a 2-week regimen if specific conditions are met. - Most cases require 4–6 weeks of therapy.
Surgical Consideration	Valve replacement and surgical debridement may be required, especially in cases of: - Staphylococcus aureus infections - Fungal IE - Severe valvular destruction or heart failure

Antithrombotic Therapy in Infective Endocarditis

Scenario	Recommendation
IE with no indication for anticoagulation	Do not start ^{unless he's already on these medications.} anticoagulants or antiplatelets to prevent embolism.
IE with a mechanical prosthetic valve	Carefully weigh the risks vs. benefits of continued anticoagulation.
IE with a CNS embolic event	Discontinue anticoagulation for 2 weeks , if possible.
Ongoing antiplatelet therapy	Can be continued if no active bleeding, but should not be initiated upon diagnosis of IE.

Key Considerations

- Managing anticoagulants and antiplatelets in IE is **challenging due to the risk of embolism and intracerebral hemorrhage.**
- Antithrombotic therapy does not reduce embolic risk in IE and is not routinely indicated.
- Patients with mechanical prosthetic valves may still require anticoagulation, but risks and benefits must be carefully evaluated.

🔴 **Before culture results**, start broad coverage:

- Native valve, no IV drug use** → **Vancomycin + Ceftriaxone.**
- IV drug users** → **Vancomycin** (covers Staph aureus).
- Prosthetic valve** → **Vancomycin + Gentamicin + Rifampin** (covers biofilm-producing bacteria).

✅ Once cultures are back, tailor the antibiotics:

- Strep viridans** → Penicillin G or Ceftriaxone for **4 weeks**.
- Staph aureus** → Nafcillin or Vancomycin for **6 weeks**.
- Enterococcus** → Ampicillin + Gentamicin.

ORGANISM	DRUG (DOSE, DURATION)	COMMENTS
Streptococci		
Penicillin-susceptible ^a streptococci, S. gallolyticus	• Penicillin G (2–3 mU IV q4h for 4 weeks) • Ceftriaxone (2 g/d IV as a single dose for 4 weeks) • Vancomycin ^c (15 mg/kg IV q12h for 4 weeks) • Penicillin G (2–3 mU IV q4h) or ceftriaxone (2 g IV qd) for 2 weeks plus Gentamicin ^d (3 mg/kg qd IV or IM as a single dose ^e or divided into equal doses q8h for 2 weeks)	Can use ceftriaxone in patients with nonimmediate penicillin allergy. Use vancomycin in patients with severe or immediate β -lactam allergy. Avoid 2-week regimen when risk of aminoglycoside toxicity is increased and in prosthetic valve or complicated endocarditis.
Relatively penicillin-resistant ^b	• Penicillin G (4 mU IV q4h) or ceftriaxone (2 g IV qd) for 4 weeks plus Gentamicin ^d (3 mg/kg qd IV or IM as a single dose ^e or divided into equal doses q8h for 2 weeks) • Vancomycin ^c as noted above for 4 weeks	Penicillin alone at this dose for 6 weeks or with gentamicin during the initial 2 weeks is preferred for prosthetic valve endocarditis caused by streptococci with penicillin MICs of ≤ 0.1 $\mu\text{g/mL}$.
Moderately penicillin-resistant ^b streptococci, nutritionally variant organisms, or Gemella species	• Penicillin G (4–5 mU IV q4h) or ceftriaxone (2 g IV qd) for 6 weeks plus Gentamicin ^d (3 mg/kg qd IV or IM as a single dose ^e or divided into equal doses q8h for 6 weeks) • Vancomycin ^c as noted above for 4 weeks	Preferred for prosthetic valve endocarditis caused by streptococci with penicillin MICs of >0.1 $\mu\text{g/mL}$. Regimen is preferred by some.
Enterococci ^b		
	• Penicillin G (4–5 mU IV q4h) plus gentamicin ^d (1 mg/kg IV q8h), both for 4–6 weeks • Ampicillin (2 g IV q4h) plus gentamicin ^d (1 mg/kg IV q8h), both for 4–6 weeks • Vancomycin ^c (15 mg/kg IV q12h) plus gentamicin ^d (1 mg/kg IV q8h), both for 4–6 weeks • Ampicillin (2 g IV q4h) plus ceftriaxone (2 g IV q12h), both for 6 weeks	Can use streptomycin (7.5 mg/kg q12h) in lieu of gentamicin if there is not high-level resistance to streptomycin. Use vancomycin plus gentamicin for penicillin-allergic patients (or desensitize to penicillin) and for isolates resistant to penicillin/ampicillin. Use for E. faecalis isolates with high-level resistance to gentamicin and streptomycin or for patients at high risk for aminoglycoside nephrotoxicity (see text).
Staphylococci		
MSSA infecting native valves (no foreign devices)	• Nafcillin, oxacillin, or flucloxacillin (2 g IV q4h for 4–6 weeks) • Cefazolin (2 g IV q8h for 4–6 weeks) • Vancomycin ^c (15 mg/kg IV q12h for 4–6 weeks)	Can use penicillin (4 mU q4h) if isolate is penicillin-susceptible (does not produce β -lactamase). Can use cefazolin regimen for patients with nonimmediate penicillin allergy. Use vancomycin for patients with immediate (urticarial) or severe penicillin allergy; see text regarding addition of gentamicin, fusidic acid, or rifampin.
MRSA infecting native valves (no foreign devices)	• Vancomycin ^c (15 mg/kg IV q8–12h for 4–6 weeks)	No role for routine use of rifampin (see text). Consider alternative treatment (see text) for MRSA with vancomycin MIC >1.0 or persistent bacteremia during vancomycin therapy.
MSSA infecting prosthetic valves	• Nafcillin, oxacillin, or flucloxacillin (2 g IV q4h for 6–8 weeks) plus Gentamicin ^d (1 mg/kg IM or IV q8h for 2 weeks) plus Rifampin ^f (300 mg PO q8h for 6–8 weeks) • Vancomycin ^c (15 mg/kg IV q12h for 6–8 weeks) plus Gentamicin ^d (1 mg/kg IM or IV q8h for 2 weeks) plus Rifampin ^f (300 mg PO q8h for 6–8 weeks)	Use gentamicin during initial 2 weeks; determine susceptibility to gentamicin before initiating rifampin (see text); if patient is highly allergic to penicillin, use regimen for MRSA; if β -lactam allergy is of the minor nonimmediate type, cefazolin can be substituted for oxacillin/nafcillin. Use gentamicin during initial 2 weeks; determine gentamicin susceptibility before initiating rifampin (see text).
MRSA infecting prosthetic valves		
HACEK Organisms	• Ceftriaxone (2 g/d IV as a single dose for 4 weeks) • Ampicillin/sulbactam (3 g IV q6h for 4 weeks)	Can use another third-generation cephalosporin at comparable dosage.

INDICATIONS FOR CARDIAC SURGICAL INTERVENTION IN PATIENTS WITH ENDOCARDITIS

Surgery Required for Optimal Outcome

- Moderate to severe congestive heart failure due to valve dysfunction**
- Partially dehiscence unstable prosthetic valve**
- Persistent bacteremia despite optimal antimicrobial therapy**
- Lack of effective microbicidal therapy** (e.g., fungal or Brucella endocarditis)
- S. aureus prosthetic valve endocarditis with an intracardiac complication**
- Relapse of prosthetic valve endocarditis after optimal antimicrobial therapy

Surgery to Be Strongly Considered for Improved Outcome^a

- Perivalvular extension** of infection
- Poorly responsive** S. aureus endocarditis involving the aortic or mitral valve
- Large (≥ 10 mm in diameter) hypermobile vegetations** with increased risk of embolism, particularly with prior embolic event or with significant valve dysfunction
- Persistent unexplained fever (≥ 10 days)** in culture-negative native valve endocarditis
- Poorly responsive** or relapsed endocarditis due to highly antibiotic-resistant enterococci or gram-negative bacilli

Triple-therapy RGE

Persistent or Recurrent Fever in IE

Causes and Considerations

Clinical Concern	Possible Causes
Failure to sterilize bloodstream	Metastatic infection (e.g., splenic abscess, mycotic aneurysm).
Fever lasting >10 days on appropriate antibiotics	Suggests suppurative complications and requires further investigation.
Return of fever after initial response (~30% of cases)	Intracardiac abscess or metastatic infection .
Unresponsive fever	- Myocardial or septal abscess - Large vegetations resistant to treatment - Metastatic infection
Persistent fever in uncomplicated IE	May take up to 3 weeks to resolve.

Prophylaxis for Infective Endocarditis

High-Risk Patients for IE Prophylaxis

Condition	Indication for Prophylaxis
Prosthetic heart valve	Yes
History of IE	Yes
Cardiac transplant with valvulopathy	Yes
Congenital heart disease (with high-pressure gradient lesion)	Yes
Completely repaired congenital heart defect (first 6 months post-repair)	Yes
Coronary artery stent	No increased risk, prophylaxis not needed

Give them amoxicillin or Ampicillin

سؤال متوقع

Procedures Requiring Prophylaxis

when? سؤال متوقع

Procedure Type	Prophylaxis Indicated?
Dental procedures (manipulation of gingival tissue, periapical region, or perforation of oral mucosa)	Yes
Respiratory tract procedures (involving mucosal incision)	Yes
Procedures on infected skin or musculoskeletal tissue (e.g., incision and drainage of an abscess)	Yes
Gastrointestinal/genitourinary procedures (without active infection)	No
Procedures on an infected genitourinary tract or skin	Yes (for high-risk patients)

→ only in high risk patients.

IE Prophylaxis Guidelines: AHA vs. NICE

Guideline	Key Recommendations
American Heart Association (AHA)	- Recommends antibiotic prophylaxis for high-risk patients undergoing certain procedures. - Focuses on three stages in IE pathogenesis: 1. Killing bacteria before adherence to valves. 2. Preventing adherence to the valve/fibrin-platelet thrombus. 3. Eradicating attached organisms.
National Institute for Health and Care Excellence (NICE - UK)	- Does not recommend antibiotic prophylaxis for dental procedures. - Aligns with AHA in not recommending prophylaxis for GI, GU, or respiratory tract procedures.

6. Prevention – Who Needs Prophylaxis?

Some patients need **antibiotics before procedures** to prevent IE:

- **Prosthetic valves.**
- **History of endocarditis.**
- **Congenital heart defects.**
- **Dental procedures with bleeding risk.**

🍬 **Prophylaxis: Amoxicillin 2g PO** before procedures.