4th Edition
Revised Reprint

ESSENTIALS OF MEDICAL MICROBIOLOGY

with Complimentary Ques

As per the Revised Competency-based Medical Ed

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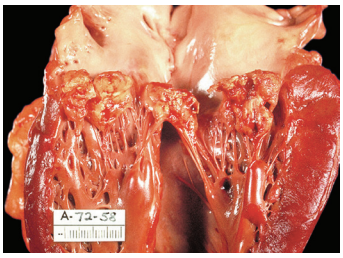
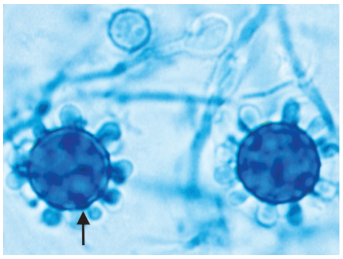
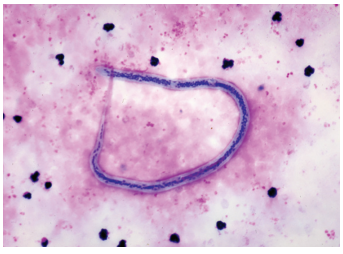






ANTIBIOTIC GUARDIAN

If we use antibiotics when not needed,
we may not have them when they are most needed

CHAPTER

28

Cardiovascular System Infections

CHAPTER PREVIEW

- Infective Endocarditis
- Other Infections of CVS
- Acute Rheumatic Fever

INTRODUCTION

Cardiovascular system infections include infections of heart and blood vessels.

- **Infections of heart:** This includes infection of the three layers of the heart wall
  - Infection of the endocardium—called *infective endocarditis*
  - Infection of the myocardium—known as *myocarditis*
  - Infection of the pericardium—called *pericarditis*. Collection of excess fluid in pericardial cavity is called pericardial effusion.
- **Infections of blood vessels**: Infections of blood vessels include mycotic aneurysm, and infective endarteritis
- **Device-related infections:** These include CRBSI (catheter-related bloodstream infection) and suppurative thrombophlebitis
- **Autoimmune-mediated:** Acute rheumatic fever.

INFECTIVE ENDOCARDITIS

Infective endocarditis (IE) refers to microbial invasion of the heart valves or mural endocardium—characteristically results in formation of bulky friable *vegetations*, composed of mass of platelets, fibrin, microcolonies of organisms, and scanty inflammatory cells.

Vegetations are most commonly present on the heart valves, followed by the low-pressure side of a ventricular septal defect, and on the mural endocardium.

Classification

Infective endocarditis can be classified into acute and subacute forms based on rapidity of evolution, severity of infection and virulence of the implicated organism (Table 28.1).

Table 28.1: Differences between acute and subacute endocarditis.

Acute endocarditis	Subacute endocarditis
Evolution is rapid	Evolution is slow
Involves normal cardiac valve	Involves previously damaged heart (scarred or deformed valve)
Implicated organism is of high virulence, e.g. *S. aureus*	Implicated organism is of low virulence, e.g. viridans streptococci
Causes substantial morbidity and mortality even with the appropriate antibiotic therapy and/or surgery	Follows a gradually progressive course of weeks to months; most patients recover after antibiotic therapy
Less common type, accounts for 10–20% of all cases	More common type, accounts for 50–60% of all cases

Pathogenesis of IE

The pathogenesis of IE involves the following sequential steps.

- **Predisposing factors:** There are several factors that can attribute the pathogenesis of IE—(i) underlying cardiac defect (e.g. mitral regurgitation), (ii) use of intravenous catheter, (iii) prosthetic valve surgery
- **Endothelial injury:** Predisposing factors produce damage to the endothelial surface of the heart, which favors deposition of platelets and fibrin, resulting in thrombus formation—a condition called *nonbacterial thrombotic endocarditis (NBTE)*
- **Colonization:** During transient bacteremia (e.g. after brushing the teeth), the bacteria adhere to the thrombus
- **Formation of vegetations:** After colonization, further deposition of platelets, fibrin, and inflammatory cells occurs surrounding the entrapped organisms—to produce **vegetation** (Fig. 28.1)
- **Metastasis:** The resulting vegetations ultimately seed bacteria into the blood at a slow but constant rate, which can metastasize to distant sites.

Etiological Agents of IE

The causative organisms of IE differ depending on the underlying risk factors such as native or prosthetic valve IE, acute or subacute IE, other risk factors such as IV drug abuser (Table 28.2).

Clinical Manifestations

The clinical spectrum of IE includes both cardiac and noncardiac manifestations.

- **Cardiac manifestations** include the appearance of a new/worsened regurgitant murmur, which is more useful for the diagnosis of IE involving a normal valve

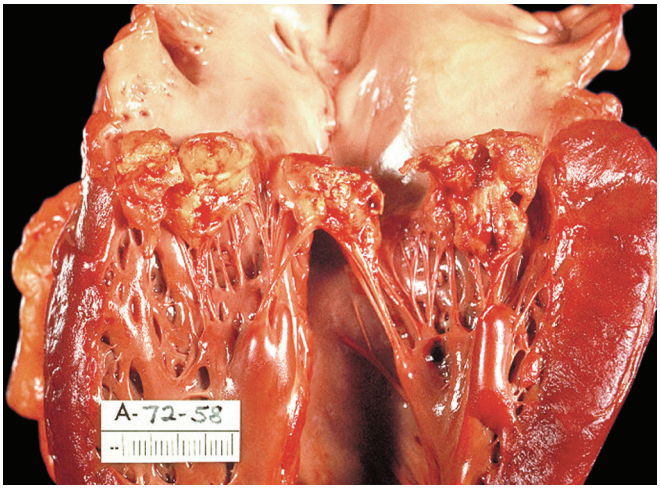


Fig. 28.1: Subacute bacterial endocarditis involving mitral valve showing large vegetations on valve leaflets.

Source: Public Health Image Library, ID # 851 (Dr Edwin)/ Centers for Disease Control and Prevention (CDC), Atlanta (*with permission*).

- **Noncardiac manifestations** include fever, chills and sweats, anorexia, weight loss, myalgia, arthralgia, arterial emboli, splenomegaly, clubbing, petechiae, neurologic manifestations and peripheral manifestations (Osler's nodes, subungual hemorrhages, Janeway lesions)
- **Laboratory findings** such as anemia, leucocytosis, microscopic hematuria, elevated ESR, CRP, or rheumatoid factor.

Diagnosis (Modified Duke Criteria)

The diagnosis of IE is established with the help of a highly sensitive and specific diagnostic schema—known as the **modified Duke criteria**; which is based on clinical, laboratory, and echocardiographic findings (Table 28.3).

Blood Cultures

Isolation of the causative microorganism from blood cultures is critical for diagnosis, determination of antimicrobial susceptibility, and planning of treatment. Blood cultures should be collected before antibiotic therapy.

- Two blood culture sets should be collected at an interval of >12hr between 1st and 2nd set
- Alternatively, three or more blood culture sets can be collected over ≥1 hour (e.g. 30 min gap between 1st and 2nd set and 30 min gap between 2nd and 3rd set).

Note: Blood culture set refers to blood collected from single venipuncture site and divided into two bottles. It is considered as positive if any of the bottle flags positive.

A major criterion can be fulfilled (Table 28.3), if:

- A typical IE organism is isolated from two separate blood cultures, or
- Agent other than typical IE organisms is isolated persistently from blood cultures in the absence of an extra-cardiac focus of infection.

A minor criterion is considered to be fulfilled (Table 28.3) if blood cultures show positive but not meeting major criterion.

Blood culture collection technique and processing is discussed in detail in Chapter 29.

Table 28.2: Agents of infective endocarditis.

Etiological agents of infective endocarditis
• *Staphylococcus aureus* • Coagulase-negative staphylococci (e.g. *S. epidermidis)* • Streptococci (Viridans streptococci and others) • Enterococci (refer Chapter 76 for details) • Pneumococci • Fastidious gram-negative coccobacilli (HACEK group) • Enterobacteriaceae • *Pseudomonas* spp. (usually in drug users) • *Candida* species • Diphtheroids
Most common agent in specific types of endocarditis
Native valve endocarditis: *Staphylococcus aureus* • Community acquired—Viridans streptococci • Healthcare associated—*S. aureus* • Overall—*S. aureus*
Prosthetic valve endocarditis (PVE): It occurs following cardiac valve replacement: • **Early PVE** (occurs within 12 months of surgery)—CoNS (e.g. *S. epidermidis*) and *S. aureus* are the most common agents • **Late PVE** (occurs after 12 months)—viridans streptococci are the most common agent • **Overall**—Regardless of the time of onset, CoNS are the most common agent, and the majority are methicillin resistant.
IE in IV drug abusers: Young males are the most common victims. The skin is the commonest source of infection. • **Right-sided IE (tricuspid valve)**—*S. aureus* is the most common agent, majority are MRSA • **Left-sided IE (mitral valve)**—more varied etiology, including Enterobacterales, *Pseudomonas aeruginosa*, *Candida,* etc. • **Overall**—Most common agent is *S. aureus*
Subacute endocarditis: Viridians streptococci
Culture-negative endocarditis: About 5–10% of IE have negative blood cultures; majority (one-third to one-half) of which are because of prior antibiotic exposure. The remainder of these patients are infected by fastidious organisms, such as: • Nutritionally variant streptococci (*Granulicatella* and *Abiotrophia*) • HACEK organisms-HACEK organisms Others: *Coxiella burnetii*, *Bartonella* species, *Brucella* species, *Tropheryma whipplei*

Note: Majority of *S. aureus* causing IE are methicillin resistant *S. aureus* (MRSA).

Abbreviations: HACEK, *Haemophilus* species, *Aggregatibacter* species, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella kingae*; CoNS, coagulase-negative staphylococci.

Non-blood-culture Tests

Various non-blood-culture tests that can be used for the diagnosis of IE include:

- Serologic tests can be used to implicate some organisms that are difficult to recover by blood culture: *Brucella, Bartonella, Legionella, Chlamydia psittaci*, and *Coxiella burnetii*
- Isolation of the pathogens in vegetations by culture
- Microscopic examination with special stains (e.g. periodic acid–Schiff stain for *Tropheryma whipplei*)
- Direct fluorescence antibody techniques
- PCR to recover unique microbial DNA or 16S rRNA that, when sequenced, allows identification of the etiologic agent.

Table 28.3: Modified Duke criteria for the clinical diagnosis of infective endocarditis.

Major Criteria
1. Positive blood culture: Any one of the following: A. Typical IE organism isolated from two separate blood culture sets (Viridans streptococci, *Streptococcus gallolyticus*, HACEK group, *S. aureus or* enterococci) or B. Persistently positive blood culture with agents other than typical IE organisms: ➢ At least two blood culture sets drawn >12 h apart; or ➢ All of 3 sets or a majority of ≥4 separate blood culture sets, with first and last drawn at least 1 h apart C. Single positive blood culture for *Coxiella burnetii* or phase I IgG antibody titer of >1:800
2. Evidence of endocardial involvement: Any one A. Positive echocardiogram ➢ Oscillating intracardiac mass on valve *or* ➢ Abscess, *or* ➢ New partial dehiscence of prosthetic valve B. New valvular regurgitation
Minor Criteria
1. **Predisposition:** Predisposing heart conditions or IV drug use 2. **Fever** ≥ 38.0°C (≥100.4°F) 3. **Vascular phenomena:** Major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages or Janeway lesions 4. **Immunologic phenomena:** Glomerulonephritis, Osler's nodes, Roth's spots or rheumatoid factor 5. **Microbiologic evidence:** Positive blood culture but not meeting major criterion as noted previously[a] or serologic evidence of active infection with organism consistent with infective endocarditis
Definite endocarditis if the followings are present: • Two major criteria or • One major criterion and three minor criteria or • Five minor criteria

[a]Excluding single positive blood cultures for coagulase-negative staphylococci and diphtheroids, which are common culture contaminants, and organisms that do not cause endocarditis frequently, such as gram-negative bacilli.
Abbreviation: IE, infective endocarditis.

Echocardiography

Echocardiography allows anatomic confirmation of infective endocarditis, sizing of vegetations, detection of intracardiac complications, and assessment of cardiac function.

TREATMENT — **Infective endocarditis**

1. Regimen for *S. aureus* IE

For native valve IE:

- ❑ **For MSSA** (methicillin susceptible *S. aureus*): Cloxacillin or nafcillin is given for 6 weeks
- ❑ **For MRSA** (methicillin resistant *S. aureus*): Vancomycin is given for 6 weeks

For prosthetic valve IE:

In addition to above regimen, rifampin (for 6 weeks) and gentamicin (for 2 weeks) are added.

2. Regimen for viridans streptococci and *S. gallolyticus* IE

- ❑ For native valve IE: Penicillin or ceftriaxone is given for 4 weeks
- ❑ For prosthetic valve IE: Gentamicin is added to the above regimen for 6 weeks

3. For HACEK endocarditis

Ceftriaxone or ciprofloxacin is given for 4 weeks. Treatment may be extended for 6 weeks in case of prosthetic valve IE.

Agents Causing IE

Infective endocarditis due to staphylococci, Viridans streptococci, nutritionally variant streptococci, and HACEK group of pathogens are discussed in this chapter. The other etiological agents of IE are discussed under different systems they principally infect.

Staphylococcal Endocarditis

S. aureus is the most common cause of IE; usually runs an acute course.

- ❖ *S. aureus* IE presents with larger vegetations (>10 mm in diameter), and therefore is more frequently associated with features of septic embolization (due to breaking of vegetations leading to formation of emboli) such as subungual hemorrhage, Osler's nodes, etc.
  - ■ Cerebrovascular emboli can cause stroke or occasionally encephalopathy
  - ■ Embolization risk is higher for mitral valve IE.
- ❖ *S. aureus* appears gram-positive cocci arranged in cluster, produces golden yellow hemolytic colonies on blood agar and gives a positive coagulase test (refer to Chapter 51)
- ❖ Coagulase-negative staphylococci (e.g. *S. epidermidis*) are increasingly associated with prosthetic valve endocarditis (at least 68–85% of cases) and majority of them are methicillin resistant.

Staphylococci can cause infections of various other systems such as skin and soft tissue (refer to Chapter 51 for details).

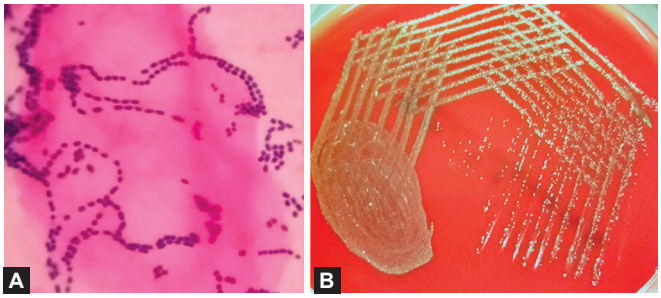
Viridans Streptococci

Viridans streptococci are commensals of mouth and upper respiratory tract. Usually, they are nonpathogenic, however occasionally cause diseases such as:

- ❖ **Subacute bacterial endocarditis (SABE):** Viridans streptococci are the most common cause of SABE. The commensal viridans streptococci (e.g. *S. mitis*—*S. sanguinis* group) in the oral cavity can enter blood to cause transient bacteremia while chewing, tooth brushing and dental procedures that can account for the predilection of these organisms to cause endocarditis
- ❖ **Dental caries**: It is mainly caused by *S. mutans* which breaks down dietary sucrose to acid and dextrans with the help of an enzyme glucosyl transferase. Acid damages the dentine, while adhesive dextran binds together with food debris, mucus, epithelial cells and bacteria to produce **dental plaques**
- ❖ **In cancer patients:** Viridans streptococci can cause prolonged bacteremia among neutropenic patients undergoing cancer chemotherapy
- ❖ ***S. milleri* group** (includes *S. intermedius*, *S. anginosus*, and *S. constellatus)*: Produce suppurative infections, particularly brain abscess and empyema.

Laboratory Diagnosis

- ❖ On Gram stain, they appear as gram-positive cocci arranged in long chains (Fig. 28.2A)
- ❖ They produce minute α-hemolytic green-colored (rarely non-hemolytic) colonies on blood agar ("*viridis*" means green, Fig. 28.2B)



Figs 28.2A and B: Viridans streptococci: **A.** Gram-positive cocci in long chains; **B.** α-hemolytic colonies on blood agar.

Source: Department of Microbiology, Pondicherry Institute of Medical Sciences, Puducherry (*with permission*).

- They can be differentiated from *Streptococcus pneumoniae* (which is also α-hemolytic) by a number of tests such as resistant to optochin and insoluble in bile (Chapter 61)
- Accurate species identification is made by automated methods such as MALDI-TOF.

TREATMENT Viridans streptococci

Most strains are susceptible to penicillin. Resistant isolates may be treated with penicillin plus gentamicin (for low level resistance) or ceftriaxone/vancomycin (for high resistance).

Nutritionally Variant Streptococci

Abiotrophia and *Granulicatella* species are known as nutritionally variant streptococci as they require vitamin B (pyridoxal) in the culture medium for their growth. Earlier, they were grouped along with viridans streptococci.

- **Manifestation:** They are normal inhabitants of the oral cavity and similar to other oral commensals, they can also cause endocarditis
- **Diagnosis:** They can be recovered in automated blood cultures such as BacT/ALERT. Multiple blood cultures and prolonged incubation may be necessary. They are gram-positive cocci and species identification can be made by automated identification systems such as MALDI-TOF.
- **Treatment:** Combination therapy with penicillin/ampicillin/ceftriaxone plus gentamicin is recommended for IE cases.

S. gallolyticus Endocarditis

S. gallolyticus (formerly *S. bovis*) is a group D *Streptococcus*, found as a commensal in intestine of animals. In humans, it occasionally causes bacteremia, subacute endocarditis, and also associated with colorectal cancer or polyps. Treatment is same as for viridans streptococcal IE cases. (Chapter 52).

HACEK Endocarditis

HACEK is an abbreviation used to represent a group of highly fastidious, slow-growing, capnophilic, gram-negative bacteria, that normally reside in the oral cavity as commensal, but occasionally have been associated with local infections of the mouth and systemic infections such as bacterial endocarditis. Species belonging to this group include:

- ***Haemophilus parainfluenzae***
- ***Aggregatibacter*** species: *A.actinomycetemcomitans,* and *A. aphrophilus*
- ***Cardiobacterium hominis***
- ***Eikenella corrodens***
- ***Kingella kingae.***

HACEK endocarditis accounts for 3% of total IE cases.

- Typically has a subacute course
- Occurs in patients with preexisting valvular defects or those undergoing dental procedures
- The aortic and mitral valves are most commonly affected.

Clinical Manifestations

- ***Haemophilus parainfluenzae:*** It is a commensal in mouth and throat
  - Occasionally, it can be an opportunistic pathogen causing endocarditis, conjunctivitis, abscesses, genital tract infections and bronchopulmonary infections in patients with cystic fibrosis
  - It can be differentiated from *H. influenzae* either by its growth requirement (requires only factor V, but not X), or by automated identification systems such as MALDI-TOF or VITEK.
- ***Aggregatibacter actinomycetemcomitans:*** Formerly called as *Actinobacillus actinomycetemcomitans*
  - It is the most common member of HACEK to cause endocarditis
  - It can also be isolated from soft tissue infections and abscesses associated with *Actinomyces israelii*
  - Rarely, it can cause periodontitis, brain abscess, meningitis and endophthalmitis.
- ***Aggregatibacter aphrophilus* and *A. paraphrophilus:*** Earlier members of *Haemophilus*, now are renamed under genus *Aggregatibacter*
  - They are commensals of mouth and occasionally cause endocarditis, head and neck infections, invasive bone and joint infections
  - *A. aphrophilus* requires only factor X, whereas *A. paraphrophilus* requires only factor V.
- ***Cardiobacterium hominis:*** It frequently affects the aortic valve. It is also associated with arterial embolization, immune complex glomerulonephritis or arthritis
- ***Eikenella corrodens:*** Apart from endocarditis, it can also occasionally cause skin and soft tissue infections. The name 'corrodens' refers to the characteristic **pitting or corroded colonies** on blood agar
- ***Kingella kingae:*** In addition to endocarditis, it can also cause infections of bones, joints and tendons.

Laboratory Diagnosis

The laboratory diagnosis of HACEK endocarditis is as follows:

- **Culture:** Blood cultures are performed on automated systems such as BacT/ALERT
  - As they are highly fastidious, require multiple blood cultures, and prolonged incubation up to 1 week
  - They are capnophilic, growth is optimum in presence of 5–10% of CO_2
  - Identification is made by automated systems such as MALDI-TOF.
- **Molecular methods:** Simultaneous detection of HACEK members from clinical specimens is possible by

performing (i) broad-range bacterial PCR targeting 16S rRNA gene followed by sequencing; (ii) multiplex PCR or multiplex real-time PCR.

TREATMENT HACEK endocarditis

The prognosis of HACEK endocarditis is good.
- ❑ Ceftriaxone (2 g/day) is the drug of choice for most of the HACEK organisms except *Eikenella corrodens* where ampicillin is indicated
- ❑ Quinolones are given if the strain is a β-lactamase producer
- ❑ Duration of treatment: Antibiotics are given for 4 weeks for native valve endocarditis and 6 weeks for prosthetic-valve endocarditis.

OTHER INFECTIONS OF CVS

Myocarditis

Myocarditis refers to inflammation of the myocardium, which is clinically manifested by chest pain, arrhythmias, or congestive heart failure. It is rapidly progressive and often fatal. It can be caused by both infectious and non-infectious etiology. The infectious etiological agents include:
- ❖ *Viruses* are the most common agents; most common being Coxsackievirus B, followed by adenoviruses, parvovirus B19, human herpesvirus 6, and dengue viruses
- ❖ *Parasitic agent* such as *Trypanosoma cruzi*, the agent of Chagas' disease
- ❖ *Bacterial agent:* It is rarely caused by bacteria, as a result of bacteremia, direct extension from a contiguous focus, or a bacterial toxin.

Pericarditis

Inflammation of the pericardium is clinically presented by one or more of the following manifestations- chest pain, pericardial friction rub, and pericardial effusion. It can be caused by both infectious and non-infectious etiology. The infectious etiologic agents include:
- ❖ *Viruses* are the most common agents; such as Coxsackievirus B (most common cause), Echovirus, Adenovirus, HIV and others
- ❖ *Bacteria* may rarely cause purulent pericarditis, usually as a complication of pneumonia due to *S. aureus*, *H. influenzae*, meningococcus and pneumococcus
- ❖ *M. tuberculosis* can cause pericarditis, usually as a complication of pulmonary tuberculosis.

Infections of Blood Vessels

Infections of blood vessels include the following conditions:
- ❖ **Mycotic aneurysm:** Refers to the inflammatory damage of an arterial wall; leading to a bulging, that can eventually rupture. The common etiologic agents are streptococci and staphylococci
- ❖ **Device-related infections:** These include infections of various devices inserted in the blood vessels such as central line and peripheral IV cannula
- ❖ **CRBSI** (Catheter-related bloodstream infection): It is a major healthcare-associated infection, discussed in detail in Chapter 22.
- ❖ **Suppurative thrombophlebitis (STP):** Refers to inflammation of a vein wall, that occurs frequently in hospitalized patients after 3–4 days of IV cannulation (e.g. veinflam), which gets colonized by the organisms present on the patient's skin or hands of the healthcare workers
  - ■ *Common etiology: S. aureus*, members of order Enterobacterales, yeasts (*Candida*) etc.
  - ■ *Lemierre's syndrome:* It is a thrombophlebitis of the internal jugular vein—caused primarily by anaerobic organism *Fusobacterium necrophorum*, following a recent oropharyngeal infection.

ACUTE RHEUMATIC FEVER

Acute rheumatic fever (ARF) is a multisystem disease that occurs in people previously affected with streptococcal (group A) sore throat, as a result of an autoimmune reaction. Although ARF may involve many parts of the body, almost all the manifestations resolve completely; except the cardiac valvular damage, which is called as rheumatic heart disease (RHD).

Group A *Streptococcus* (*S. pyogenes*) principally causes infections of skin and soft tissues and is discussed in Chapter 52.

Pathogenesis

Primary ARF is mainly a disease of children (5–14 years). However, recurrent episodes of ARF are more common in young adults. There is no gender association for ARF, but RHD more commonly affects females.
- ❖ **URTI:** ARF occurs following upper respiratory tract infection with group A streptococci (usually by M-serotypes 1, 3, 5, 6,14, 18, 19, 24, 27, and 29)
- ❖ **Genetic predisposition:** People with HLA-DR7 and HLA-DR4 appear to be more susceptible
- ❖ **Autoimmune theory:** Pathogenesis is based on the theory of **molecular mimicry**—the antibodies targeted against streptococcal antigens (M protein) cross-react with human tissue antigens (e.g. heart and joint). These cross-reactive antibodies bind to valvular endothelium, leading to damage to the heart valves (Table 52.3, Chapter 52).

Clinical Manifestations

The clinical manifestations usually appear after a period of ~3 weeks following precipitating group A streptococcal infection. The prior streptococcal infection may be either subclinical (more common) or presents as sore throat.

Acute rheumatic fever affects heart, joints, skin and brain. The common manifestations in the order of frequency include:
- ❖ **Migrating polyarthritis:** It is the most common manifestation. Joints become hot, swollen, red, and/or tender, which moves from one joint to another over a period of hours. Most commonly affected joints are the knees, ankles, hips, and elbows
- ❖ **Pancarditis,** affecting endocardium, pericardium, or myocardium

- **Subcutaneous nodules:** Occur as painless, small, mobile lumps beneath the skin overlying bony prominences, particularly of the hands, feet, and elbows
- **Chorea (Sydenham's):** It is an abnormal involuntary movement disorder, mainly affecting head and limbs
- **Erythema marginatum:** They are pink macular rashes that appear and disappear before the examiner's eyes.

Diagnosis of ARF (Jones Criteria)

The diagnosis of ARF is made based on diagnostic criteria known as revised Jones criteria (2015). It is based on the presence of a combination of typical clinical features together with ECG and laboratory findings (ESR, CRP) (Table 28.4).

Elevated anti-streptolysin O (ASO) titer was a part of Jones criteria 1992; has been removed from the revised Jones criteria (2015), as it is not a consistent marker for recent streptococcal infection.

TREATMENT — **Rheumatic fever**

Penicillin is the drug of choice; can be given orally (as penicillin V or amoxicillin for 10 days) or intramuscularly as single dose of 1.2 million units of benzathine penicillin G. Supportive treatment (e.g. aspirin) should be given for arthritis, arthralgia, and fever.

Prevention

Primary Prevention

It includes timely and complete treatment of group A streptococcal sore throat with antibiotics (penicillin) within 9 days of sore throat onset, which will prevent almost all cases of ARF.

Secondary Prevention

The mainstay of controlling ARF and RHD is secondary prevention. Patients with ARF are at much higher risk of developing recurrent ARF. Therefore, long-term penicillin prophylaxis is indicated to prevent recurrences. The drug of choice for secondary prophylaxis is benzathine penicillin G (1.2 million units, or 600,000 units if ≤27 kg) given every 4 weeks. In case of penicillin allergy, erythromycin (250 mg, twice a day) can be given as an alternative. The duration depends upon underlying carditis.

Table 28.4: Diagnostic criteria for rheumatic fever—modified Jones criteria (2015).

Major criteria	
Low-risk population*	**Moderate- and high-risk populations**
Carditis (clinical or subclinical)	Carditis (clinical or subclinical)
Arthritis—only polyarthritis	Arthritis—monoarthritis or polyarthritis
	Polyarthralgia
Chorea	Chorea
Erythema marginatum	Erythema marginatum
Subcutaneous nodules	Subcutaneous nodules
Minor criteria	
Low-risk population*	**Moderate- and high-risk populations**
Polyarthralgia	Monoarthralgia
Hyperpyrexia (≥ 38.5°C)	Hyperpyrexia (≥ 38.0°C)
ESR ≥ 60 mm/h and/or CRP ≥ 3.0 mg/dL	ESR ≥ 30 mm/h and/or CRP ≥ 3.0 mg/dL
Prolonged PR interval	Prolonged PR interval
Diagnostic criteria	
Initial ARF	Two major or One major + two minor
Recurrent ARF (with a reliable past history of ARF/RHD)	Two major or One major + two minor or Three minor criteria

Abbreviations: ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; ARF, acute rheumatic fever; RHD, rheumatic heart disease.

*Low risk is defined as an ARF incidence <2 per 100,000 school-aged children (5–14 years old) per year, or an all-age prevalence of RHD of ≤1 per 1,000 population per year.

- **ARF without carditis:** For 5 years after the last attack or 21 years of age (whichever is longer)
- **ARF with carditis but no residual valvular disease:** For 10 years after the last attack, or 21 years of age (whichever is longer)
- **ARF with persistent valvular disease:** For 10 years after the last attack, or 40 years of age (whichever is longer) or sometimes lifelong prophylaxis.

EXPECTED QUESTIONS

I. Write essay on:

Case Study Infective Endocarditis

1. A 75-year-old man was hospitalized with fever (101°F), severe back-pain and weakness in lower limbs. On examination, few non-tender, small erythematous nodular lesions on soles were seen. Echocardiogram showed valvular vegetations on mitral valve. Two pairs of blood cultures were sent which subsequently were positive for viridans streptococci. The patient was immediately started on benzyl penicillin.
   a. What is the probable clinical diagnosis?
   b. What are the typical etiological agents?
   c. Describe the diagnostic criteria used for this condition.
   d. How will you collect specimen for this clinical condition?

Case Study Acute Rheumatic Fever

2. A 7-year-old female child presented to the cardiology OPD with swollen, red, and/or tender joints, which migrates from one joint to another (knees, ankles, hips, and elbows) over a period of hours. The child was having an abnormal gait. She also complained of painless, small, mobile lumps beneath the skin overlying bony prominences, particularly of the hands, feet, and elbows. On auscultation, murmur was heard over the mitral valve area. ECG showed prolongation of P-R interval. On inquiry, it was found that the child had an episode of sore throat 3 weeks back.
   a. What is the probable clinical diagnosis?
   b. Describe the diagnostic criteria used for this condition.
   c. How will you prevent the recurrence of such episodes?

ESSENTIALS OF
MEDICAL MICROBIOLOGY

What is new in this edition?

Thoroughly revised, edited, proofread and updated; prepared based on infective syndromes, in accordance with the revised competency-based MBBS curriculum.

- **Question bank booklet** has been given complimentary, which contains chapter-wise questions as per NMC's recommendation—long essays, short notes, case scenario-based questions, reasoning type questions, MCQs (direct as well as case scenario based)
- **Divided into two parts**—(I) General Microbiology, Immunology and Hospital Infection Control and (II) Systemic Microbiology
- **Systemic Microbiology (Infectious Diseases)**—eight sections, each comprises of a first chapter on clinical infective syndrome followed by several chapters covering detailed information about the etiological agents
- **Chapters on Clinical Infective Syndromes**—represent the first chapter of every Systemic Microbiology section; covers various infective syndromes pertaining to that system in detail
- **Parasitology Topics**—have been updated as per the current need of Indian Medical Graduate and discussed under respective infective syndromes
- **Hospital Infection Control Section**—thoroughly updated in various topics such as major HAI types, monitoring of antimicrobial stewardship, escalation vs de-escalation strategy, donning/doffing of PPE and transmission-based precautions
- **General Microbiology Section**—meticulously restructured with the inclusion of general virology, general parasitology and general mycology chapters. General bacteriology is reorganized into a single chapter with several subchapters
- **Overview Chapters**—incorporated in the General Microbiology section, which will help in better understanding of organisms when discussed under Systemic Microbiology
- **COVID-19 Chapter**—has been revised and updated as per the current scenario
- **Recent Advances**—updates made in laboratory diagnosis, treatment guidelines and vaccine prophylaxis of infectious diseases; of note tuberculosis and HIV
- **Epidemiology Updates Included on**—recent outbreaks and geographical distribution with special emphasis on Indian epidemiology
- **References** followed from the latest editions of Harrison, Mandell, Bailey & Scott, CDC and WHO websites and healthcare program/guidelines such as ICMR, NCDC, NTEP, NACO, NVBDCP, Immunization and GPEI, etc.
- **Inclusion** of more tables, flowcharts, real images and schematic diagram—for better understanding
- **One weapon, two targets—MBBS exam and NExT entrance**: Only textbook written in a National Exit Test (NExT)—oriented way so that the matter of textbook does not differ a lot from a PG entrance book (major deficiency with all other Indian UG textbooks).

Most features of the previous editions are maintained

- More content, less pages—handy look, saves student's time
- Concise, bulleted format and to-the-point text—easy to read during MBBS examination
- Simple and lucid language makes the understanding easy
- Separate boxes for summary of laboratory diagnosis and treatment—for quick review
- Clinical case-based essay questions and MCQs incorporated at the end of each chapter.

Apurba S Sastry MD (JIPMER) DNB MNAMS PDCR has done his MBBS from MKCG Medical College, Berhampur, Odisha, India and residency from the prestigious institute, JIPMER, Puducherry, India. He is currently working as an Additional Professor, Department of Microbiology in the same institute. He is also the Hospital Infection Control Officer and Antimicrobial Stewardship Lead (Microbiology) of JIPMER. He is the course coordinator of the most popular HIC workshop of India—'**National Workshop on Hospital Infection Control**', conducted annually by HICP, JIPMER; in which more than 1,500 infection control practitioners across India have been trained. He is a field expert in the core area of HIC and antimicrobial stewardship program in Indian scenario. He has developed '**Clinical Microbiology Reporting Software**' based on CLSI and EUCAST guidelines, which will be revolutionary in upgrading the diagnostic stewardship to the next level. He has guided and co-guided more than 60 student dissertations and contributed more than 100 research publications. He is passionate about teaching and has been a popular teacher among the students.

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Dr Apurba Sastry and his team have also authored several other popular books which are extremely appreciated among faculty and students:

1. Essentials of Microbiology for Postgraduates (Volume 2—Systematic Bacteriology)
2. Essentials of Hospital Infection Control, 1st edition
3. Essentials of Antimicrobial Stewardship, 2nd edition
4. Essentials of Medical Parasitology, 2nd edition for MBBS
5. Review of Microbiology and Immunology, 9th edition for PG entrance examination
6. Essentials of Practical Microbiology, 2nd edition for MBBS
7. Essentials of Applied Microbiology for Nurses, 1st edition
8. Microbiology for FMGE, 2nd edition for foreign medical graduate entrance examination
9. Essentials of Microbiology, Organism Based, for Allied Health Science Courses

If any reader wishes to share feedback, suggestions, updates and errata, please feel free to write to us by the google form link https://forms.gle/vS6TqJVT3EhrNSL87. As a token of gratitude, the reader will be acknowledged in the subsequent edition of the book.

Feedback on Apurba Sastry Books

Shelving Recommendation
MICROBIOLOGY

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