

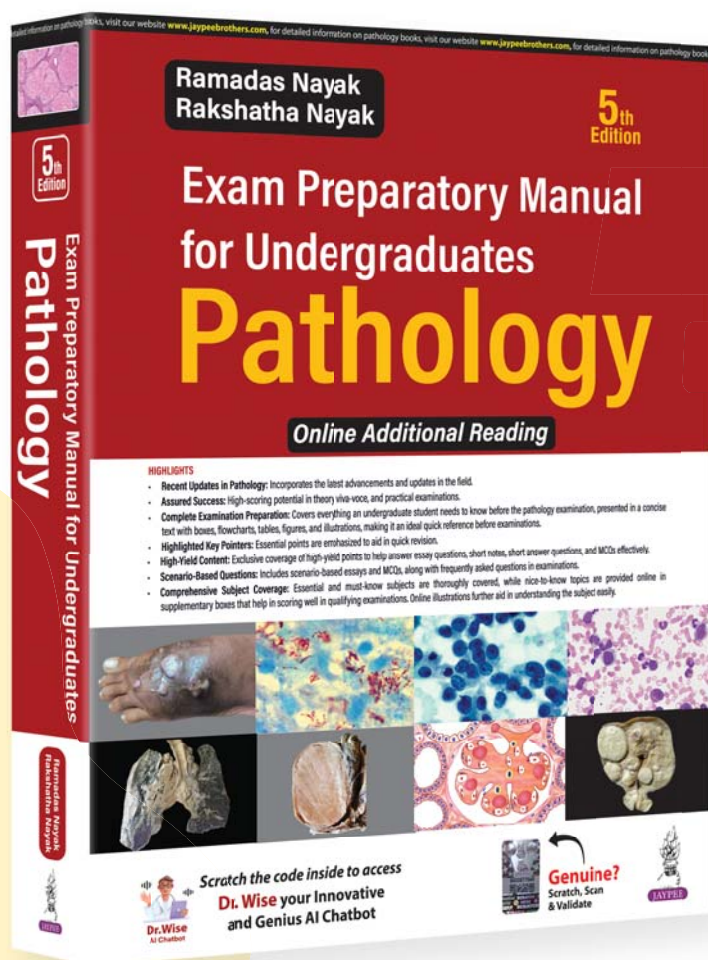
Exam Preparatory Manual for Undergraduates

Pathology

Online Additional Reading

Ramadas Nayak
Rakshatha Nayak

5th Edition



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Key Features

- This book aligns with National Medical Commission (NMC) guidelines, covering both Core and Non-Core Competencies for Indian Medical Students.
- Comprehensive Content: Covers essential disease concepts simply and clearly for undergraduates.
- Online Supplement: Additional illustrations and information are available online, aiding exam preparation beyond standard Pathology texts.
- Rich Illustrations: Features multicolor visuals for better understanding.
- Structured Organization: Divided into three sections –
 - General Pathology and Basic Cytology (Chapters 1 to 10)
 - Hematology, Blood Banking and Transfusion, and Clinical Pathology (Chapters 11 to 15)
 - Systemic Pathology (Chapters 16 to 27)
- Concise & Effective: Bullet points with key highlights for quick revision.
- Rapid Review: Designed for quick review before exams, covering usual exam questions, essays, MCQs, and clinical case interpretations.
- Enhanced Learning: Includes flowcharts, tables, and visual aids like photomicrographs and radiographs.

Chapter Outlines are Highlighted in front.

CHAPTER 4

CHAPTER OUTLINE

- ◆ Mycobacterial Infections
- ◆ Spirochete Infections
- ◆ Bacterial Diseases
- ◆ Viral Diseases
- ◆ Rickettsial Infections
- ◆ Chlamydial Infections
- ◆ Fungal Infections and Opportunistic Infections
- ◆ Mycetoma
- ◆ Parasitic Diseases
- ◆ Malaria
- ◆ Other Infections

MYCOBACTERIAL INFECTIONS

Mycobacteria are bacteria that appear as slender, aerobic rods. They grow in straight or branching chains. Mycobacteria have a characteristic waxy cell wall composed of several glycolipids and lipids, including mycolic acid. This wall is responsible for its acid fast nature on staining. Acid fast means that they will retain stains even on treatment with a mixture of acid and alcohol. They are weakly gram positive. Two important species that cause granulomatous disease are *Mycobacterium tuberculosis* (tuberculosis) and *Mycobacterium leprae* (leprosy).

PA 10.3 (CC): Define and describe the pathogenesis and pathology of leprosy.

Key information is highlighted using Competencies

pathology of leprosy

Leprosy or Hansen disease (named after the discoverer of the causative organism by Hansen), is a chronic, granulomatous, slowly progressive, destructive infection caused by *Mycobacterium leprae*. Leprosy is one of the oldest human diseases and leprosy was isolated from the coon in the 1940s. Mycobacterium leprae mainly affects the peripheral nerves, skin, and mucous membranes (nasal) and results in disabling deformities. Mycobacterium leprae The characteristic lesions of this mycobacteria are:

TABLE 4.1 Ridley and Jopling (1966) classification and associated immune response.

Group	Immune response
Tuberculoid leprosy (TT)	Polar form that has maximal immune response
Borderline tuberculoid (BT)	Immune response falls between BB and TT
Borderline leprosy (BB)	Immune response exactly falls between two polar forms of leprosy
Borderline lepromatous (BL)	Immune response that falls between BB and LL
Lepromatous leprosy (LL)	Polar form with the least immune response

CHAPTER 4: Infections and Inflammation

Classification

1. Classify leprosy

A. Ridley and Jopling (1966) Classification (Table 4.1)

It depends on the immunopathological spectrum of the immune response to the bacilli. The immune response is classified into: (1) tuberculoid, (2) borderline tuberculoid, (3) borderline leprosy, (4) borderline lepromatous, and (5) lepromatous.

B. WHO Classification

Progression of leprosy is determined by macrophages and proliferation. The leprosy is classified into: (1) tuberculoid, (2) borderline tuberculoid, (3) borderline leprosy, (4) borderline lepromatous, and (5) lepromatous. The WHO classification is based on the Ridley and Jopling classification.

Pathogenesis

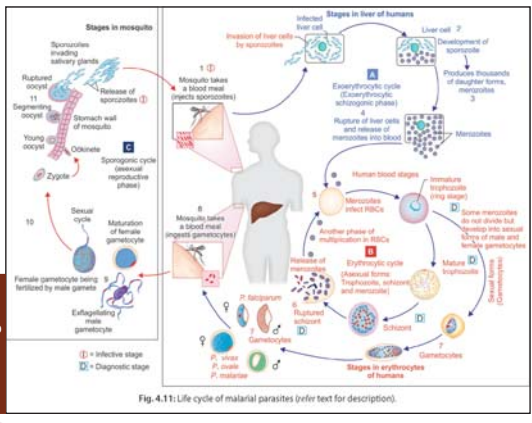
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Flowchart 4.1: Ridley-Jopling classification of leprosy

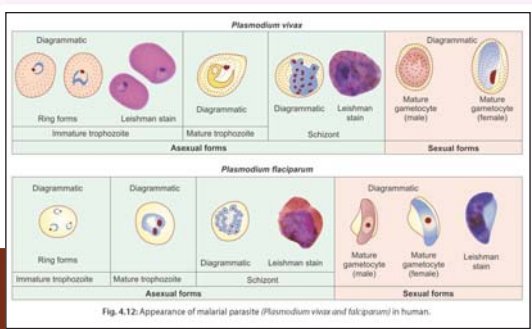
Tables are given to simplify complex topics in structured parts

Line Diagrams, Figures, & Understandable Flow Charts Given for detail understanding of concepts.

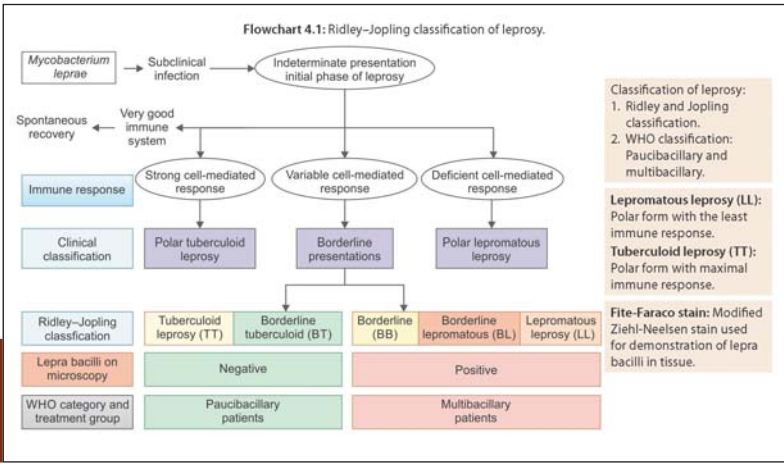
Line Diagrams



Figures



Flow Charts



BOX 7.3: Precancerous lesions of the gastrointestinal tract.

Esophagus
Barrett esophagus, achalasia, post-ingestion of caustic soda lye (alkaline burn)

Stomach

- Atrophic gastritis, especially if treated with PPI without eradication of *H. pylori*
- Gastric polyps
- Ménétrier's disease (hypertrophic gastropathy)
- Achlorhydria, pernicious anemia
- Post-gastrectomy, post-vagotomy, post-gastrojejunostomy

Small intestine
Hereditary cancer syndromes, FAP (especially post-cholecystectomy), Peutz-Jeghers syndrome

Colon (large intestine)

- Hereditary cancer syndromes, FAP, Gardner's, HNPCC (Lynch syndrome)
- Inflammatory bowel disease, especially chronic extensive ulcerative colitis
- Sporadic adenomatous polyps, especially villous and tubulovillous polyps

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TABLE 7.10: Common location of tumors as determined according to the incidence

Male	Female
Prostate	Breast
Lung and bronchus	Lung and bronchus
Colon and rectum	Colon and rectum
Urinary bladder	Cervix corpus
Melanoma of the skin	Thyroid
Kidney	Melanoma of skin
Non-Hodgkin lymphoma	Non-Hodgkin lymphoma

BOX 7.4: Major functional classes of cancer genes

- Oncogenes
- Tumor suppressor genes
- Genes that regulate apoptosis
- Genes that regulate interactions between tumor cells and host cells

Boxes are used to highlight various diagnostic understanding.

Question answers, & other Scenario-based MCQs with Explanations are given at the end of each chapter.

SCENARIO-BASED MCQs

1. A 29-year-old man presents to the dermatology outpatient department with maculopapular and nodular skin lesions. These lesions are mainly seen on his face, elbows, wrists, and knees. These lesions are has reduced to absent sensation. Microscopy of these lesions showed nodular aggregates of foamy cells (lipid-filled macrophages). Fite Feraco stain revealed numerous bacilli arranged in cigar bundles. The most likely diagnosis is:
 - A. Anthrax
 - B. Hansen disease
 - C. Neurofibroma
 - D. Neurilemmoma
2. A 22-year-old man attends the skin outpatient with a nontender ulcer on his penis for the past week. On enquiry, he revealed that he has multiple sexual partners and does not use barrier precautions. Physical examination showed a 0.7 cm lesion with a firm, erythematous, indurated base, and sharply demarcated borders. The lesion is scraped, and microscopic darkfield examination shows positive for motile spirochetes. Which of the following condition is responsible for the lesion?
 - A. Primary syphilis
 - B. Gonococcal infection
 - C. HIV infection
 - D. Tuberculous ulcer
3. A 65-year-old male has developed aortic root dilation and aortic insufficiency. He has a long history of unprotected sexual intercourse with multiple partners. The most likely disease that has produced the lesions in aorta is:
 - A. Primary syphilis
 - B. Secondary syphilis
 - C. Tertiary syphilis
 - D. Congenital syphilis

Answers and Explanation

1. Answer is B: Hansen disease (leprosy)-lepomatous form. It is caused by *Mycobacterium leprae*, which infects peripheral nerves and skin. Diagnosis is made by biopsy of a skin lesion. Large aggregates of lipid-filled macrophages contain acid-fast bacilli demonstrated by the Fite Feraco stain. Cutaneous anthrax is caused by *Bacillus anthracis*. It produces a necrotic skin lesion with eschar at the site of inoculation. The other two options are tumors of peripheral nerves and they do not show foamy macrophages with acid-fast bacilli.
2. Answer is A: Primary syphilis. Infection with *Treponema pallidum* produces characteristic painless syphilitic chancres in the primary stage of syphilis. It is nontender with an indurated base and is known as hard chancre. Gonorrhoea produces acute inflammation with abscess formation and mainly involves urethral region. HIV infection is also sexually transmitted disease but do not produce chancre. Tuberculous ulcer usually do not involve glans.
3. Answer is C: Tertiary syphilis. If infection with *Treponema pallidum* is not treated, it can progress to tertiary syphilis

OTHER INFECTIONS

Rhinospordiosis

Q. Write a short note on rhinosporidiosis.
Rhinospordiosis is an inflammatory disease caused by *Rhinosporidium seberi*. Usually, it occurs in the nasopharynx as a polyp but may also be observed in the larynx and conjunctiva. It is also observed in the oropharynx, rectum, and external genitalia. It is endemic in India and Sri Lanka and sporadic in other parts of the world.

Microscopy (Figs. 4.17A and B)

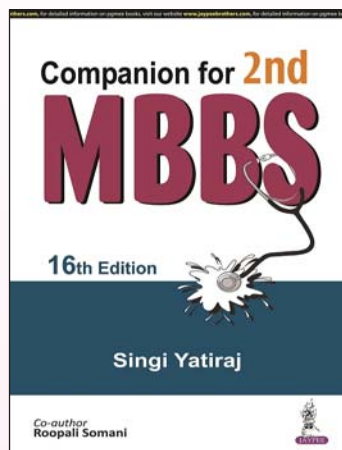
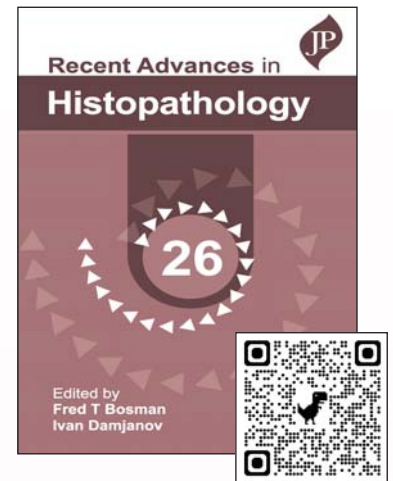
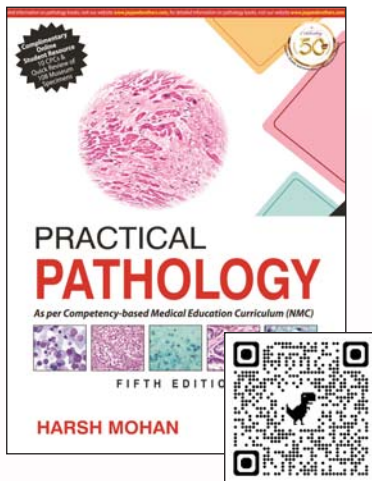
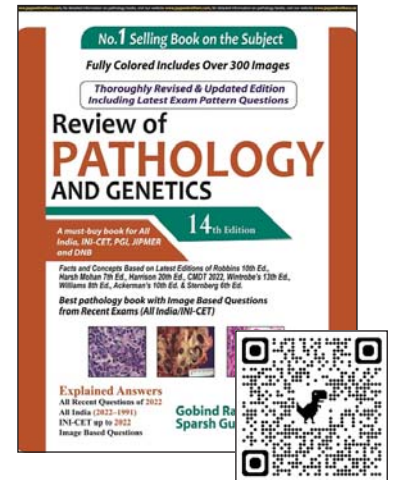
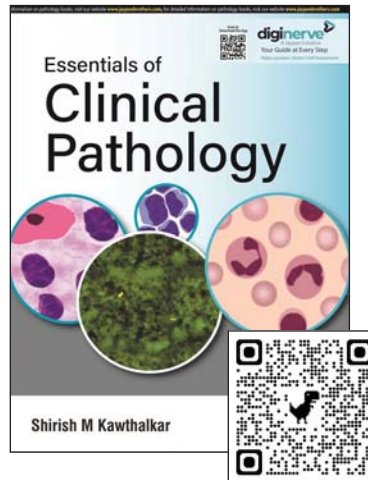
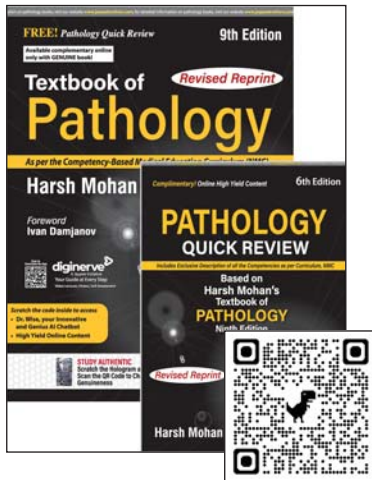
- Structure of nasal mucosa.
- Many spherical cysts called sporangia measuring up to 200 nm in diameter and having thick walls (chitinous walls) are seen. Each of these cysts (i.e., sporangium) contains numerous small basophilic round spores of the size of erythrocytes. On rupture of a sporangium, the spores may be discharged into the submucosa or onto the surface of the mucosa.
- Chronic inflammatory (plasma cells, lymphocytes, histiocytes, neutrophils) infiltrate in the intervening and subepithelial layer.

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Fig. 4.17B: Rhinosporidiosis. Sporangia with numerous small basophilic round spores of the size of erythrocytes are seen. The spores are discharged into the submucosa or onto the surface of the mucosa.

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