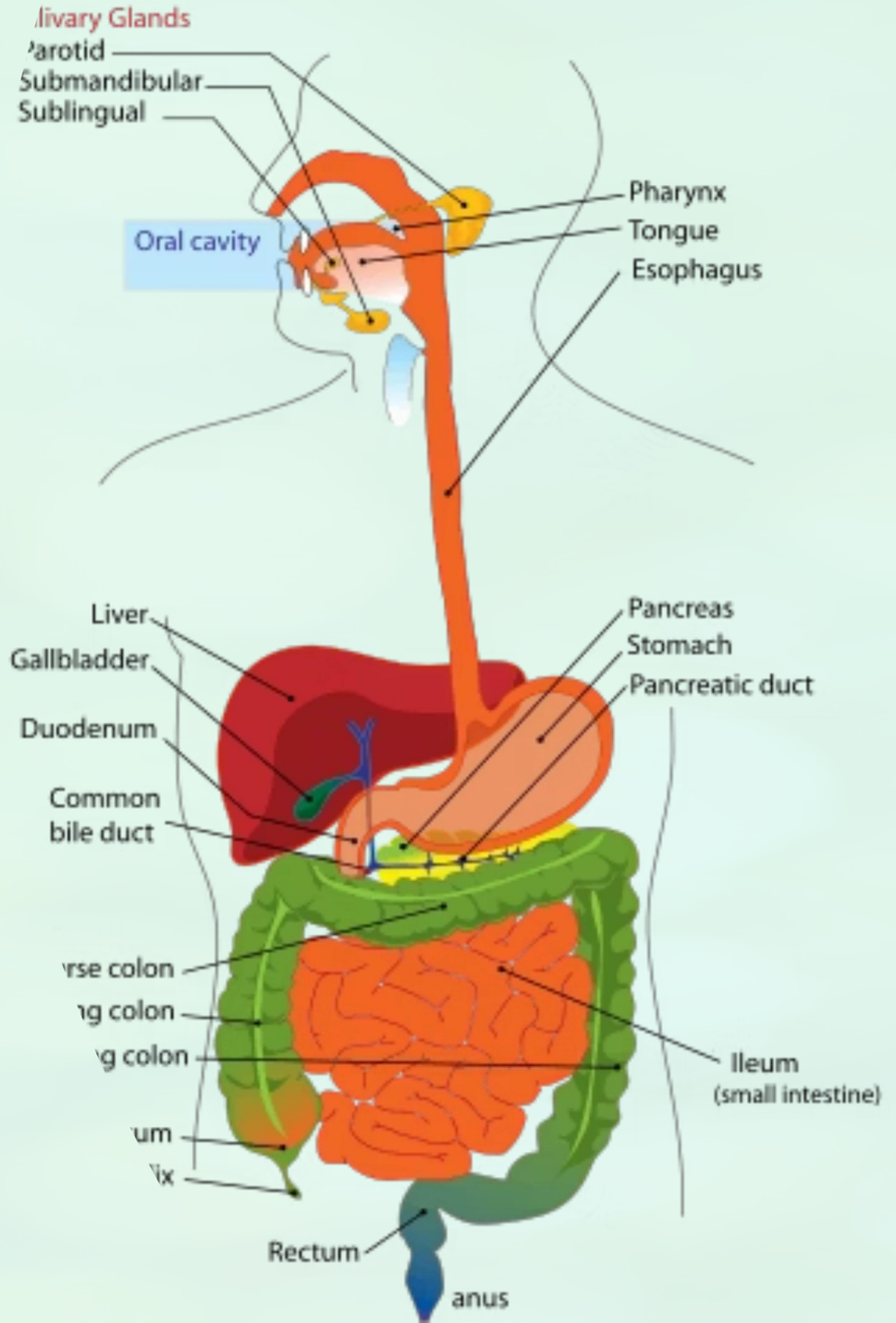
GIT Diseases

A comprehensive overview of gastrointestinal tract diseases — from oral cavity pathologies to salivary gland disorders — for medical faculty students.

MEDICINE FACULTY

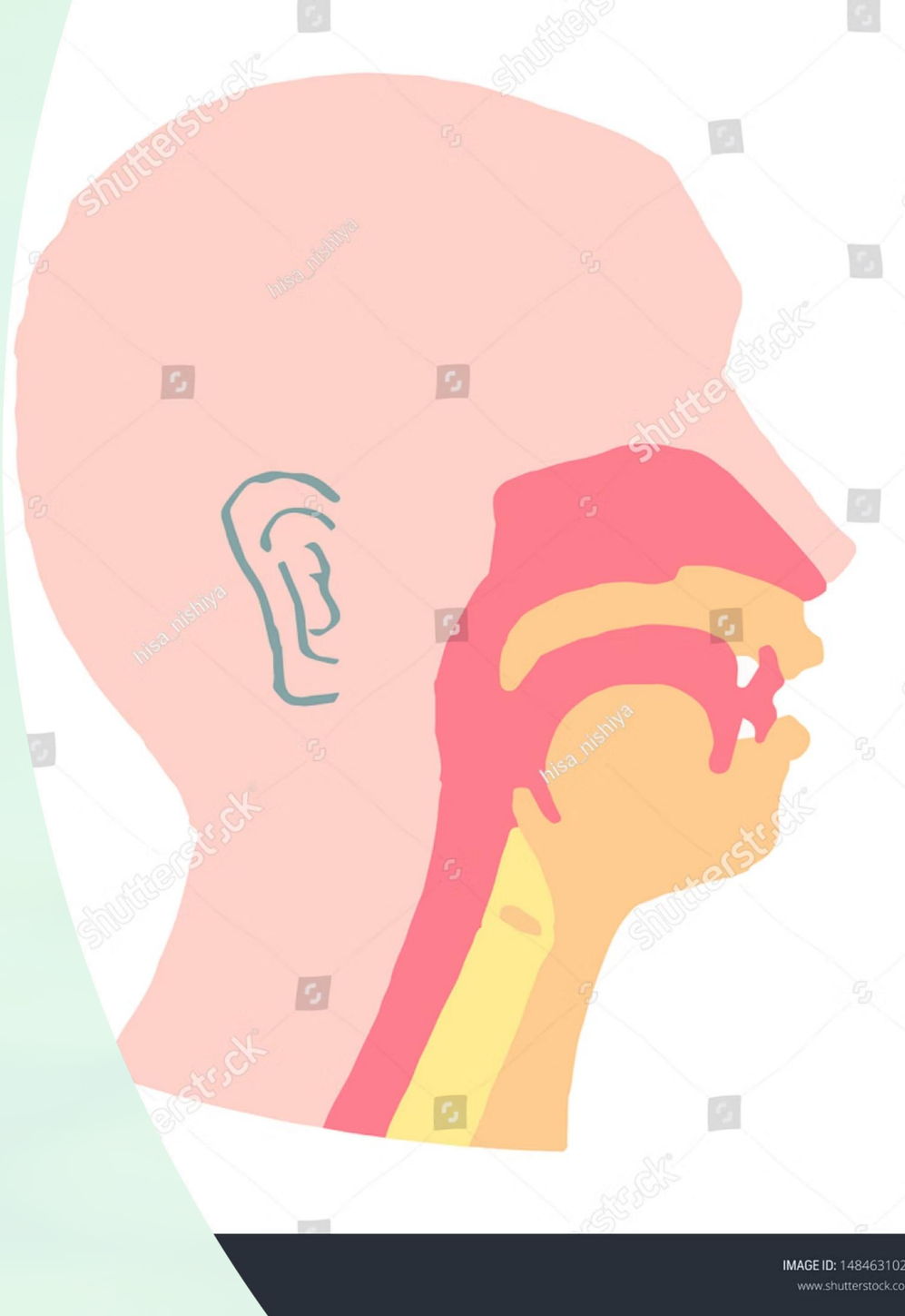
GASTROINTESTINAL PATHOLOGY



Chapter 1

The Oral Cavity

The oral cavity is the entry point of the gastrointestinal tract and the site of numerous primary and systemic diseases. Understanding its pathologies is foundational to GIT medicine.



Oral Cavity — Disease Classification

Diseases of the oral cavity can be grouped into two broad categories, each with distinct diagnostic and management implications:

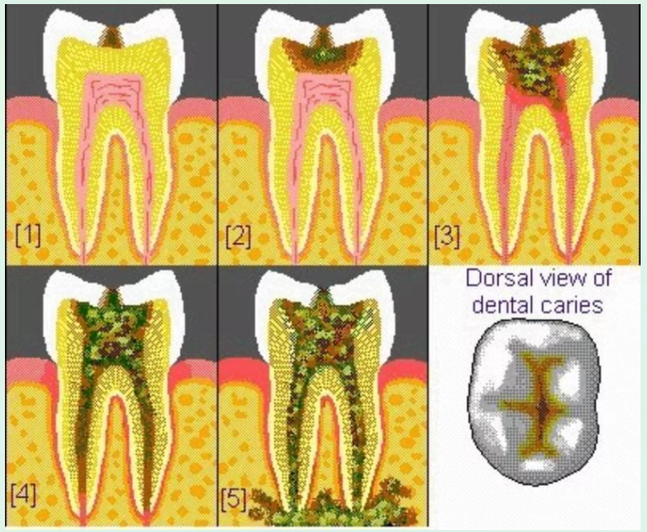
Type 1 — Primary Oral Diseases

The cause and all presenting symptoms are confined to the oral cavity. Pathological processes originate locally and do not reflect systemic illness. Examples include dental caries and periodontitis.

Type 2 — Systemic Diseases with Oral Manifestations Manifestations

The oral symptoms are only one component of a broader systemic condition. Oral findings serve as diagnostic clues to underlying diseases such as vitamin deficiencies, autoimmune disorders, or infections.

Recognizing whether oral pathology is isolated or systemic is critical for accurate diagnosis and appropriate referral.



Dental Caries

Tooth decay / cavity — progressive damage to the hard tooth structures: enamel, dentin, and cementum.

Causative Organisms

Streptococcus mutans, *Lactobacillus* spp. — acid-producing bacteria colonizing the tooth surface.

Risk Factors

Dietary sugars (sucrose, glucose, fructose, starch) are fermented into **lactic acid**. Acid + bacteria + food debris + saliva → plaque → mineralization into **tartar**.

Signs, Symptoms & Consequences

Tooth sensitivity and pain, localized infection, abscess formation, and ultimately **loss of the affected tooth** if untreated.

Periodontitis

Inflammation of the **periodontium** — the supporting structures of the teeth including the gums (gingiva), periodontal ligament, cementum, and alveolar bone.

Cause & Risk Factors

- Bacterial and mycotic infection
- Poor oral hygiene → plaque accumulation → gingival irritation
- Genetic predisposition (suspected)
- Diabetes mellitus (impairs immune response)

Signs & Symptoms

- Redness, swelling, and spontaneous bleeding of gums
- Halitosis (bad breath)
- Gingival recession — deep pockets between teeth and gums
- Pain and tooth mobility; ultimately loose or lost teeth

Oral Precancerous Lesions

Precancerous lesions of the oral mucosa require early identification, as they carry significant malignant transformation potential — especially in the presence of known risk factors.

Leukoplakia

White plaques on the oral mucosa or tongue that cannot be wiped off. Classified as a precancerous lesion. Cause: likely somatic mutation. Risk factors include smoking, human papillomavirus (HPV), and alcohol use.

Erythroplakia

Red, velvet-like plaques on the oral mucosa. Higher risk of malignant transformation than leukoplakia. Cause: suspected mutation. Risk factors: smoking and alcohol — often used in combination, which greatly amplifies the risk.

⚠ Both leukoplakia and erythroplakia must be biopsied and monitored — they may progress to invasive squamous cell carcinoma.

Oral Cancers

Malignancies of the oral cavity are predominantly epithelial in origin and strongly associated with modifiable lifestyle risk factors.

Squamous Cell Carcinoma

~90% of oral cancers. Arises from oral mucosa epithelium.

Adenocarcinoma

Originates from salivary gland tissue.

Lymphoma

Typically involves the tonsillar lymphoid tissue.

Risk Factors

- Tobacco smoking and smokeless tobacco
- Chronic alcohol consumption
- Human papillomavirus (HPV, especially HPV-16)

Clinical Progression

Initially: small, white, painless mucosal or skin lesion. Later: pain, difficulty eating and talking, visible ulceration or mass.

Oral Infections — Herpes Labialis

Herpes labialis ("cold sore") is one of the most prevalent viral infections affecting the perioral region, caused by latent reactivation of HSV-1.

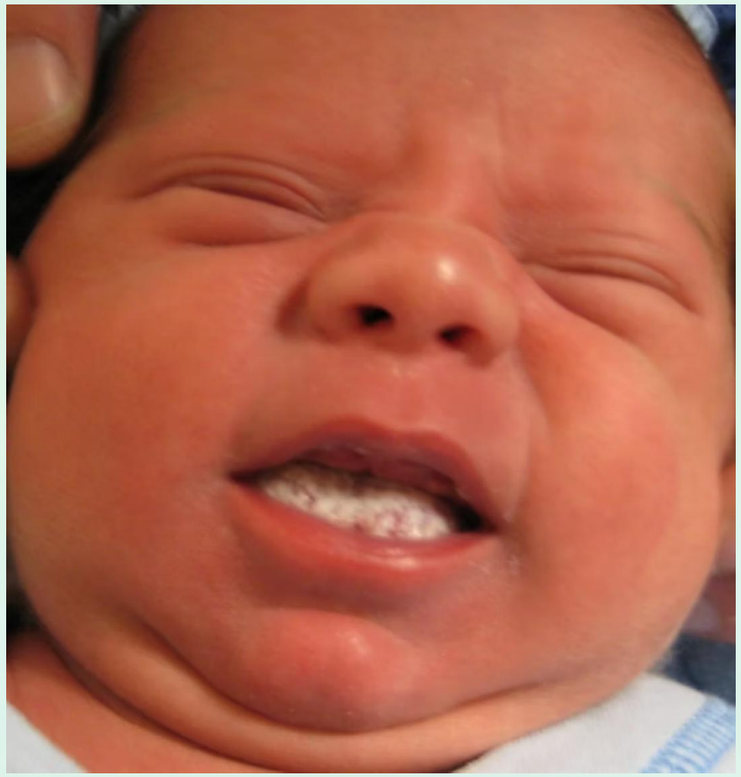
Causative Agent

Herpes Simplex Virus type 1 (HSV-1) — a neurotropic DNA virus that establishes latency in the trigeminal ganglion and reactivates under stress, immunosuppression, or UV exposure.

Signs & Symptoms

Prodromal tingling or burning, followed by clusters of **fluid-filled blisters** at the mucocutaneous junction of the lips. Blisters rupture, crust, and heal within 7–10 days. Highly contagious during active outbreak.

In immunocompromised patients (HIV, chemotherapy), HSV-1 reactivation can be severe and disseminated.



Oral Candidiasis

A fungal infection of the oral mucosa caused by *Candida albicans*, an opportunistic pathogen that proliferates when host immunity is compromised.

Risk Factors

- Newborns — immature immune system
- Immunodeficiency — diabetes mellitus, HIV/AIDS
- Antibiotic or corticosteroid therapy — disrupts normal flora and immune balance
- Hormonal changes — pregnancy alters mucosal environment
- Denture wearers — poor oral hygiene creates favorable conditions

Signs & Symptoms

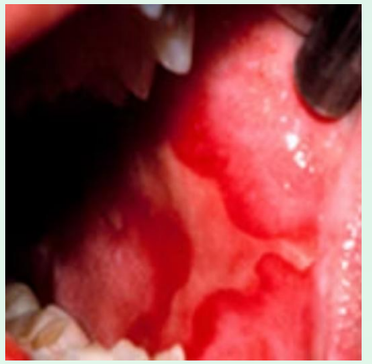
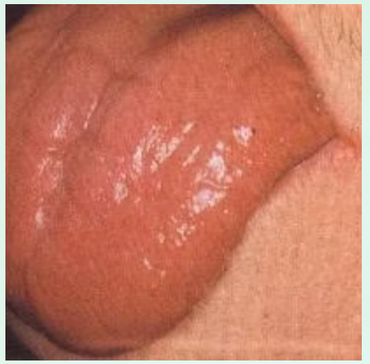
White, removable plaques ("thrush") coating the oral mucosa, tongue, or palate. May cause soreness, altered taste, or difficulty swallowing.

Chapter 2

Avitaminoses — Vitamin B Deficiency

B-group vitamins (B2, B3, B6, B9, B12) are essential for mucosal integrity and immune function. Their deficiency produces characteristic oral manifestations.

- **Angular cheilosis** — fissures and crusting at the corners of the mouth
- **Glossitis** — shiny, red, sore tongue; depapillation
- **Mucositis & oral ulceration** — painful erosions of the mucosa
- **Aphthous ulcers** — recurrent, painful oral ulcerations
- **Inflamed gingiva** and increased risk of **candidiasis**

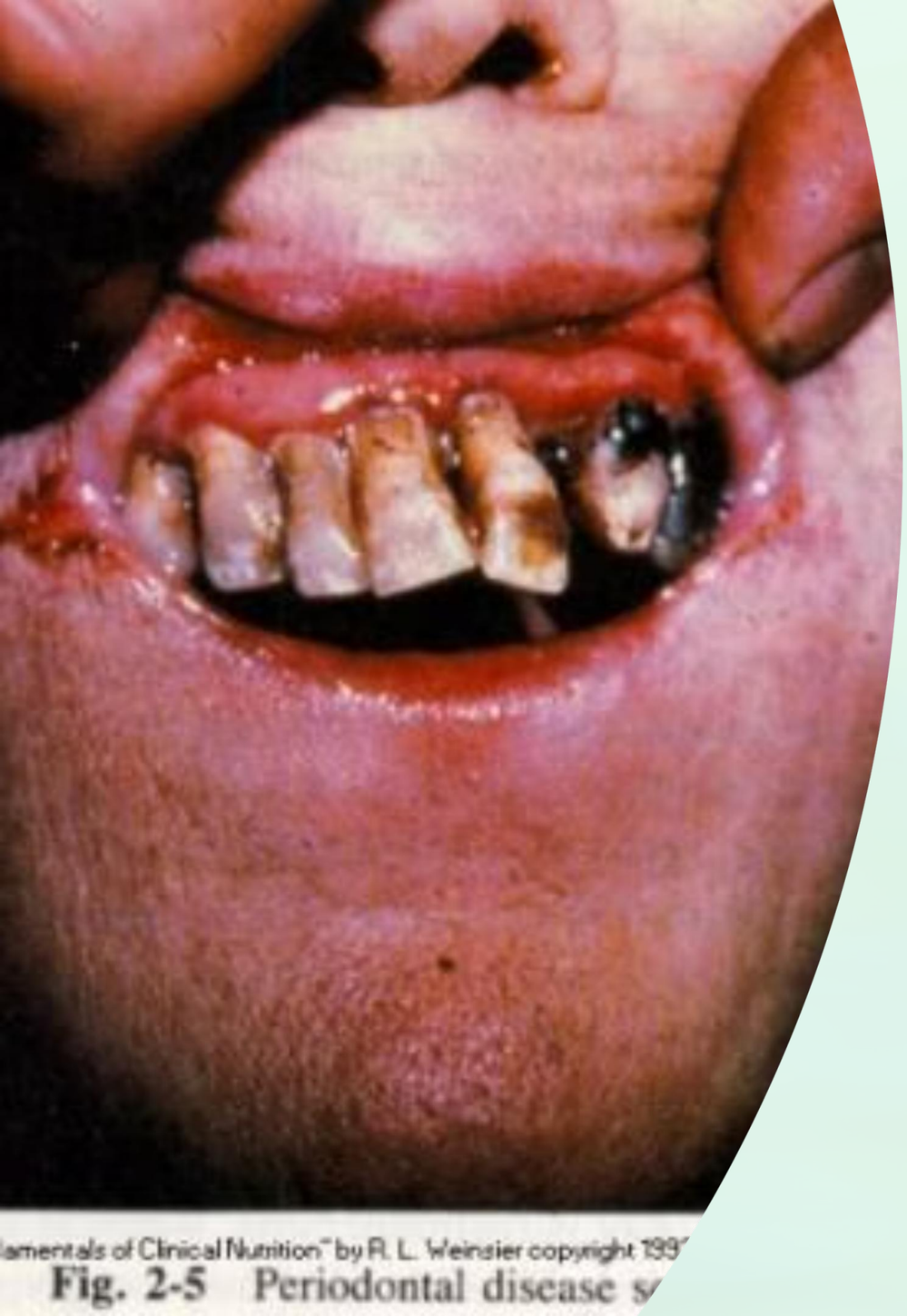


Fig. 2-5 Periodontal disease s

Avitaminoses — Vitamin C Deficiency (Scurvy)

Vitamin C (ascorbic acid) is indispensable for collagen synthesis and connective tissue repair. Deficiency causes profound gingival and periodontal breakdown.

Gingival Changes

Red, swollen, spongy gingiva with marked friability and spontaneous hemorrhage. Soft tissue ulceration may develop.

Periodontal Destruction

Progressive periodontal destruction leads to increased tooth mobility and ultimately tooth exfoliation (tooth loss).

Dental & Mucosal Effects

Sore, burning mouth; malformed teeth due to inadequate dentine formation; increased susceptibility to candidiasis.

Avitaminoses — Vitamin A Deficiency

Vitamin A is critical for epithelial differentiation, immune function, and tooth development. Deficiency leads to widespread oral and dental abnormalities.

Mucosal & Gingival Changes

Desquamation of oral mucosa, keratosis, gingival hypertrophy and inflammation, and development of leukoplakia.

Salivary & Sensory Effects

Xerostomia (dry mouth), decreased taste sensitivity, and impaired wound healing and tissue regeneration.

Dental Effects

Disturbed enamel development, increased caries risk, and increased susceptibility to candidiasis.

Avitaminoses — Vitamin D: Deficiency & Excess

Vitamin D regulates calcium and phosphate metabolism, both critical for normal mineralization of teeth and bone. Both deficiency and excess produce distinct oral and dental pathologies.

Deficiency

Inadequate vitamin D impairs calcium absorption, resulting in **incomplete mineralization of teeth** (hypomineralization). This manifests as weakened, chalky enamel prone to rapid decay. In children, deficiency during tooth development causes **rickets-associated dental defects**.

Excess (Hypervitaminosis D)

Excessive vitamin D causes hypercalcemia and pathological calcium deposition. Oral manifestations include **pulp calcification** (dystrophic calcification within pulp chambers) and **enamel hypoplasia** — defective enamel formation resulting in pitting, grooves, or complete enamel absence.

Disorders of Mineral Metabolism

Mineral imbalances — whether from dietary insufficiency, malabsorption, or metabolic disorders — have significant oral and dental consequences.

Fluoride

Deficiency: decreased enamel resistance → increased caries risk.
Excess (fluorosis): enamel hypoplasia — white spots, brown staining, pitting.

Iron

Deficiency: angular cheilosis, pallor of lips and oral mucosa, sore burning tongue, glossitis (iron-deficiency anemia features).

Calcium

Deficiency: incomplete tooth mineralization, rickets, osteomalacia, osteoporosis — increased tooth mobility and premature loss.

Zinc

Deficiency: loss of taste/tongue sensation, delayed wound healing, increased susceptibility to periodontal disease, candidiasis, xerostomia, and caries.

Chapter 3

Salivary Gland Diseases

The salivary glands (parotid, submandibular, sublingual) are susceptible to a range of conditions — inflammatory, infectious, autoimmune, obstructive, and neoplastic.

Sialolithiasis

Formation of calcified stones within salivary gland ducts, causing obstruction, pain, and swelling — typically at mealtime when salivary flow increases.

Sialadenitis

Acute or chronic bacterial inflammation — caused by *Staphylococcus*, *Streptococcus*, *Haemophilus influenzae*. Often secondary to obstruction or dehydration.

Viral Infections

Mumps (paramyxovirus), influenza, Coxsackie viruses, and cytomegalovirus (CMV) can all cause parotitis and salivary gland enlargement.

Sjögren's Syndrome

Autoimmune destruction of salivary and lacrimal glands → severe dry mouth (xerostomia), keratoconjunctivitis sicca (dry eyes), and dryness of skin, nose, and vagina.

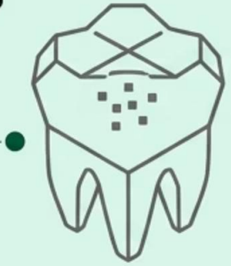
Cysts & Sialadenosis

Salivary cysts arise from injury, infection, stones, or tumors. Sialadenosis is painless, non-inflammatory parotid enlargement of unknown cause — often associated with metabolic disorders.

Comparative Overview: Oral Cavity Diseases at a Glance

The following schema summarizes the key oral cavity conditions covered, organized by primary cause, for rapid clinical reference.

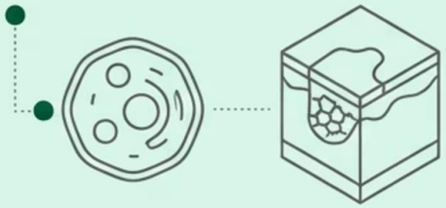
Infectious bacterial (Dental caries, Periodontitis, Sialadenitis)



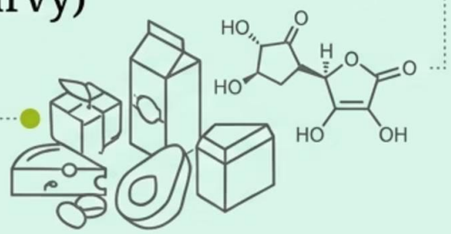
Infectious viral (Herpes labialis, Viral parotitis)

Infectious fungal (Oral candidiasis)

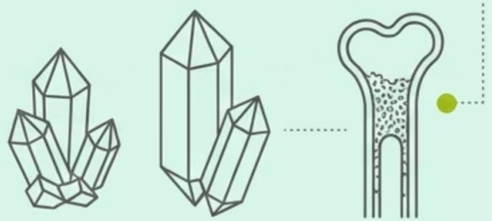
Precancerous and neoplastic (Leukoplakia, Squamous cell carcinoma, Adenocarcinoma, Lymphoma)



Nutritional/vitamin deficiency (Vitamin B deficiency, Vitamin C Scurvy)



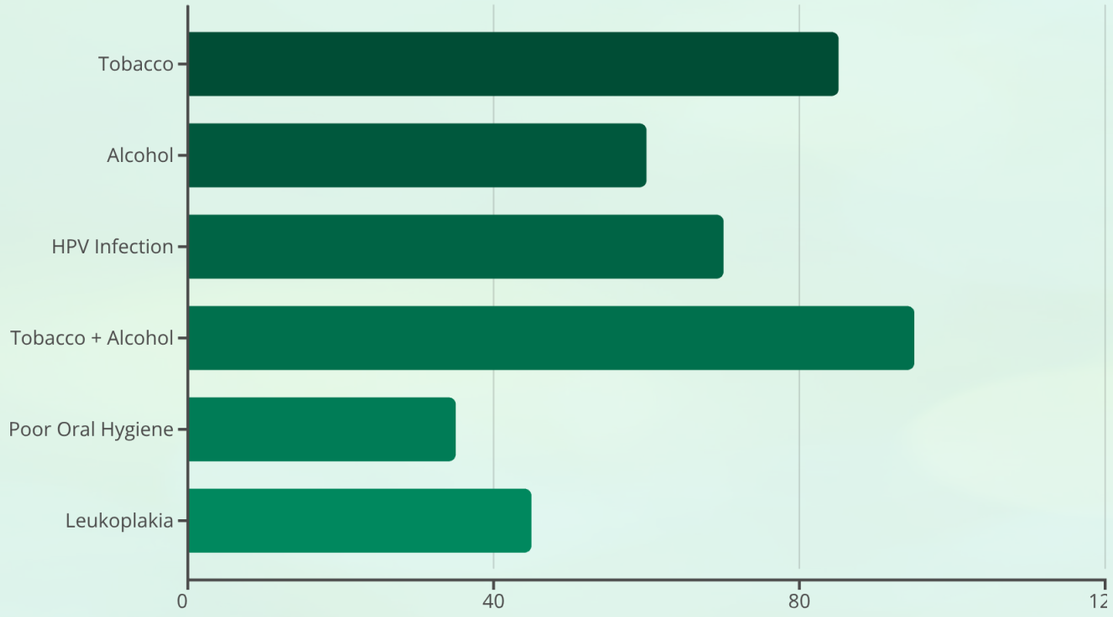
Mineral metabolism disorders (Fluoride, Iron, Calcium, Zinc deficiency)



This schema provides a structured view for differential diagnosis. Always consider systemic disease when multiple oral manifestations are present simultaneously.

Oral Cancer Risk Factors — Relative Impact

The following chart illustrates the relative contribution of major risk factors to oral cancer development, based on epidemiological data. Combined tobacco and alcohol use has a synergistic — not merely additive — effect on risk.



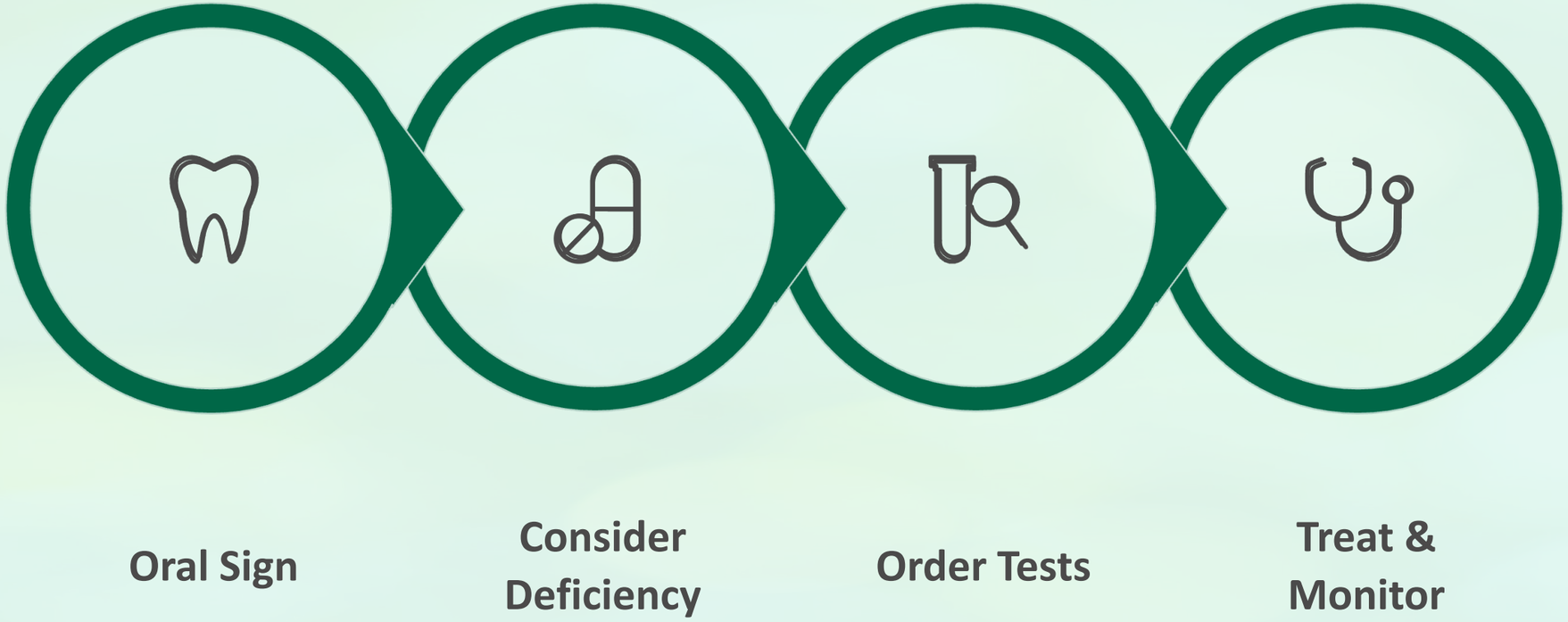
Key Insights

Tobacco and alcohol together represent the strongest modifiable risk factors for oral squamous cell carcinoma. HPV-16 is an increasingly recognized independent driver, especially in oropharyngeal cancer in younger patients. Precancerous lesions (leukoplakia, erythroplakia) elevate risk substantially and require active surveillance.

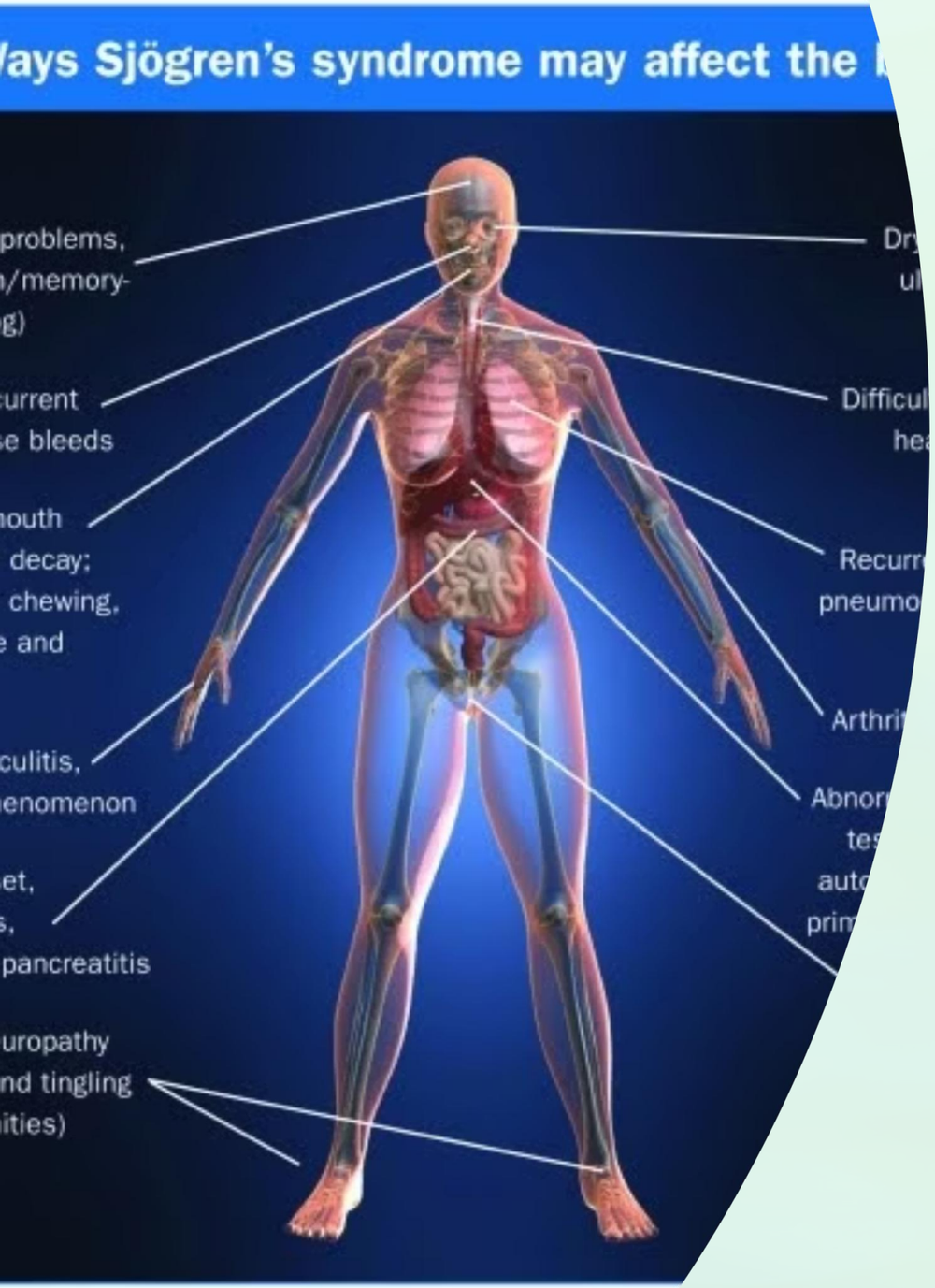
⚠ Synergistic effect of tobacco + alcohol increases oral cancer risk by up to 30× compared to non-users.

Vitamin & Mineral Deficiencies — Oral Manifestation Profile

This diagram shows the diagnostic pathway from presenting oral signs to suspected nutritional deficiency — a critical clinical reasoning tool.



Oral manifestations of nutritional deficiencies are often the **first clinically visible signs**, appearing before systemic symptoms. Early recognition by dental and medical practitioners can prompt timely nutritional intervention and prevent further systemic deterioration.



Salivary Gland Anatomy & Disease Disease Summary

The three paired major salivary glands — parotid, submandibular, and sublingual — each have distinct anatomical locations, duct systems, and disease predispositions.

Parotid Gland

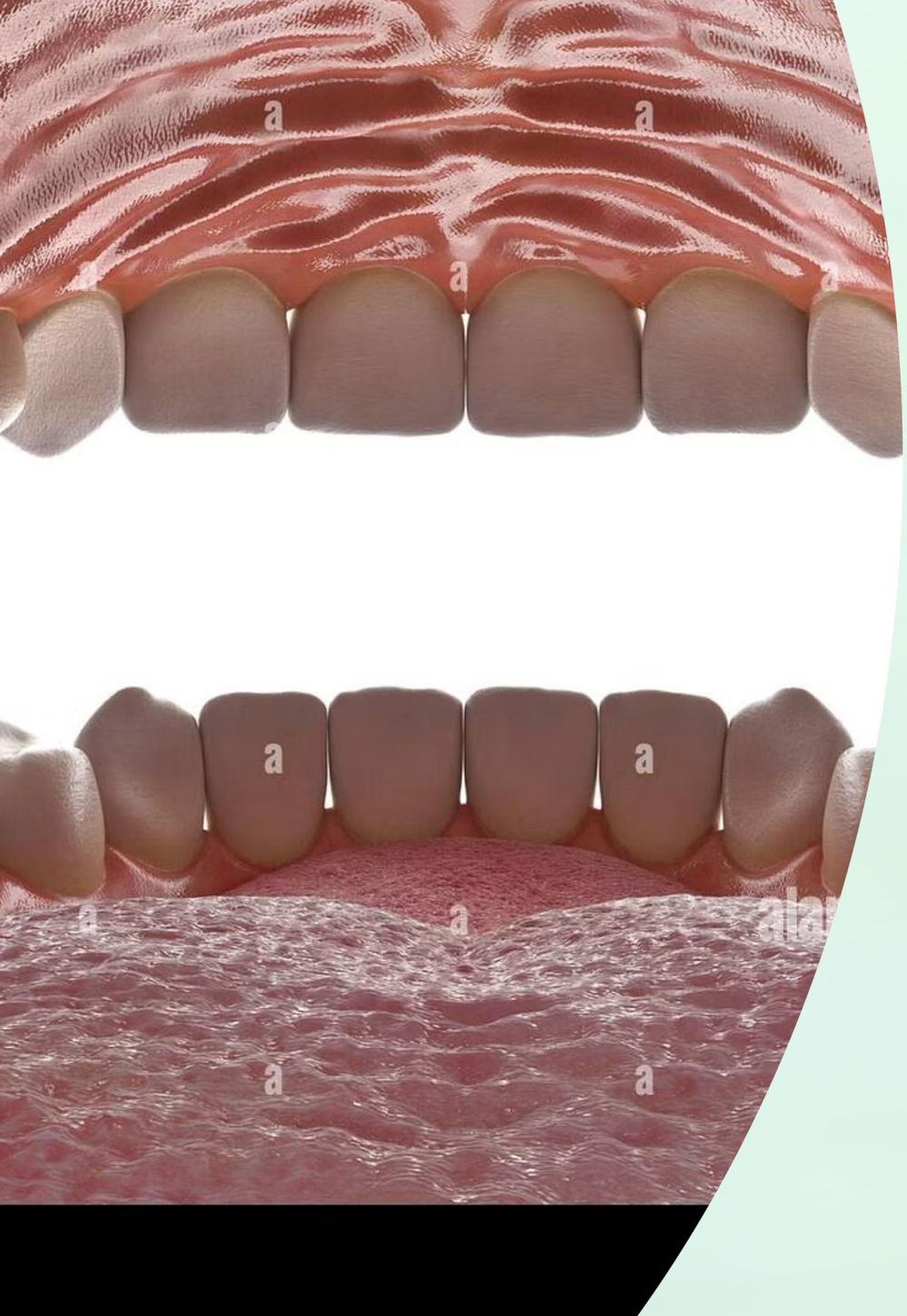
Largest salivary gland. Drained by Stensen's duct. Most commonly affected by mumps, sialadenosis, and parotid tumors (pleomorphic adenoma, Warthin tumor).

Submandibular Gland

Drained by Wharton's duct. Most commonly affected by **sialolithiasis** — due to duct anatomy (upward flow against gravity). Site of frequent sialadenitis.

Sublingual Gland

Smallest of the three major glands. Most commonly affected by mucous retention cysts (**ranulas**). Less frequently affected by stones than submandibular.



Oral Manifestations of Systemic Diseases

The oral cavity serves as a mirror of the body's overall health. Systemic diseases frequently produce recognizable signs and symptoms in the mouth — often preceding or paralleling the course of the underlying condition. This presentation provides a structured clinical reference covering gastrointestinal, hematologic, connective-tissue, pulmonary, cutaneous, endocrine, and renal diseases and their characteristic oral presentations.

ORAL MEDICINE

CLINICAL REFERENCE

CHAPTER 1

Gastrointestinal Diseases

Inflammatory bowel diseases — particularly Crohn's disease and ulcerative colitis — are among the most clinically significant GI disorders with well-documented oral manifestations. Recognizing these signs aids in early diagnosis and monitoring of disease activity.

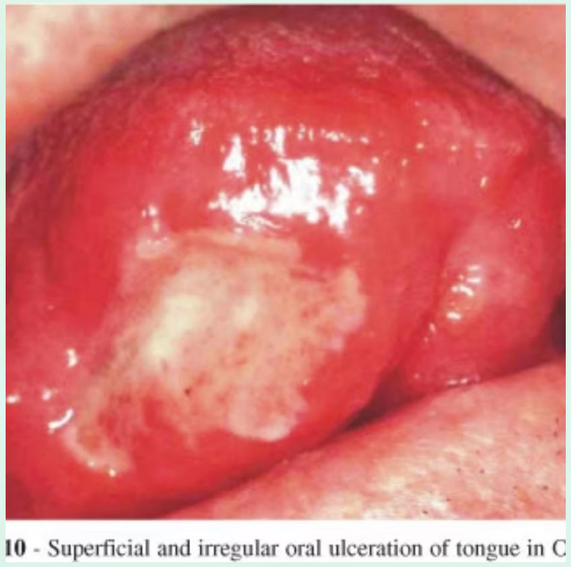
Crohn's Disease

- Diffuse labial, gingival, or mucosal swelling
- "Cobblestoning" of buccal mucosa and gingiva
- Aphthous ulcers — often large and persistent
- Mucosal tags and linear fissures
- Angular cheilitis
- Oral granulomas (orofacial granulomatosis)

Ulcerative Colitis

Oral signs typically emerge during **periods of disease exacerbation** and may parallel bowel activity:

- Aphthous ulceration or superficial hemorrhagic ulcers
- Angular stomatitis
- Pyostomatitis vegetans — pathognomonic pustular lesions
- Pyostomatitis gangrenosum in severe cases



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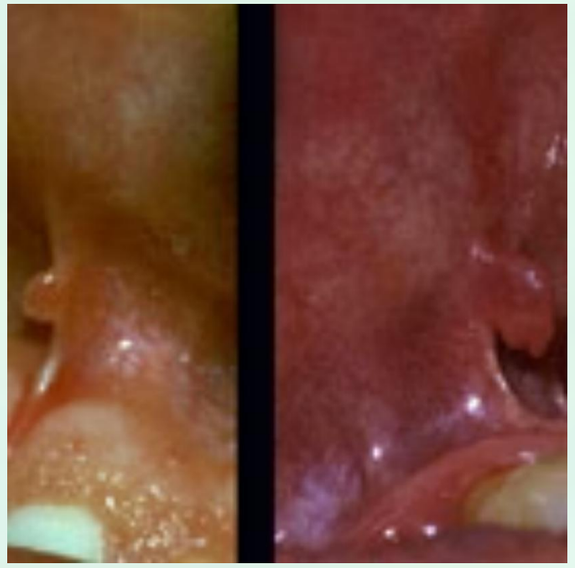


Left to right: labial swelling in Crohn's; tongue ulceration (OFG); cobblestoning of buccal mucosa; pyostomatitis vegetans of the gingiva.

CHAPTER 1

GI Diseases: Reflux & Chronic Liver Disease

Gastroesophageal Reflux Disease (GERD)

Chronic acid reflux reduces intraoral pH below **5.5** — the critical threshold for enamel dissolution. Repeated acid exposure causes:

- Erosion of enamel, predominantly on the palatal surfaces of upper anteriors
- Dentin exposure → hypersensitivity to temperature and chemical stimuli
- Increased susceptibility to dental caries
- Erosive tooth surface loss that may mimic bruxism

Severe erosion of enamel and dentin secondary to chronic gastroesophageal reflux.

Chronic Liver Diseases

Hepatic dysfunction impairs synthesis of clotting factors and bilirubin metabolism, producing characteristic oral findings:

- **Jaundice** — icteric discoloration of oral mucosa and sclera
- **Petechiae** — pinpoint hemorrhages on the palate or mucosa
- **Gingival bleeding** — due to coagulopathy (reduced prothrombin, thrombocytopenia)
- Spider telangiectasias on the lips in cirrhosis

CHAPTER 2

Hematologic Diseases: Anemias

Deficiency anemias — particularly **iron deficiency**, **folate deficiency**, and **vitamin B12 deficiency** (pernicious anemia) — produce a consistent constellation of oral signs driven by epithelial atrophy and impaired mucosal immune defense.

Key Oral Manifestations

- **Glossitis** — erythema, atrophy of filiform and fungiform papillae ("bald tongue")
- Recurrent aphthous ulcers
- Angular stomatitis / angular cheilitis
- Candidal infections — due to mucosal barrier compromise
- Oral pain and burning sensation
- Pallor of the oral mucosa reflecting systemic anemia

Angular cheilitis; atrophic glossitis; oral candidiasis in iron-deficiency anemia.

CHAPTER 2

Hematologic Diseases: Leukemia

Leukemia — particularly **acute myeloid leukemia (AML)** — produces characteristic oral manifestations through leukemic infiltration of gingival tissues, thrombocytopenia-related bleeding, and therapy-induced immunosuppression.

Primary Oral Signs of Leukemia

- **Gingival hypertrophy** — diffuse, spongy enlargement due to leukemic cell infiltration; most pronounced in AML (M4/M5)
- **Petechiae** — palate and buccal mucosa; hallmark of thrombocytopenia
- **Mucosal ulcers** — painless or painful, slow to heal
- **Spontaneous gingival hemorrhage** — disproportionate to plaque levels

Treatment-Related Oral Complications

Chemotherapy and bone marrow transplantation significantly compromise oral mucosal integrity:

- **Oral mucositis** — diffuse painful ulceration of non-keratinized mucosa
- **Reactivation of herpes simplex virus (HSV)** — vesicular lesions with secondary bacterial superinfection
- Opportunistic fungal infections (Candida, Aspergillus)
- Graft-versus-host disease (GVHD) — lichenoid oral lesions post-transplant

SUMMARY TABLE

Oral Manifestations: GI & Hematologic Diseases — Part I

The following summary maps key oral findings to their associated systemic conditions, facilitating rapid differential diagnosis at the chairside.

Angular Cheilitis

- Iron-deficiency anemia
- Crohn's disease

Hemorrhage / Petechiae

- Pyostomatitis vegetans
- Ulcerative colitis
- Scurvy
- Leukemia

Aphthous Ulcers

- Crohn's disease
- Pernicious anemia
- Ulcerative colitis

Intraoral Burning

- Iron-deficiency anemia
- Pernicious anemia

Candidiasis

- Crohn's disease (steroid therapy)
- Iron-deficiency anemia
- Pyostomatitis vegetans (steroid therapy)
- Ulcerative colitis (steroid therapy)

SUMMARY TABLE

Oral Manifestations: GI & Hematologic Diseases — Part II

Labial Swelling

- Crohn's disease (orofacial granulomatosis)
- Cystic fibrosis

Enamel / Dentin Erosion

- Anorexia nervosa / bulimia nervosa
- Gastroesophageal reflux disease

Gingivitis

- Anorexia nervosa / bulimia
- Crohn's disease
- Scurvy (vitamin C deficiency)
- Diabetes mellitus

Glossitis

- Crohn's disease
- Iron-deficiency anemia
- Pernicious anemia
- Ulcerative colitis

Ulcerations & Erosions

- Crohn's disease
- Iron-deficiency anemia
- Pernicious anemia
- Pyostomatitis vegetans
- Ulcerative colitis

This cross-reference table is a diagnostic aid. Always correlate oral findings with patient history, laboratory values, and systemic evaluation before reaching a diagnosis.

CHAPTER 3

Connective-Tissue Diseases: Sjögren Syndrome

Sjögren syndrome is a chronic autoimmune exocrinopathy predominantly affecting middle-aged women (M:F = 1:9). It targets exocrine glands — particularly salivary and lacrimal — through lymphocytic infiltration.

Hallmark Features (Sicca Syndrome)

- Keratoconjunctivitis sicca — dry, gritty, red eyes
- Xerostomia — subjective complaint of dry mouth

Oral Manifestations

- Dry, red, wrinkled, and atrophic oral mucosa
- Difficulty swallowing and eating (dysphagia)
- Disturbance in taste (dysgeusia) and speech
- Markedly increased dental caries — especially cervical caries
- Recurrent oral infections; candidiasis
- Atrophy of filiform papillae on the tongue
- Bilateral parotid gland enlargement

Keratoconjunctivitis sicca; dry atrophic oral mucosa; xerostomia-associated caries.

⚠ Secondary Sjögren syndrome may occur in the context of rheumatoid arthritis, SLE, or scleroderma. Screen all patients with unexplained xerostomia.

CHAPTER 3

Connective-Tissue Diseases: Kawasaki Disease

Kawasaki disease (mucocutaneous lymph node syndrome) is an acute, self-limiting vasculitis of medium and large arteries, predominantly affecting children under age 5. Oral signs are part of the classic diagnostic criteria and are often the presenting feature that brings the child to clinical attention.

Diagnostic Oral Signs

- Strawberry tongue — diffuse erythema with prominent, swollen papillae, resembling a strawberry surface
- Intense erythema of oral and pharyngeal mucosal surfaces
- Cracked, cherry-red, swollen, and hemorrhagic lips — pathognomonic
- Cervical lymphadenopathy often accompanies oral changes

⊗ Coronary artery aneurysm is the most serious complication. Early diagnosis via oral signs can be life-saving.

Child with Kawasaki disease
(red eyes, dry cracked lips, red tongue)

Strawberry tongue; hemorrhagic, cracked lips; oropharyngeal erythema in Kawasaki disease.

CHAPTER 3

Connective-Tissue Diseases: Scleroderma

Systemic sclerosis (scleroderma) is a chronic autoimmune disease characterized by diffuse fibrosis of the skin, gastrointestinal tract, heart, lungs, and kidneys. Oral manifestations are prominent and may significantly impact quality of life and dental management.

Oral & Perioral Signs

- Microstomia — pursed, restricted lips; severe limitation of mouth opening
- Esophageal sclerosis → GERD → enamel erosion (palatal surfaces)
- Pale, rigid, and bound-down oral mucosa
- Telangiectasias — dilated capillaries on lips and buccal mucosa
- Decreased tongue mobility — fibrosis of floor of mouth
- Salivary hypofunction → xerostomia
- Gingival retraction — widened periodontal ligament space on radiograph

Limited mouth opening (microstomia) and gingival retraction in systemic sclerosis.

⚠ Dental treatment planning must account for severely restricted mouth opening. Consider custom prosthetics and high-frequency preventive care.

CHAPTER 3

Connective-Tissue Diseases: Lupus Erythematosus

Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease with a broad spectrum of oral manifestations. Oral lesions may occur during flares or persist independently and can mimic other mucosal diseases, complicating diagnosis.

Oral Signs

- **Ulcerations** — painless or painful, typically on the hard palate
- **Lichen planus–like lesions** — white radiating striae on buccal mucosa; often painful
- **Petechiae** — related to thrombocytopenia
- **Salivary gland damage → xerostomia** — secondary Sjögren overlap
- Discoid LE may produce erythematous plaques with central atrophy on labial mucosa

Palatal ulceration; mucosal ulcer; lichen planus–like lesions in SLE.

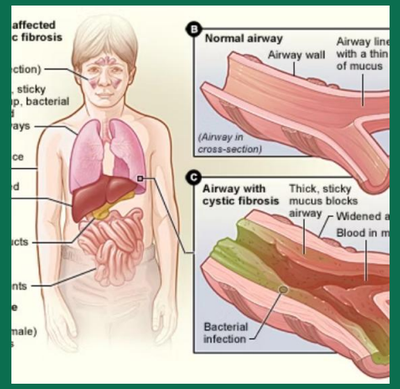
CHAPTER 4

Pulmonary Diseases: Cystic Fibrosis & Sarcoidosis

Cystic Fibrosis

A hereditary multisystem disorder caused by CFTR gene mutations, affecting exocrine gland secretions throughout the body, including salivary glands.

- Salivary gland dysfunction — abnormal electrolyte composition of saliva
- Lip swelling and angular cheilitis
- Gingivitis — related to altered saliva and mouth breathing
- Oral dryness and increased caries risk



Sarcoidosis

A systemic granulomatous disease of unknown etiology, most commonly affecting the lungs and lymph nodes, with variable oral involvement.

- Multiple nodular, painless ulcerations of gingiva, buccal mucosa, labial mucosa, and palate
- Tumor-like swelling of salivary glands — particularly parotid (Heerfordt syndrome)
- Tongue swelling and macroglossia
- Xerostomia secondary to salivary gland infiltration
- Facial nerve palsy (CN VII) — may be the presenting sign

CHAPTER 5

Cutaneous Diseases: Psoriasis & Acanthosis Nigricans

Psoriasis

A chronic immune-mediated inflammatory skin disease. Oral involvement is less common than cutaneous disease but is increasingly recognized.

- Fissured tongue (lingua plicata) — most frequent oral finding
- Small white papules on the buccal mucosa
- Red and white plaques resembling geographic tongue
- Bright red, well-demarcated erythematous patches

Acanthosis Nigricans

A dermatosis characterized by velvety hyperpigmentation and papillomatosis, strongly associated with insulin resistance, obesity, and internal malignancy.

- Gingival hyperplasia with papillomatous texture
- Papillomas of gingiva, tongue, and lips
- Mucosal hyperpigmentation

Oral acanthosis nigricans may be a paraneoplastic sign — particularly associated with gastric adenocarcinoma. Urgent workup is warranted.

Fissured tongue in psoriasis (left); gingival papillomatosis in acanthosis nigricans (right).

CHAPTER 6

Endocrine Diseases: Diabetes Mellitus

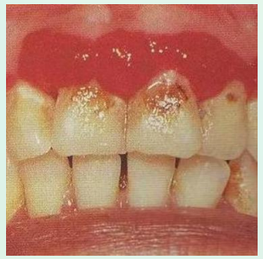
Diabetes mellitus has a well-established bidirectional relationship with oral disease. Hyperglycemia impairs immune function, alters salivary composition, and promotes microvascular changes — all of which profoundly affect the oral environment.

Xerostomia
Decreased salivation combined with elevated glucose in saliva creates a permissive environment for candidiasis, caries, and gingival inflammation.

Periodontal Disease
Severe, rapidly progressing gingivitis and periodontitis. Considered the sixth complication of diabetes; bidirectional link with glycemic control.

Parotid Enlargement
Bilateral, non-tender enlargement of parotid glands (sialosis) — related to fatty infiltration and metabolic dysfunction.

Burning Mouth / Altered Taste
Dysgeusia and burning mouth syndrome are reported in a significant proportion of diabetic patients, linked to peripheral neuropathy.



Diabetic hyperplastic gingivitis; bilateral parotid gland enlargement (sialosis) in diabetes mellitus.

CHAPTER 6

Endocrine Diseases: Parathyroid Disorders

Hypoparathyroidism

Deficient PTH secretion leads to hypocalcemia, which may manifest systemically as tetany and neuromuscular irritability. Oral signs include:

- Oral candidiasis — chronic mucocutaneous form in association with autoimmune hypoparathyroidism (APECED syndrome)
- Upper lip twitching — related to hypocalcemic neuromuscular excitability (Chvostek's sign)
- Dental hypoplasia and enamel defects if onset during tooth development

Hyperparathyroidism

Excess PTH stimulates osteoclastic bone resorption, producing characteristic skeletal and dental radiographic changes:

- Loss of lamina dura — the dense radiopaque line surrounding tooth roots disappears; a hallmark radiographic finding
- Decreased trabecular bone density — ground-glass appearance of jaw bones
- Brown tumors (giant cell lesions) — expansile radiolucent lesions of the mandible or maxilla; may mimic odontogenic cysts
- Pathological jaw fractures in severe cases

Loss of lamina dura on dental radiograph is a classic early sign of hyperparathyroidism — always consider biochemical workup (calcium, PTH levels).

CHAPTER 6

Endocrine Diseases: Cushing's Syndrome & Addison's Disease

Cushing's Syndrome

Chronic hypercortisolism — from exogenous steroids or endogenous overproduction — leads to redistribution of adipose tissue and immunosuppression.

- "Moon face" — fatty tissue deposition in the cheeks, obscuring facial angles
- Osteoporosis → pathological fractures of mandible, maxilla, and alveolar bone
- Delayed healing of fractures and soft tissue wounds
- Increased susceptibility to oral infections

Addison's Disease

Primary adrenocortical insufficiency causes deficiency of cortisol and aldosterone, with compensatory elevation of ACTH and MSH — producing characteristic pigmentary changes.

- "Bronzing" — diffuse hyperpigmentation of skin, particularly in sun-exposed areas and pressure points
- Oral mucosal melanosis — irregular, macular brown-black pigmentation of buccal mucosa, tongue, and gingiva
- Pigmentation may precede systemic diagnosis

Oral mucosal hyperpigmentation (melanosis) in Addison's disease.

CHAPTER 7

Renal Diseases: Uremic Stomatitis

Uremic stomatitis is a rare but diagnostically significant condition occurring in undiagnosed or inadequately treated **chronic renal failure**. Mucosal injury results from chemical irritation by **ammonia and ammonium compounds**, produced when elevated urinary urea is hydrolyzed by salivary urease.

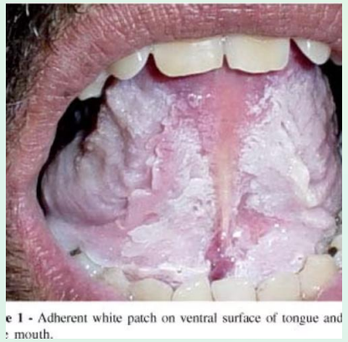
Type I — Erythematous Form

- Generalized or localized **erythema** of the buccal mucosa and tongue
- Pseudomembranous **exudate**
- Pain and burning sensation
- Xerostomia and halitosis (ammoniacal odor)
- Gingival bleeding
- Secondary candidiosis

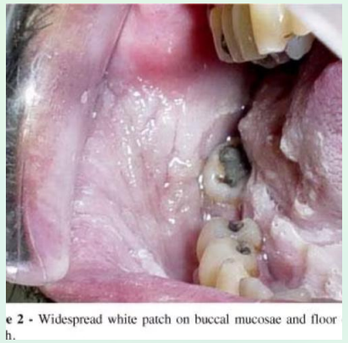
Type II — Ulcerative Form

- Painful **ulcerations** with necrotic base — buccal mucosa, floor of mouth, dorsum of tongue
- Painful crusts and plaques
- Susceptible to **secondary bacterial infection**
- Associated with systemic **anemia** of chronic kidney disease

⊗ The presence of uremic stomatitis indicates severe, often life-threatening renal failure. Immediate nephrology referral is mandatory.



e 1 - Adherent white patch on ventral surface of tongue and mouth.

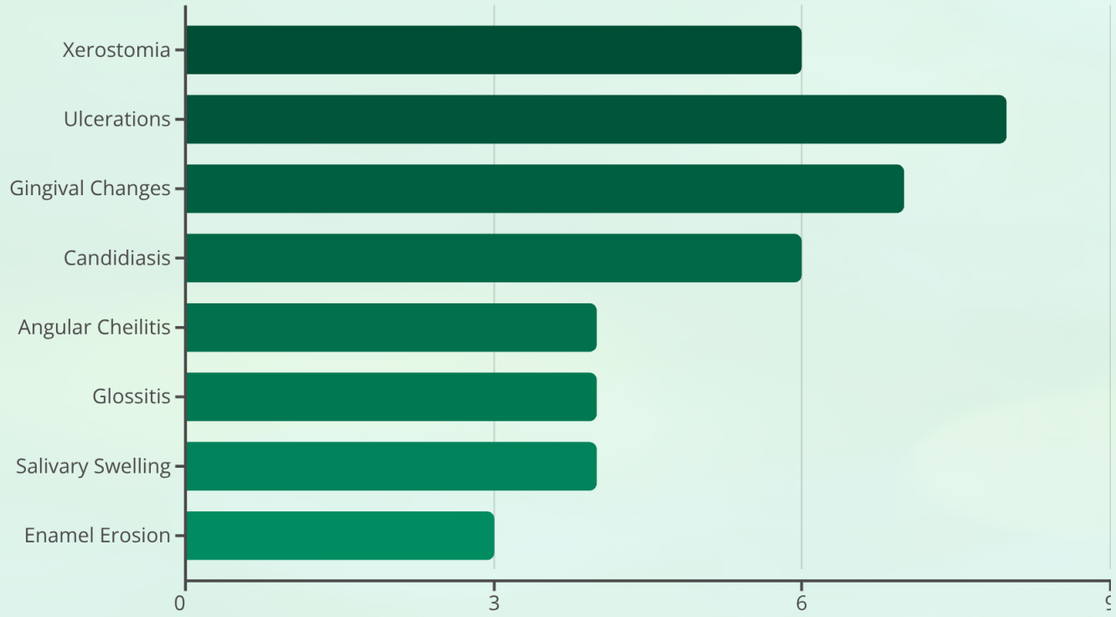


e 2 - Widespread white patch on buccal mucosae and floor h.

Type I (erythematous) and Type II (ulcerative) uremic stomatitis in chronic renal failure.

Systemic Diseases with Oral Manifestations: Overview Chart

The following chart illustrates the relative frequency of key oral manifestations across the major systemic disease categories covered in this presentation. Xerostomia, ulceration, and gingival changes are the most cross-cutting findings.



Clinical Insight

Oral findings rarely occur in isolation. A single mucosal sign — such as xerostomia or unexplained ulceration — may be the clinician's first opportunity to detect an underlying systemic condition. The mouth should be examined systematically at every appointment.

Most Cross-Cutting Sign

Xerostomia and ulcerations span the widest range of systemic diseases.

High-Yield Differentials Differentials

Gingival changes are diagnostic across GI, hematologic, endocrine, and renal conditions.

Cross-System Summary: Key Oral Signs by Disease Category

GI: CROHN'S,

UC, GERD: COBBLESTONING, APHTHAE, EROSION

HEMATOLOGIC: ANEMIA,

LEUKEMIA: GLOSSITIS, CHEILITIS, HYPERTROPHY

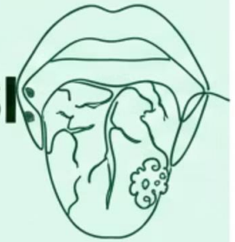
CONNECTIVE: SJÖGREN, SLE, SCLERODERMA:

XEROSTOMIA, MICROSTOMIA

PULMONARY: CF, SARCOIDOSIS:

SALIVARY DYSFUNCTION, ULCERS

CUTANEOUS: PSORIASIS, ACANTHOSI



FISSURED TONGUE, PAPILLOMAS

ENDOCRINE/RENAL: DIABETES, ADDISON'S, UREMIA: XEROSTOMIA, PIGMENTATION, PLAQUES

This matrix maps each organ system category to its most clinically significant oral findings, enabling rapid differential recognition at the chairside. When multiple signs co-occur, consider systemic workup beyond isolated dental management.

Key Clinical Takeaways

1 The Mouth as a Diagnostic Window

Oral signs frequently precede or parallel systemic disease. Systematic mucosal examination at every visit can enable early referral and improve patient outcomes.

2 Xerostomia Is Never Trivial

Dry mouth spans six or more systemic categories — from Sjögren syndrome to diabetes to renal failure. Always investigate the cause rather than managing symptoms alone.

3 Gingival Changes Demand a Differential

Unexplained gingival hypertrophy, hemorrhage, or recession in the absence of local irritants should prompt investigation for leukemia, diabetes, or connective-tissue disease.

4 Radiographs Are Diagnostic Tools

Loss of lamina dura (hyperparathyroidism), widened PDL space (scleroderma), and decreased trabecular density (Cushing's) are detectable on routine dental radiographs.

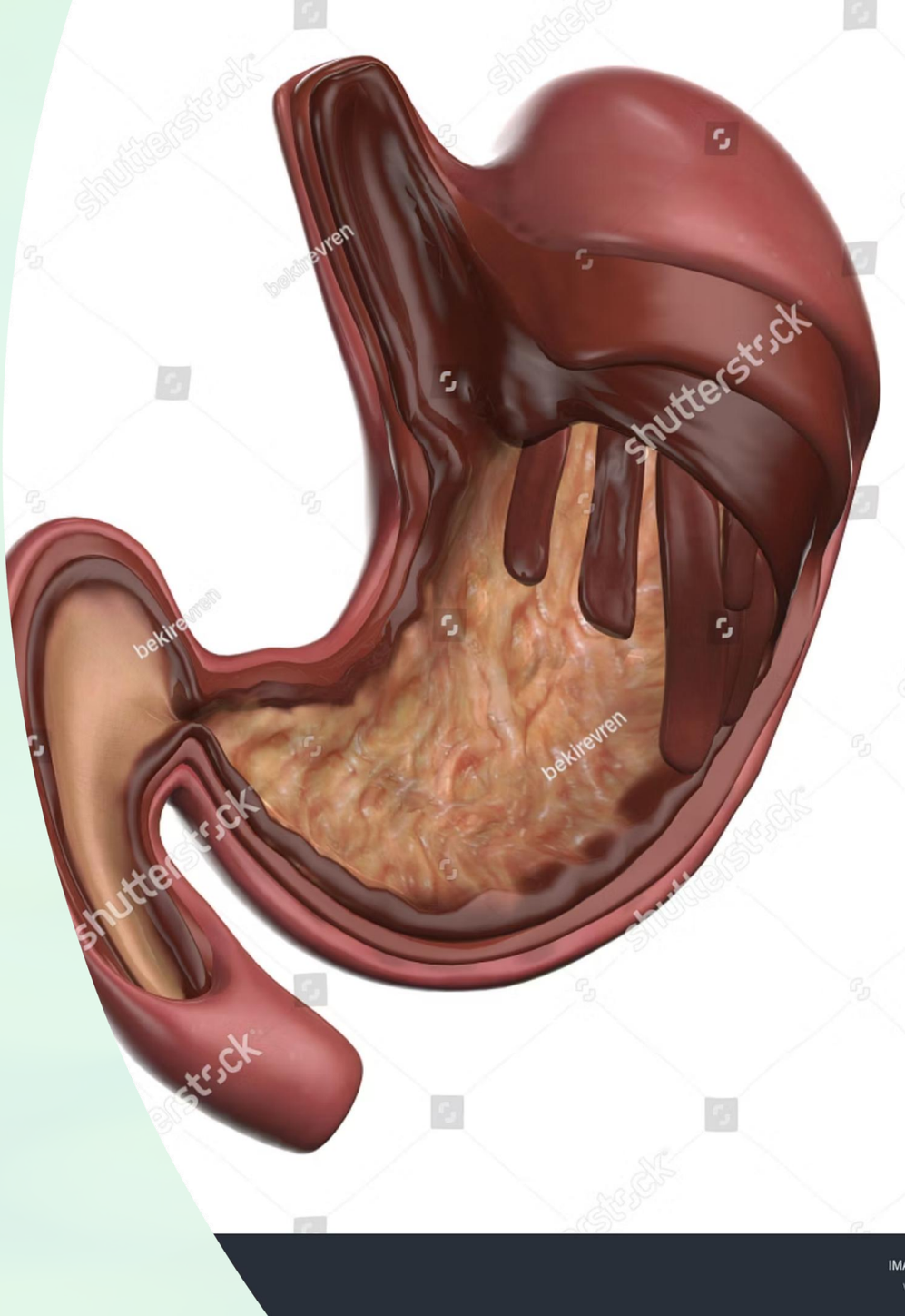
Always integrate oral findings with patient history, laboratory data, and multidisciplinary consultation. The dental clinician is an essential member of the systemic disease management team.

Peptic Ulcer Disease (PUD)

Pathophysiology — Gastroenterology

A comprehensive overview of peptic ulcer disease: etiology, pathogenesis, clinical presentation, complications, and diagnostic approach. Designed for medical students and junior clinicians in gastroenterology.

PATOFYZIOLÓGIA GASTROENTEROLÓGIA



Definition & Localization

Peptic ulcer disease (PUD) refers to a **disruption in the mucosal integrity** of the upper gastrointestinal tract, resulting in ulceration that extends through the mucosa into the submucosa or deeper layers. Ulcers may occur in the following locations:

Stomach

Gastric ulcers — typically located along the lesser curvature and antrum

Duodenum

Duodenal ulcers — most commonly in the proximal duodenum (bulb); most frequent type overall

Esophagus

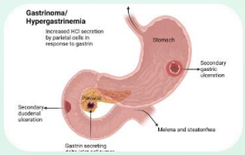
Esophageal ulcers — less common; typically associated with severe GERD or Barrett's esophagus

PUD affects approximately **10% of the global population** at some point in their lifetime, with duodenal ulcers being roughly 4 times more common than gastric ulcers.

Etiology — Primary Causes

H. pylori Infection

Accounts for **70–90%** of all peptic ulcers. The most common cause of chronic active gastritis and ulceration worldwide.



NSAIDs / Aspirin

Inhibit cyclooxygenase (COX), reducing prostaglandin synthesis and thereby impairing mucosal protection. Second most common cause.

Gastrinoma (ZES)

Zollinger-Ellison syndrome: gastrin-secreting tumors of the pancreas or duodenum cause massive acid hypersecretion and severe ulceration.

Stress Ulcers

Acute mucosal ulceration triggered by severe physiological stress — burns (Curling's ulcer), trauma, sepsis, or CNS injury (Cushing's ulcer).

Risk Factors

Modifiable Risk Factors

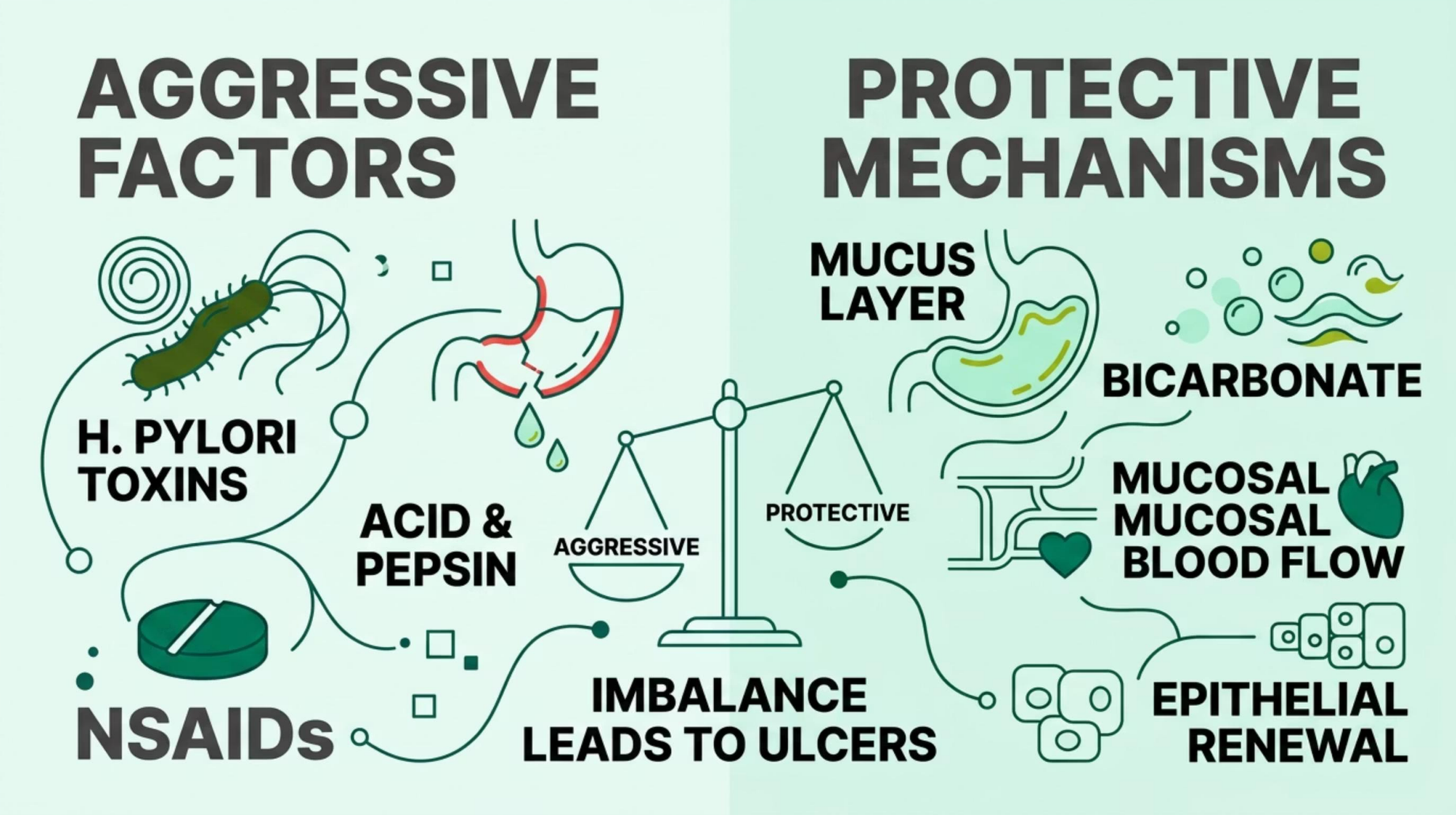
- **Smoking** — increases acid secretion, reduces prostaglandin and bicarbonate production, impairs mucosal healing
- **NSAIDs & Aspirin** — regular use significantly increases ulcer risk; highest risk in elderly patients
- **Coffee & Alcohol** — stimulate acid secretion; alcohol directly damages the gastric mucosa
- **Spicy diet** — may exacerbate symptoms in susceptible individuals

Non-Modifiable Risk Factors

- **Blood group O** — increased incidence of duodenal ulcers; O-type individuals lack certain mucosal protective antigens
- **HLA-B5 antigen** — genetic association with duodenal ulcer susceptibility
- **Positive family history** — particularly relevant for duodenal ulcers; suggests polygenic predisposition
- **Age & sex** — higher incidence in older males; postmenopausal women approach similar risk

Pathogenesis — The Core Concept

The fundamental mechanism of PUD is a **disrupted equilibrium between aggressive and protective factors** acting on the gastroduodenal mucosa. When aggressive forces overwhelm the defensive mechanisms, ulceration ensues.



Aggressive Factors in Detail

Hydrochloric Acid (HCl)

Secreted by **parietal cells** in the gastric corpus. Stimulated by **gastrin** (from G cells), **acetylcholine** (vagal nerve), and **histamine** (from enterochromaffin-like cells). HCl activates pepsinogen and creates the acidic environment necessary for pepsin activity.

Pepsin

A proteolytic enzyme produced by conversion of **pepsinogen** (secreted by chief cells). Active at low pH. Pepsin directly degrades the mucosal protein matrix, worsening epithelial damage initiated by acid.

H. pylori Virulence Factors

Produces **urease, proteases, phospholipases, and cytotoxins**. These directly damage epithelial cell membranes, trigger inflammatory cascades, and disrupt the mucus-bicarbonate barrier.

NSAIDs

Inhibit COX-1 and COX-2 enzymes, reducing prostaglandin synthesis. This impairs mucus and bicarbonate secretion and reduces mucosal blood flow — both systemic and via local topical injury to epithelium.

Protective Mechanisms

Mucus Layer

A viscous gel secreted by surface mucous cells creates a physical barrier that prevents direct contact between the epithelium and luminal acid/pepsin.

Prostaglandins

Stimulate mucus and bicarbonate secretion, regulate mucosal blood flow, and promote epithelial healing. NSAIDs reduce their synthesis — central to NSAID-induced ulcers.

Bicarbonate

Secreted beneath the mucus layer by surface epithelial cells. Neutralizes luminal acid before it reaches the epithelial surface, maintaining a near-neutral microenvironment at the cell surface.

Mucosal Blood Flow

Adequate perfusion delivers oxygen and nutrients, removes toxic metabolites, and supports rapid cell renewal. Ischemia — as in stress ulcers — impairs all protective mechanisms simultaneously.

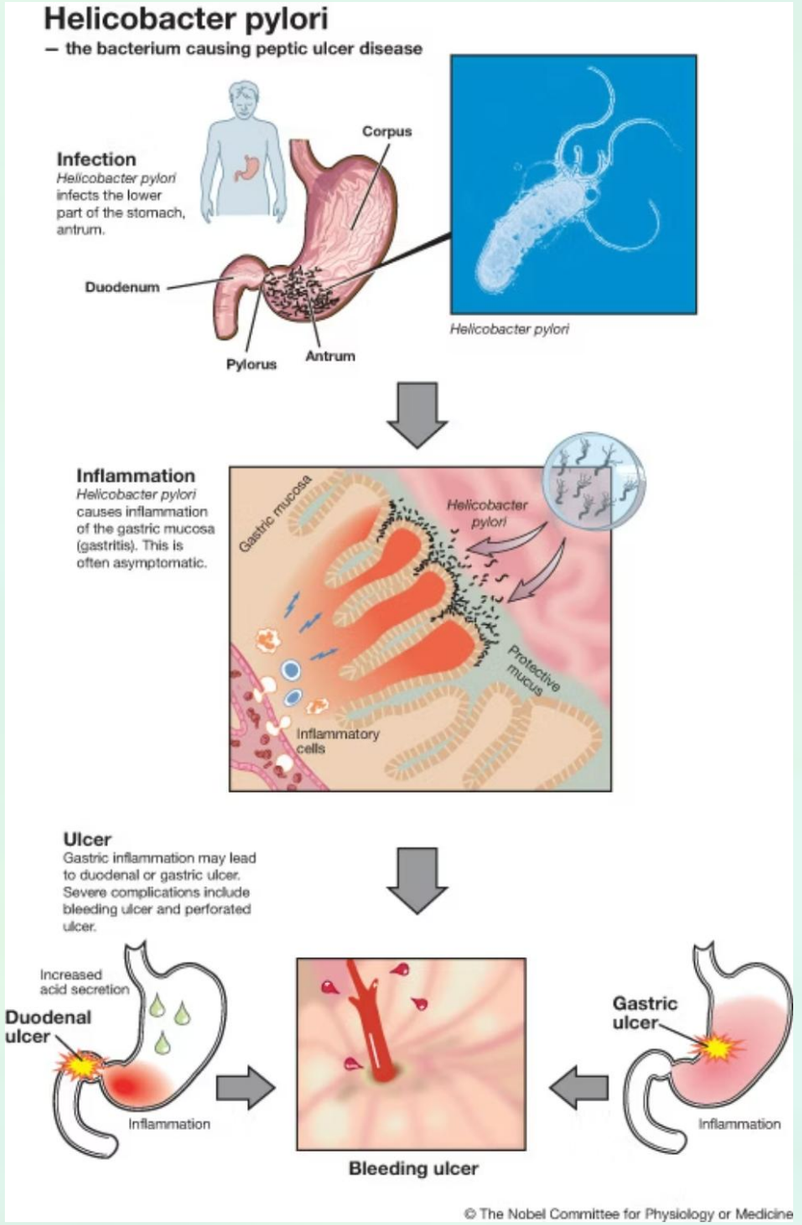
Helicobacter pylori — Microbiology & Pathogenicity

H. pylori is a **gram-negative, spiral-shaped, microaerophilic bacterium** that colonizes primarily the **gastric antrum**. Seroprevalence exceeds 60% in many populations, yet over 80% of infected individuals remain asymptomatic.

Key Virulence Mechanisms

- **Urease**: metabolizes urea → CO_2 + NH_3, alkalizing the local environment for bacterial survival. Ammonia is directly toxic to the epithelium.
- **Proteases & phospholipases**: degrade the mucus gel layer and damage cell membranes
- **Cytotoxins (CagA, VacA)**: induce vacuolation, apoptosis, and chronic inflammatory infiltration
- **Inflammation**: triggers neutrophil and mononuclear infiltration → chronic active gastritis → ulceration

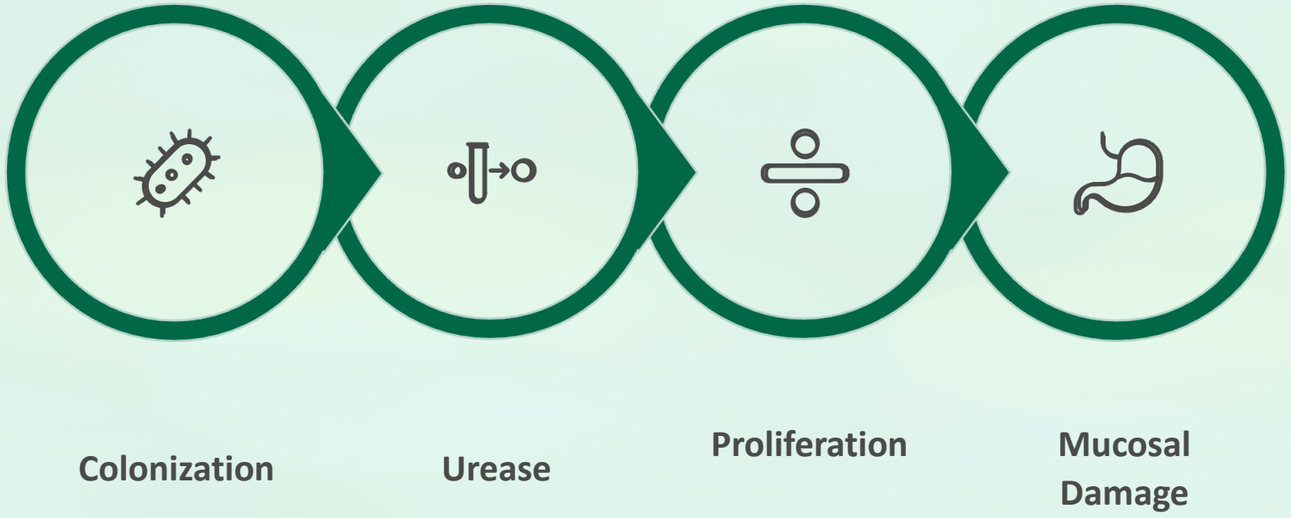
⚠ H. pylori causes **80–100% of duodenal ulcers** and **~80% of gastric ulcers**. It is also a major risk factor for gastric adenocarcinoma and MALT lymphoma.

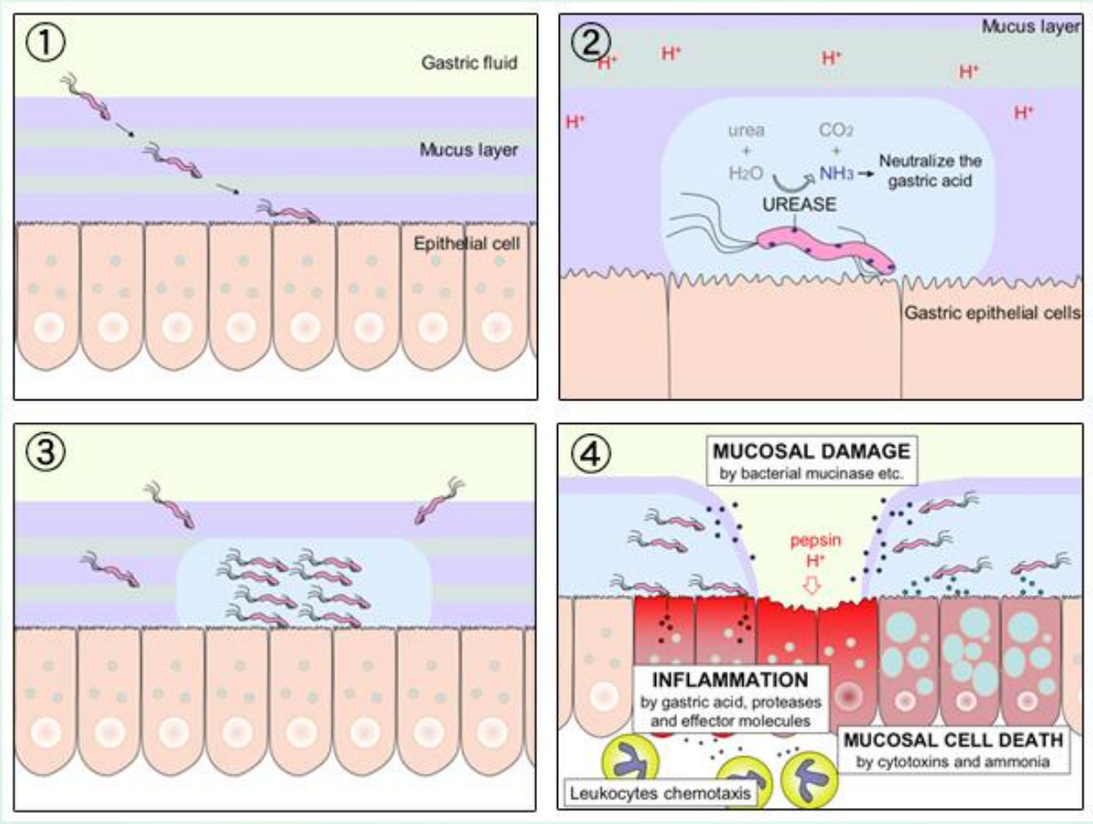


Source: Nobel Prize Committee for Physiology or Medicine — Diagram illustrating H. pylori infection, inflammation, and progression to ulceration.

H. pylori — Mechanism of Mucosal Damage

Source: Wikimedia Commons — Four-step diagram of H. pylori pathogenesis: (1) colonization beneath mucus, (2) urease-mediated acid neutralization, (3) bacterial proliferation, (4) mucosal damage by bacterial mucinase, pepsin, and inflammatory cells including leukocyte chemotaxis, leading to mucosal cell death via cytotoxins and ammonia.





Clinical Symptoms

Gastric Ulcer

- Diffuse epigastric pain — often described as burning or gnawing
- Pain exacerbates after eating — food stimulates acid secretion and directly contacts the ulcer
- Radiation to the back (suggests posterior penetration)
- Nausea, vomiting, anorexia
- Weight loss due to food avoidance

Duodenal Ulcer

- Localized epigastric pain — more circumscribed than gastric ulcer pain
- Pain relieved by eating — food buffers acid in the duodenum
- Nocturnal pain — classic feature; acid secretion peaks at night
- Bloating, fullness, belching
- Less weight loss than gastric ulcer

Shared & Alarm Symptoms

Dyspepsia

Bloating, fullness, salivation

Hematemesis

Vomiting of blood — suggests active bleeding

Melena

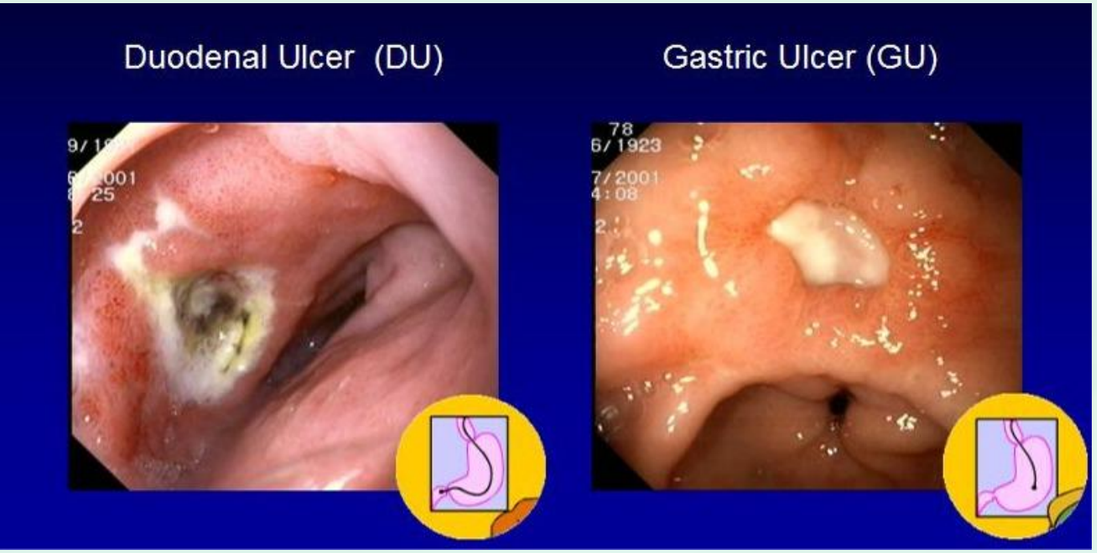
Dark, tarry stools — digested blood from upper GI bleed

Anemia Signs

Pallor, fatigue, tachycardia — from chronic or acute blood loss

Endoscopic Appearance

Source: Wikimedia Commons — Endoscopic images comparing duodenal ulcer (DU, left) and gastric ulcer (GU, right). Duodenal ulcer appears as a deep, punched-out lesion with fibrinous base in the duodenal bulb. Gastric ulcer is visible along the lesser curvature with raised, edematous margins and a clean white base.



Duodenal Ulcer (DU)

- Located in the duodenal bulb
- Deep, punched-out appearance with fibrinous exudate
- Surrounding mucosal erythema and edema
- Biopsy not mandatory — malignancy rare in duodenum

Gastric Ulcer (GU)

- Most commonly along the lesser curvature
- Raised, edematous margins with central crater
- **Biopsy mandatory** — must exclude malignancy
- Repeat endoscopy at 8–12 weeks to confirm healing

Diagnosis

History & Clinical Assessment

Detailed symptom history: character and timing of pain, relation to meals, use of NSAIDs, smoking, family history of PUD. Assess for alarm features: weight loss, dysphagia, hematemesis, melena, palpable mass.

Laboratory Tests

FBC (anemia from bleeding), serum gastrin (elevated in ZES), H. pylori serology (IgG), stool antigen test (sensitivity ~94%), urea breath test (UBT) — gold standard for non-invasive H. pylori detection.

Upper GI Endoscopy (Gastroscopy)

Gold standard for diagnosis. Allows direct visualization, biopsy (for gastric ulcers), and therapeutic intervention. Rapid urease test (CLO test) can confirm H. pylori at biopsy.

Barium Meal / Imaging

Upper GI barium series as alternative when endoscopy is unavailable — detects ulcer crater as a niche. CT abdomen is used if perforation is suspected (free air under diaphragm on erect CXR).

H. pylori Diagnostic Tests — Comparison

Test	Sensitivity	Specificity	Notes
Urea Breath Test (UBT)	~95%	~96%	Non-invasive; gold standard for confirmation and eradication check
Stool Antigen Test	~94%	~97%	Non-invasive; good for test-and-treat strategy in low-risk patients
Serology (IgG)	~85%	~79%	Cannot distinguish active vs past infection; not for post-treatment
Rapid Urease (CLO test)	~90%	~98%	Requires endoscopic biopsy; fast result in ~1 hour
Histology (biopsy)	~93%	~99%	Most accurate; also detects gastritis, metaplasia, and malignancy
Culture	~70–80%	~100%	Allows antibiotic sensitivity testing; technically demanding

PPIs, antibiotics, and bismuth must be stopped **2–4 weeks before UBT or stool antigen testing** to avoid false-negative results.

Complications of PUD

Bleeding

Most common complication. Erosion of submucosal vessels causes hematemesis or melena. Requires urgent endoscopy for diagnosis and hemostasis (clips, injection, coagulation).

Perforation

Full-thickness ulcer rupture leads to spillage of gastric contents → chemical peritonitis. Presents as sudden severe abdominal pain with board-like rigidity. Surgical emergency.

Penetration

Ulcer erodes into adjacent organs — most commonly the **pancreas** (causing pancreatitis) or **liver**. Pain radiates to the back; serum amylase/lipase may be elevated.

Gastric Outlet Obstruction

Scar tissue or inflammatory edema narrows the pylorus/duodenum → projectile vomiting of undigested food, metabolic alkalosis (hypochloremic, hypokalemic).

Malignant Transformation

Chronic H. pylori-associated gastric ulcers carry increased risk of **gastric adenocarcinoma**. All gastric ulcers must be biopsied and followed endoscopically to confirm healing.

Treatment Principles

H. pylori Eradication (Triple Therapy)

- PPI (e.g., omeprazole 20 mg) twice daily
- **Clarithromycin** 500 mg twice daily
- **Amoxicillin** 1 g twice daily *or* Metronidazole 500 mg twice daily
- Duration: **14 days** (preferred over 7-day course)
- Confirm eradication with UBT or stool antigen ≥4 weeks post-treatment

Additional Therapeutic Measures

- **PPIs**: mainstay of acid suppression — reduce acid secretion by blocking H^+/K^+-ATPase on parietal cells; continue 4–8 weeks
- **Discontinue NSAIDs** where possible; switch to COX-2 selective inhibitors with PPI cover if necessary
- **H_2 receptor antagonists**: second-line (ranitidine, famotidine)
- **Lifestyle modification**: cessation of smoking, alcohol reduction, dietary adjustments
- **Surgery**: reserved for complications (perforation, refractory bleeding, obstruction)

Successful H. pylori eradication achieves ulcer healing in **>90% of cases** and dramatically reduces recurrence rates from ~80% to <5% per year.

Summary — Key Takeaways

Etiology

H. pylori 70–90%, NSAIDs, ZES, stress

Pathogenesis

Imbalance: aggressive vs protective factors

Complications

Bleeding, perforation, penetration, obstruction, cancer

Diagnosis & Treatment Treatment

Endoscopy, H. pylori testing, PPI + eradication

Remember

Always biopsy gastric ulcers — never duodenal ulcers — to exclude malignancy.

Remember

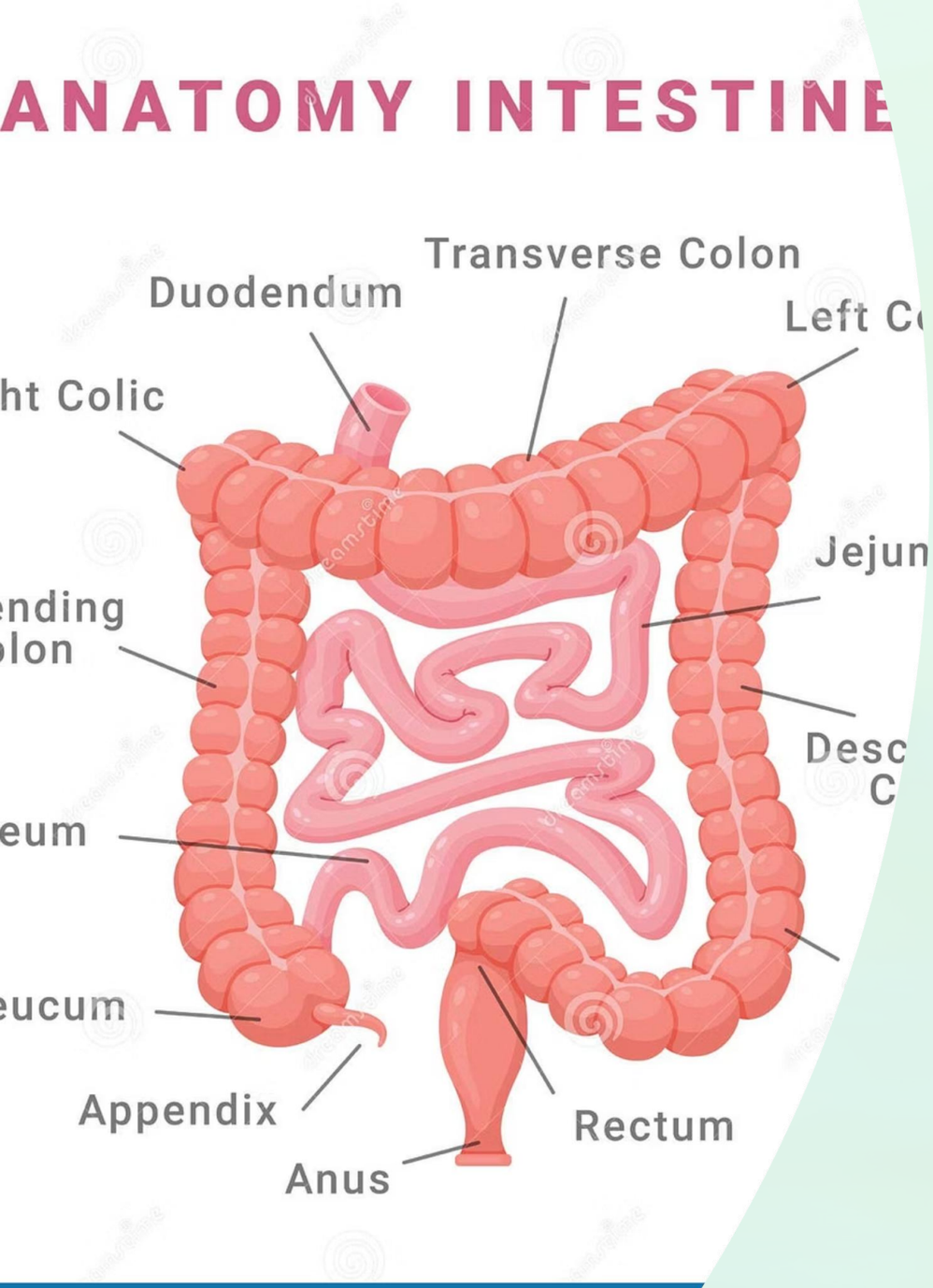
Test-and-treat H. pylori in uninvestigated dyspepsia in patients under 55 without alarm features.

Remember

Stop PPIs 2–4 weeks before UBT or stool antigen testing to avoid false negatives.

Remember

Sudden severe abdominal pain in a PUD patient = perforation until proven otherwise — urgent CT + surgical consult.



Ileus: Pathophysiology, Classification & Clinical Management

A comprehensive review of intestinal obstruction — covering etiology, classification, pathomechanism, symptomatology, complications, and diagnostic approach — for students of pathophysiology and junior clinicians.

PATHOPHYSIOLOGY | GENERAL SURGERY | GASTROENTEROLOGY

Lecture Overview

Topics Covered

01

Definition & Classification

Mechanical vs. paralytic ileus; subtypes and anatomical locations

02

Etiology & Pathomechanism

Causes of obstruction; cascade of physiological dysfunction

03

Symptomatology

Cardinal symptoms, clinical signs, and their pathophysiological basis

04

Special Forms

Intussusception, volvulus, and adhesions — presentation and management

05

Diagnosis & Complications

Imaging, laboratory findings, and life-threatening sequelae

Chapter 1 — Definition & Classification

What Is Ileus?

Ileus is defined as the failure of normal intestinal transit, resulting in the accumulation of intestinal contents proximal to the point of obstruction or functional failure. It represents a spectrum of conditions ranging from complete mechanical blockage to total inhibition of intestinal motility.

Mechanical Ileus

A physical barrier obstructs the intestinal lumen. Peristalsis is initially preserved or even increased proximal to the obstruction.

Paralytic (Adynamic) Ileus

No physical obstruction is present. Instead, neuromuscular dysfunction abolishes peristaltic activity throughout the gut.

Strangulation Ileus

A subtype of mechanical ileus in which vascular compromise accompanies obstruction, rapidly leading to bowel ischemia and necrosis.

Etiology of Ileus

Mechanical Causes

- Colorectal and small bowel tumors
- Inflammatory bowel disease (Crohn's disease)
- Fecal impaction
- Intestinal atresia (congenital)
- Gallstone ileus
- Intestinal adhesions (post-surgical)
- Intussusception
- Volvulus
- Incarcerated hernia

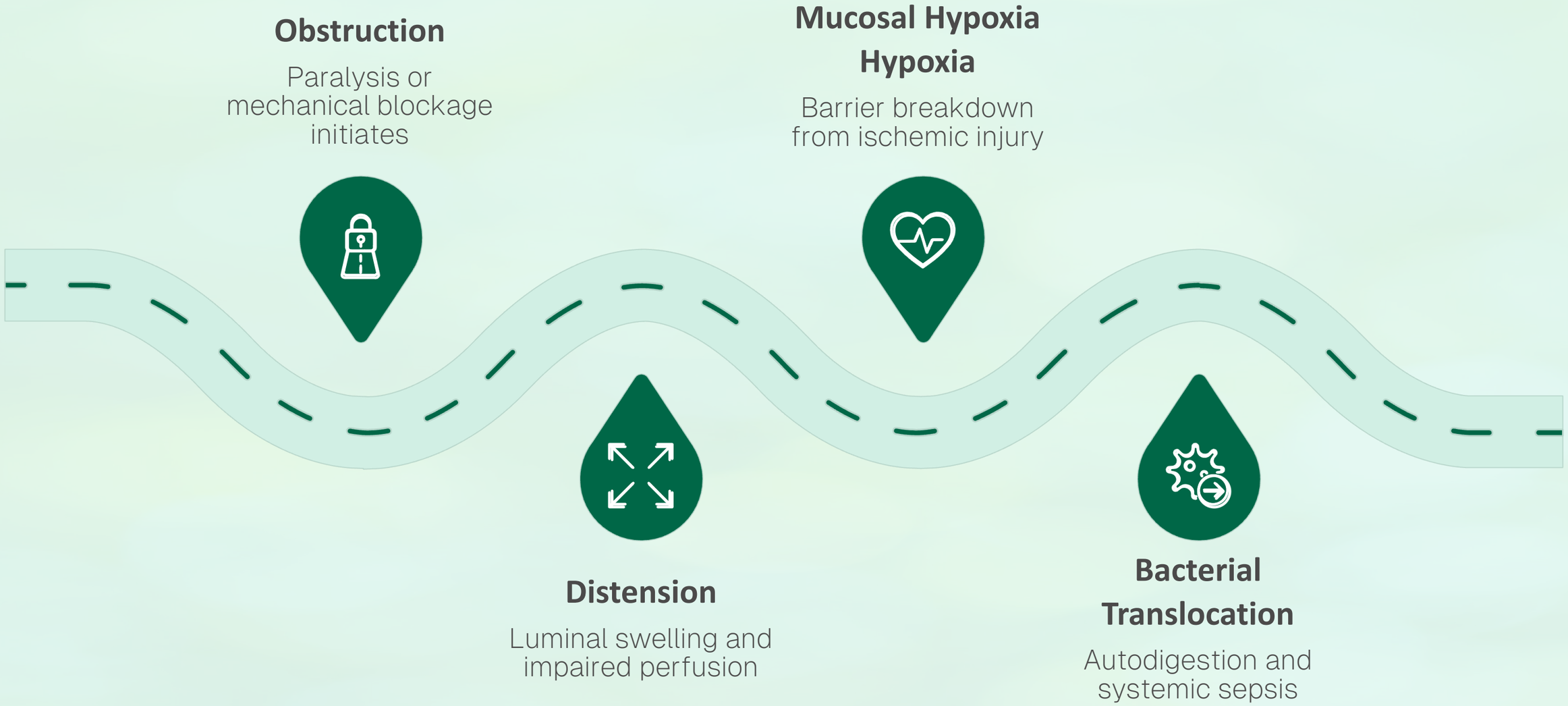
Functional / Paralytic Causes

- Post-surgical ileus (most common)
- Acute pancreatitis
- Peritonitis
- Intestinal myopathy or neuropathy
- Strangulation (hernia with vascular compromise)
- Metabolic disorders (hypokalemia, hypothyroidism, uremia)
- Opioid-induced bowel dysfunction

Pathomechanism

Cascade of Intestinal Dysfunction

Regardless of the primary cause, ileus disrupts all major intestinal functions — transport, secretion, absorption, digestion, and mucosal immunity — through a common downstream pathway of ischemia and autodigestion.



Bowel distension raises intraluminal pressure, compressing mucosal capillaries and triggering a cycle of ischemia → oxidative injury → increased permeability → translocation of gut bacteria into the systemic circulation. This cascade can rapidly progress to sepsis, multi-organ failure, and death if uncorrected.

Clinical Presentation

Symptomatology of Ileus

Abdominal Pain

Moderate, diffuse, cramping discomfort. In mechanical ileus, pain is often colicky and progressive. In paralytic ileus, pain may be dull or even absent.

Abdominal Distension

Gas and fluid accumulate proximal to the obstruction. Tympany on percussion is a hallmark finding. Distension can become severe enough to compromise respiration.

Nausea & Vomiting

Especially pronounced after meals. In proximal obstructions, vomiting occurs early and is bilious. In distal obstructions, it is delayed and may become feculent.

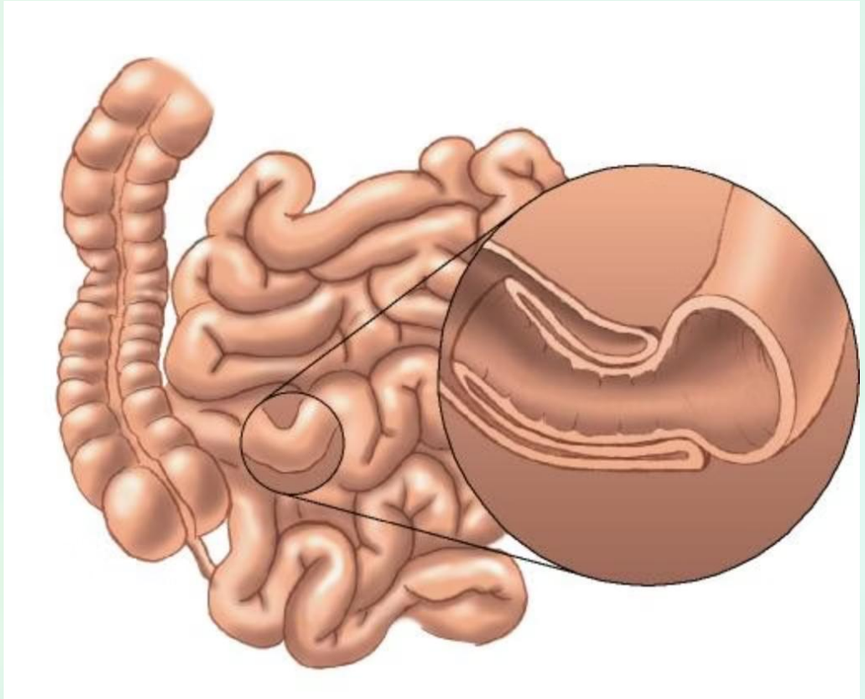
Obstipation

Absence of passage of stool and flatus is the cardinal sign of complete obstruction. Excessive belching may indicate small bowel involvement.

Intussusception: Definition & Epidemiology

Intussusception is the invagination of one bowel segment (the intussusceptum) into the adjacent distal segment (the intussuscipiens), creating a "bowel-within-bowel" configuration that obstructs both the lumen and the mesenteric blood supply.

- Location: 90% involve the ileocolic region
- Age: Most common in children 5 months–1 year; peak at 6–9 months
- Sex: Boys are affected twice as often as girls
- Etiology: Often idiopathic; associated with preceding viral/bacterial enteritis, lymphoid hyperplasia (Peyer's patches acting as lead points), intestinal parasites, foreign bodies, or tumors



In adults, intussusception accounts for only 1–5% of bowel obstructions and is more commonly associated with a pathological lead point such as a tumor.

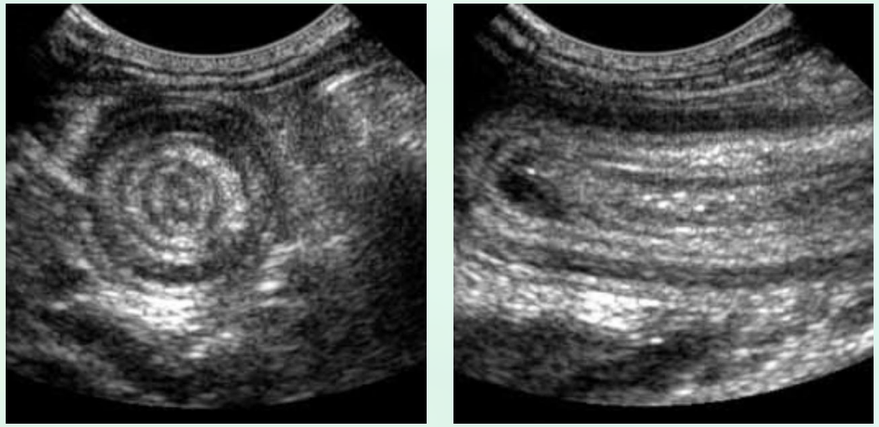
Intussusception

Pathomechanism & Clinical Features

As the intussusceptum telescopes inward, mesenteric vessels are compressed, leading to venous congestion, mucosal edema, and — if untreated — arterial ischemia and full-thickness necrosis. The classic clinical triad in infants is: sudden colicky abdominal pain + palpable sausage-shaped mass + currant-jelly (blood-mucus) stool.

Imaging: "Target Sign" on Ultrasound

Abdominal ultrasound is the diagnostic modality of choice, demonstrating concentric rings of bowel layers — the pathognomonic "target" or "donut" sign on transverse view, and "pseudokidney" sign on longitudinal view.



Ultrasound images showing the "target sign" (concentric rings) and longitudinal view of intussusception.

Intussusception

Intraoperative Findings

When pneumatic or hydrostatic enema reduction fails — or in cases with signs of peritonitis — surgical intervention is required. The intussusceptum may appear congested, edematous, or frankly necrotic intraoperatively. Manual reduction is attempted; non-viable bowel requires resection and primary anastomosis.

Intraoperative images demonstrating intussuscepted bowel with vascular congestion and early ischemic changes. Source: surgical case archives.

Special Forms — Volvulus

Volvulus: Definition & Classification

Volvulus is an abnormal axial rotation of a bowel segment around its mesenteric pedicle, producing a closed-loop obstruction. Unlike simple obstruction, volvulus simultaneously occludes both the afferent and efferent limbs, preventing decompression and accelerating ischemia.

Classification by Location

Neonatal Volvulus

Associated with intestinal malrotation

Small Bowel Volvulus

Often related to adhesions or congenital bands

Cecal Volvulus

Mobile cecum with incomplete fixation

Sigmoid Volvulus

Most common type; elongated sigmoid loop

Gastric Volvulus

Rare; rotation of the stomach

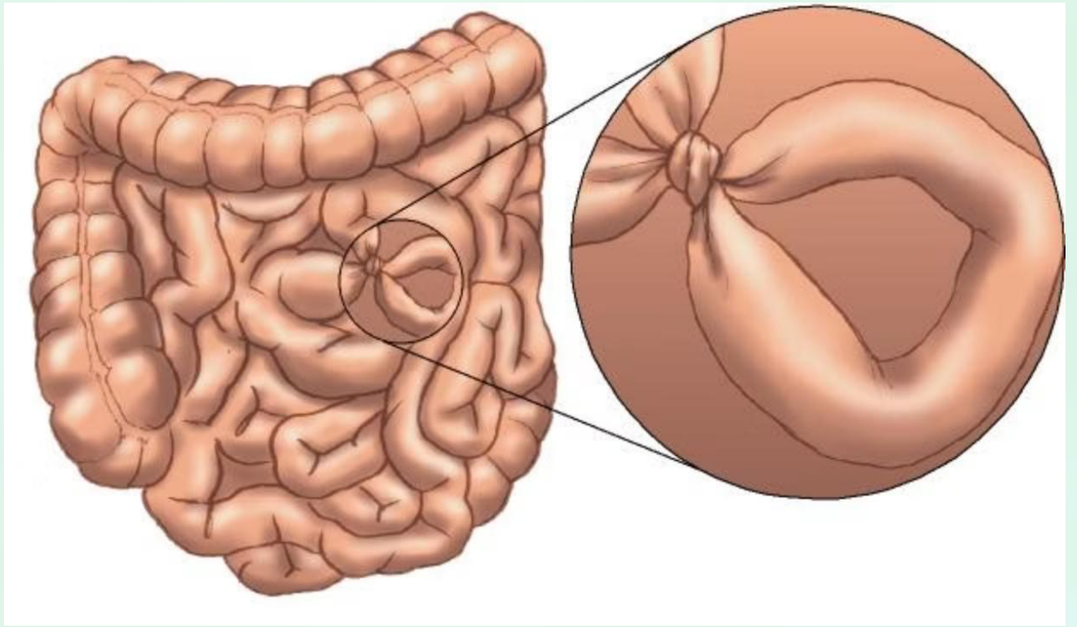


Illustration of sigmoid volvulus: the bowel loop twists on its mesenteric axis, creating a closed-loop obstruction.

Volvulus

Pathomechanism, Symptoms & Complications

Volvulus produces pathology through two simultaneous mechanisms: **(1) mechanical obstruction** causing distension, nausea, vomiting, and obstipation; and **(2) mesenteric ischemia** causing severe pain, mucosal necrosis, and systemic toxicity. The degree of injury depends on the degree and duration of rotation.

Obstruction

Accumulation of gas and fluid; progressive abdominal distension; high-pitched or absent bowel sounds

Ischemia

Venous then arterial occlusion → transmural infarction → perforation → fecal peritonitis

Systemic Sequelae

Bacterial translocation → sepsis → metabolic acidosis → multi-organ failure → death if untreated

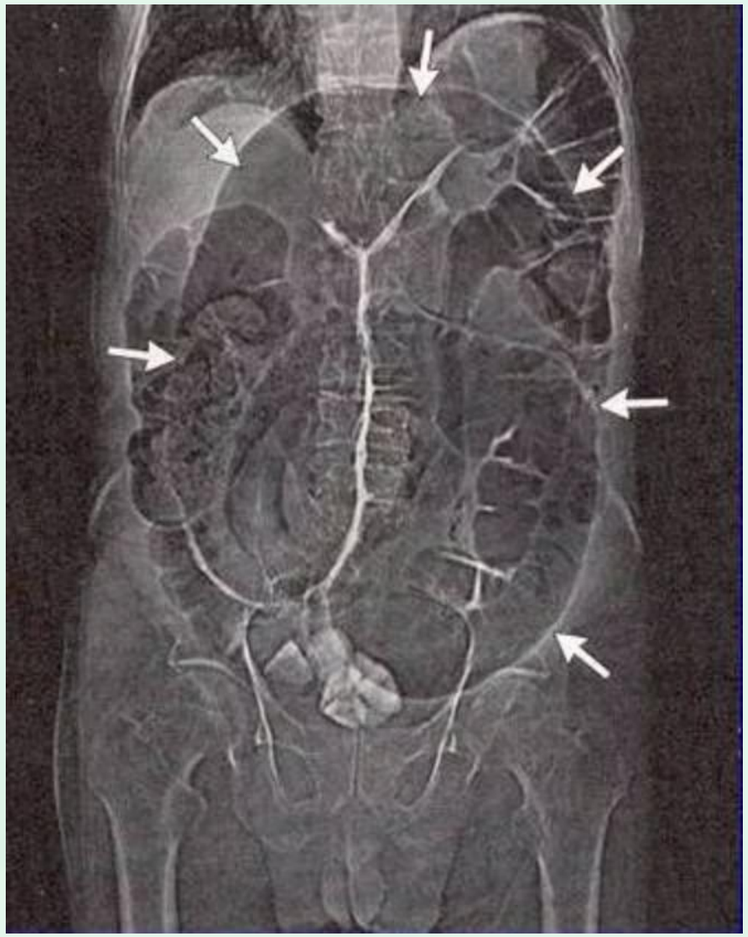
Acute volvulus with signs of strangulation (fever, tachycardia, peritonism, rising lactate) requires **immediate surgical intervention**. Delay increases mortality exponentially.

Volvulus — Imaging & Surgical Findings

Radiological Diagnosis

Plain abdominal X-ray may show the classic **"coffee bean" sign** (sigmoid volvulus) or **"bent inner tube" sign**. CT scan with IV contrast is the gold standard, demonstrating the whirl sign at the point of torsion, transition point, and bowel wall enhancement loss indicating ischemia.

- **X-ray:** Massively dilated loop; absent haustra
- **CT:** Whirl sign, mesenteric vessel torsion, free fluid
- **Most common in:** Middle-aged and elderly men; also in Duchenne muscular dystrophy (smooth muscle dysfunction)



CT abdomen (coronal view) showing massively dilated bowel loops with displaced mesenteric vessels — consistent with sigmoid volvulus. Source: surgical case archive.

Volvulus — Case Report 1

Sigmoid Volvulus with Gangrene: 62-Gangrene: 62-Year-Old Male

A 62-year-old patient presented with a 2-day history of **abdominal pain, constipation, and fever**. On examination: dehydration, toxemia, massive abdominal distension, tenderness, and absent bowel sounds. Emergency laparotomy revealed a **twisted and enormously dilated sigmoid colon with gangrenous changes**. Resection of the gangrenous segment with end colostomy was performed.

Intraoperative images showing gangrenous, massively distended sigmoid colon following volvulus. The necrotic bowel wall appears black/dark green — a sign of full-thickness infarction. Source: surgical case archive.

Key learning point: Gangrenous bowel cannot be safely reduced or preserved. Resection with proximal diversion (colostomy) is the standard of care. Mortality rises sharply when perforation has occurred.

Volvulus — Case Report 2

Mesenteric Venous Thrombosis Secondary to Volvulus: 15-Year-15-Year-Old Male

A 15-year-old boy presented with a 2-day history of upper abdominal pain migrating to the right lower quadrant, initially diagnosed as appendicitis. During appendectomy via Lanz incision, 400 mL of **dark hemoserous fluid** was drained — prompting conversion to full laparotomy.

Intraoperative findings revealed **mesenteric venous thrombosis secondary to mid-small bowel volvulus**, possibly complicated by herniation through a congenital band. Following resection of ischemic bowel, the patient was left with approximately **90 cm of viable small intestine** (from a normal 250–300 cm) — placing him at high risk for **short bowel syndrome**.

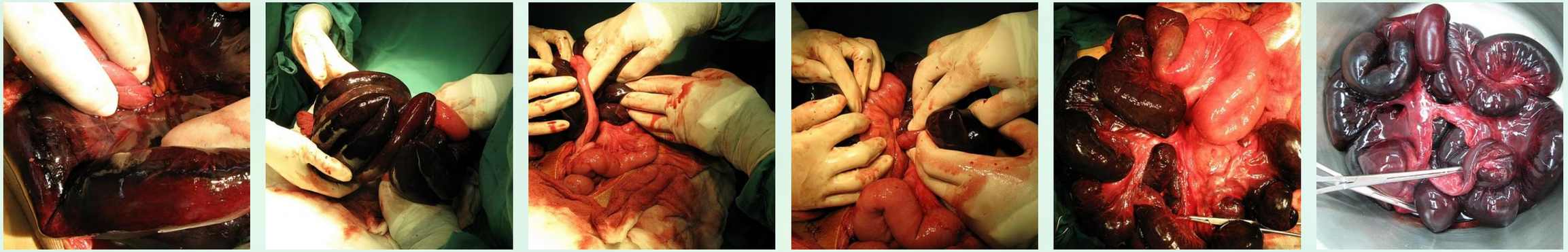
⚠ Short bowel syndrome is a life-altering complication requiring long-term parenteral nutrition and potential intestinal transplantation.

Intraoperative view: ischemic mid-small bowel surrounded by hemorrhagic peritoneal fluid following venous thrombosis secondary to volvulus. Source: wellsphere.com — surgical case report.

Volvulus — Additional Surgical Images

Operative Findings Gallery

The following images illustrate the range of intraoperative findings encountered in volvulus cases — from early venous congestion to frank transmural necrosis with perforation risk.



Surgical case archive images depicting volvulus at various stages of ischemic injury — congested (early), cyanotic (intermediate), and gangrenous (late/irreversible). Source: surgical case archives.

Special Forms — Adhesions

Intestinal Adhesions: Pathophysiology

Adhesions are fibrous bands forming between peritoneal surfaces, visceral organs, or the bowel wall — most commonly as a consequence of abdominal surgery. They represent the most frequent cause of small bowel obstruction in developed countries, accounting for up to 60–70% of all cases.

Formation Mechanism

Tissue Injury

Surgical trauma, infection, or ischemia disrupts peritoneal mesothelium

Fibrin Deposition

Coagulation cascade deposits fibrin "glue" across the peritoneal cavity

Fibrinolysis vs. Persistence

Normal fibrinolytic enzymes may dissolve the adhesion; impaired activity allows persistence

Fibrosis

Macrophages, fibroblasts, and endothelial cells invade → permanent fibrous adhesion with collagen

Types of Adhesions

Fibrinous Adhesions

Early post-operative (days 1–5); soft, vascular, often resolve spontaneously with fibrinolysis

Fibrous (Permanent) Adhesions Adhesions

Persist when fibrinolysis fails; contain mature collagen and neovascularization; require surgical lysis if obstructive

Risk factors for adhesion formation include prolonged surgery, bowel spillage, ischemia, infection, foreign bodies (mesh, suture material), and radiation.

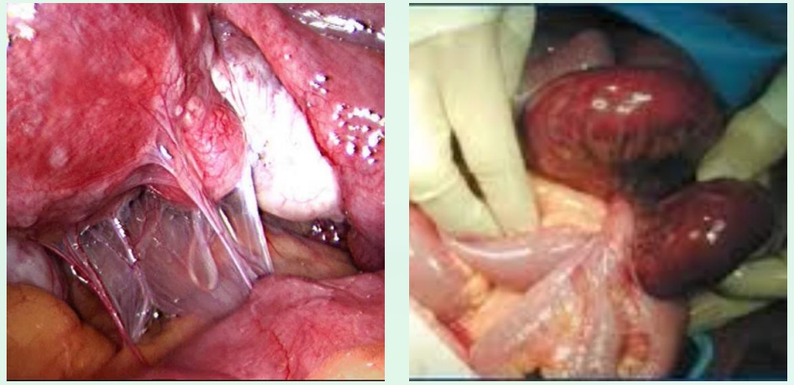
Adhesions

Clinical Impact & Imaging

Many adhesions remain asymptomatic. However, dense fibrous bands can tether adjacent bowel loops, mesentery, or the anterior abdominal wall, causing intermittent or complete obstruction. Angulation and kinking lead to luminal occlusion; constriction of the mesentery causes ischemia.

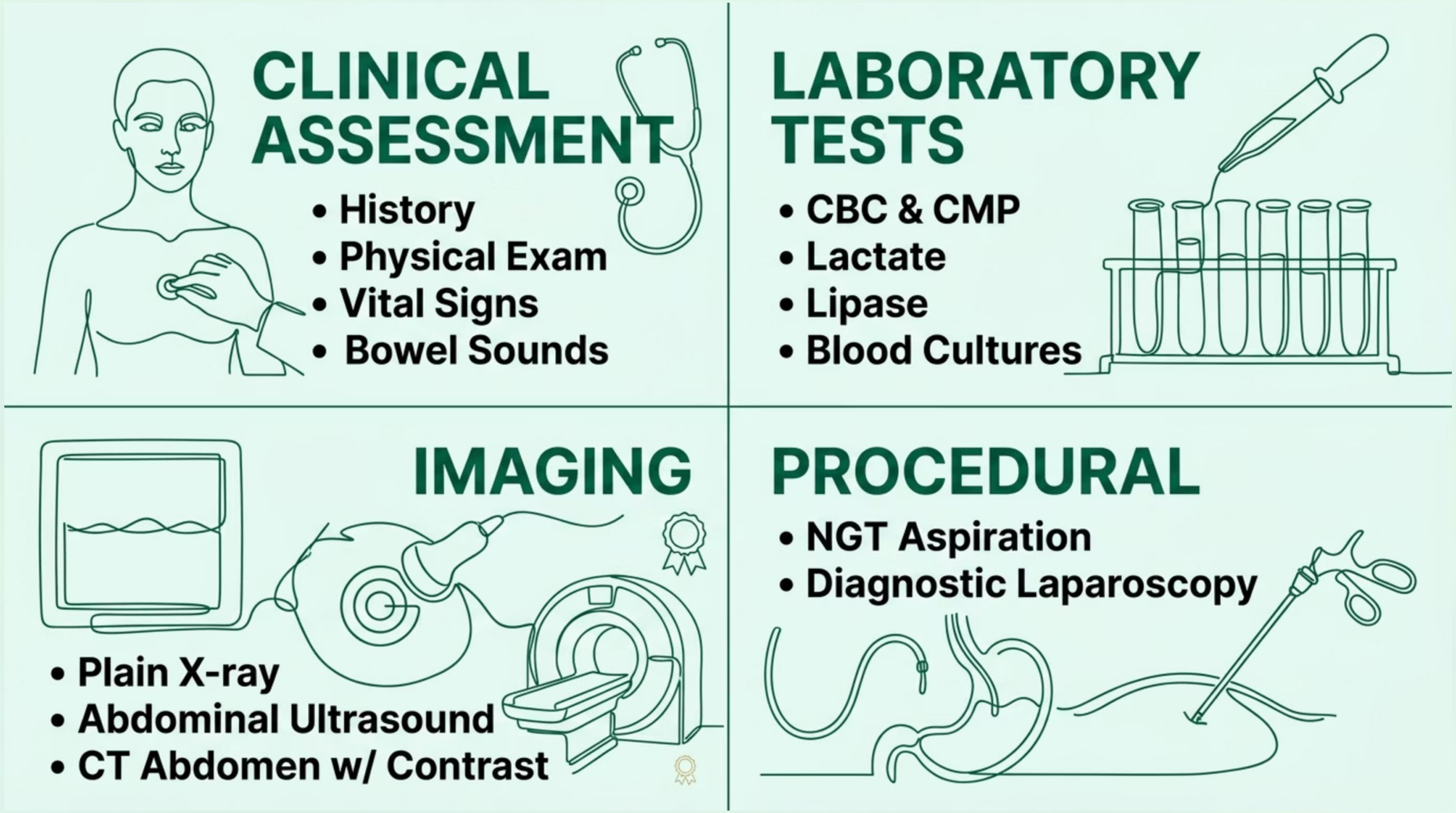
- Symptoms: Recurrent colicky abdominal pain, distension, nausea, vomiting, obstipation
- CT findings: Transition point, dilated proximal bowel, decompressed distal bowel, no mass
- Management: Conservative initially (NGT decompression, IV fluids); surgical adhesiolysis for complete/strangulating obstruction

⚠ Up to 30% of adhesion-related obstructions have an element of strangulation, requiring urgent operative intervention.

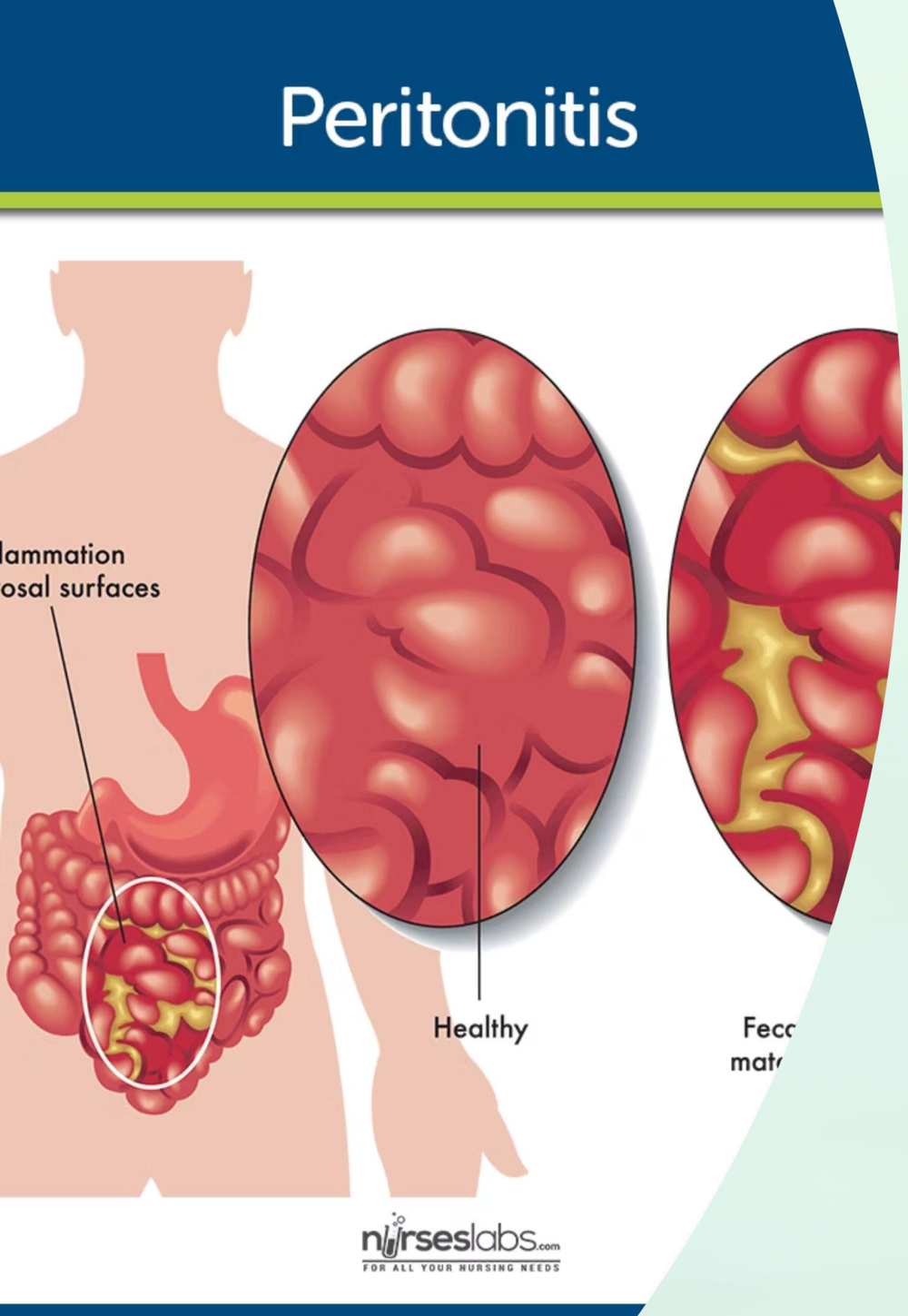


Illustrative and intraoperative images demonstrating fibrous adhesion bands causing bowel kinking and obstruction. Source: surgical case archives.

Diagnostic Approach to Ileus



Diagnosis integrates clinical findings with laboratory and imaging data. CT abdomen with IV contrast is the gold standard for identifying the level, cause, and severity of obstruction. Rising serum lactate, leukocytosis, and fever indicate strangulation and mandate urgent surgical consultation.



Chapter 4 — Complications

Complications of Untreated Ileus

Bowel Ischemia & Necrosis

Sustained distension compresses mucosal vessels → transmural infarction → perforation. Risk is highest in closed-loop obstruction (volvulus) and strangulated hernia.

Perforation & Peritonitis

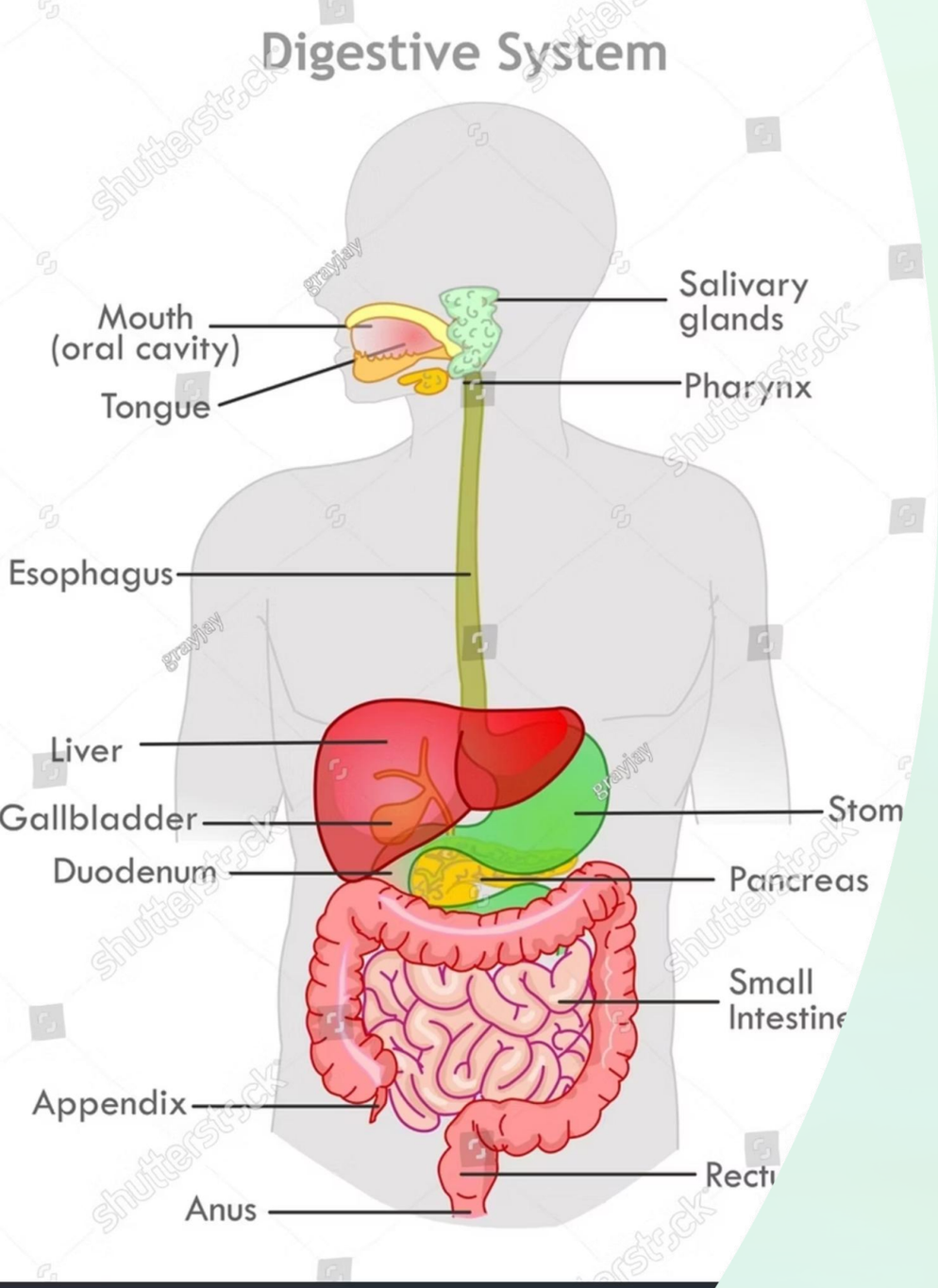
Full-thickness necrosis leads to intestinal perforation, spillage of fecal contents, and fecal peritonitis — a life-threatening surgical emergency with 30–50% mortality.

Sepsis & Multi-Organ Failure

Bacterial translocation across the ischemic mucosa drives systemic inflammatory response syndrome (SIRS), progressing to septic shock, acute kidney injury, and respiratory failure.

Short Bowel Syndrome

Extensive bowel resection (as in volvulus cases) reduces absorptive surface area below functional threshold, requiring long-term parenteral nutrition.



PATHOPHYSIOLOGY FUNCTIONAL GASTROENTEROLOGY

Irritable Bowel Syndrome (IBS)

A comprehensive overview for the course in **pathophysiology** — etiology, pathomechanism, symptomatology, diagnosis, and complications.

Definition and Basic Concepts

What is IBS?

IBS is a **functional gastrointestinal disorder (FGID)** characterized by chronic abdominal pain and changes in bowel habits **without identifiable structural or biochemical pathology** in the GI tract.

According to current terminology, IBS belongs to the **disorders of gut–brain interaction (DGBI)**, reflecting dysregulation between the enteric nervous system and the CNS.

Modern Classification

Chronic, Recurrent Course

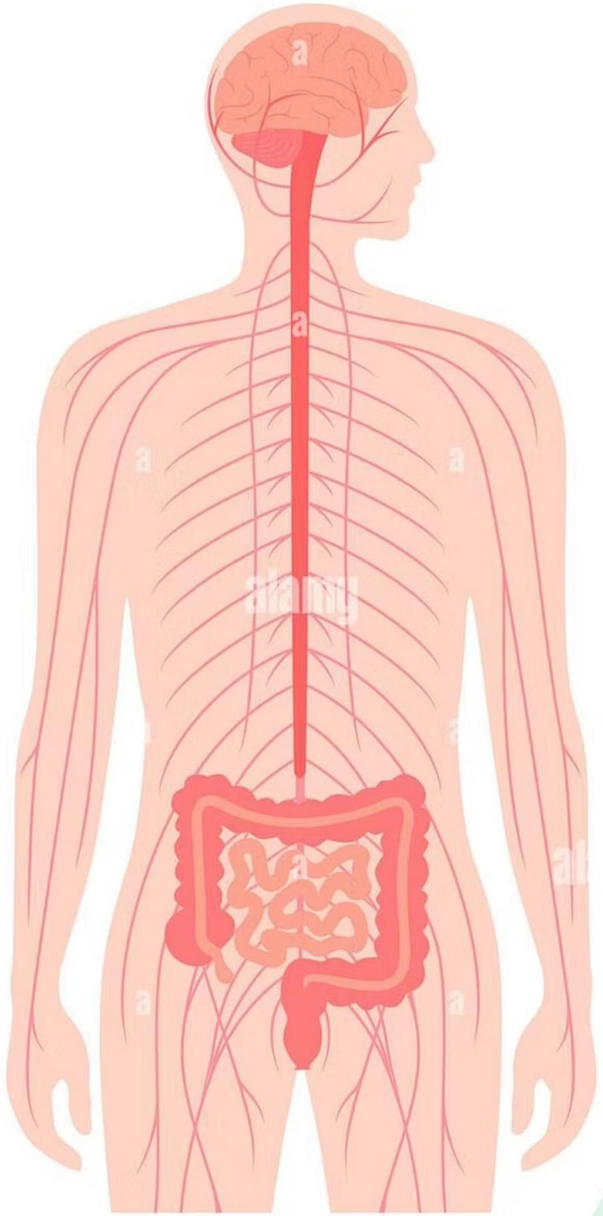
Symptoms persist for months to years with periods of remission and relapse.

No Detectable Organic Pathology

Standard structural investigations yield normal results.

Symptom-Based Diagnosis

Diagnosis relies on clinical criteria — **Rome IV Criteria**.



Etiology — Multifactorial Causes

Gut–Brain Axis

Abnormal bidirectional signaling between the CNS and the enteric nervous system leads to altered motility and visceral hypersensitivity.

Microbiome Alterations

Dysbiosis (altered composition of gut bacteria) can activate the immune system and generate symptoms.

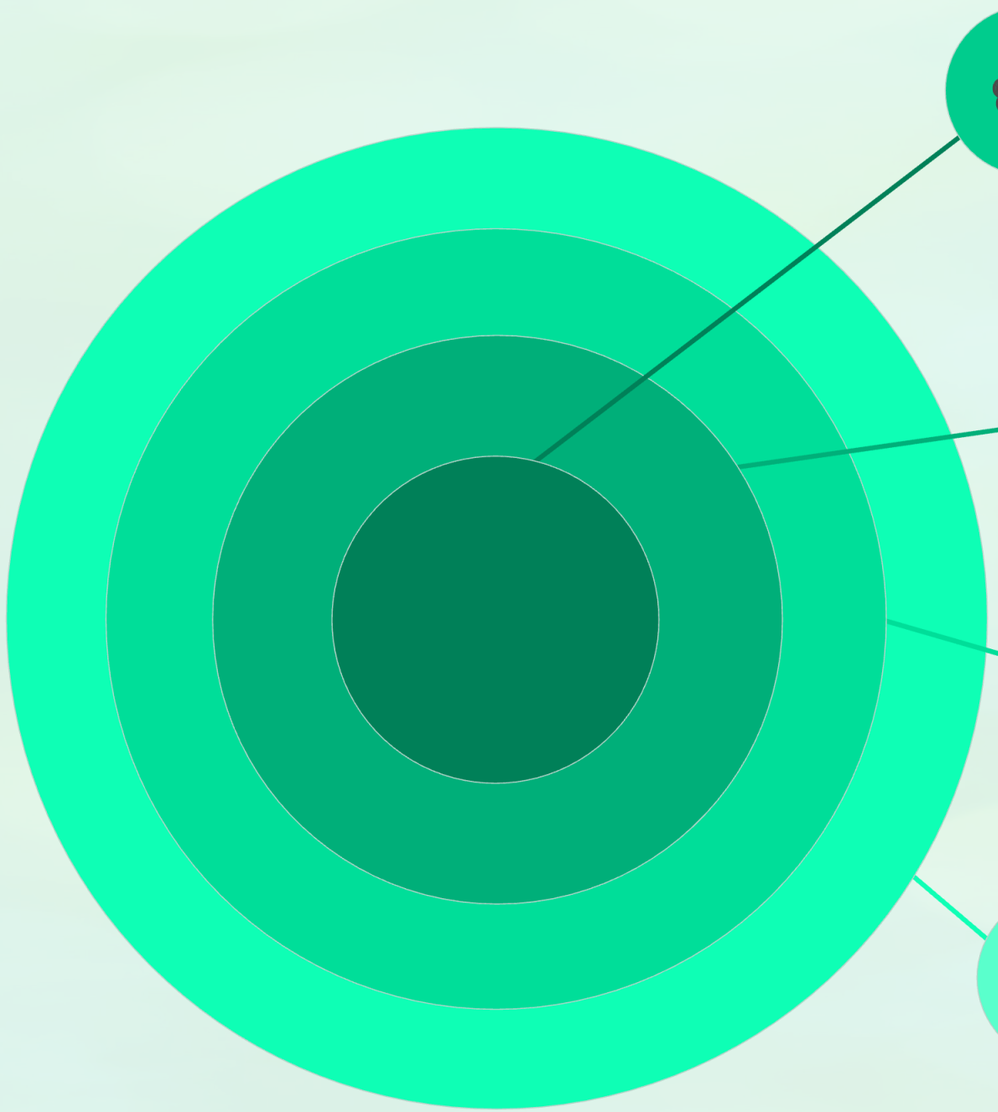
Psychosocial Factors

Stress, anxiety, depression, and early-life trauma are associated with increased risk of developing IBS.

Genetic Predisposition

Familial occurrence suggests a partial genetic contribution to disease development.

Pathomechanism — Key Processes



Gut–Brain Axis

Dysregulated signaling between gut and CNS.

Altered Motility

Hyper- or hypocontractions change transit.

Visceral Hypersensitivity

Heightened pain response to gut stimuli.

Post-infectious Inflammation

Infection-triggered immune changes persist.

The pathomechanism of IBS is complex: gut–brain axis dysfunction leads to altered motility (hypercontractions → diarrhea; hypocontractions → constipation) and visceral hypersensitivity — an increased sensitivity to luminal distension by gas or stool. Following gastroenteritis, **post-infectious IBS** may develop with persistent inflammatory changes of the mucosa.

Visceral Hypersensitivity — A Detailed View

Mechanism

Patients with IBS have a **lowered pain threshold** in response to intestinal distension. A normal physiological stimulus (gas, stool) is perceived as painful. This phenomenon is called **central sensitization** — alterations in pain processing at the level of the spinal cord and brain.

- Increased expression of pain receptors (e.g., TRPV1) in the intestinal wall
- Altered activity of serotonin pathways (5-HT)
- Dysregulation of the HPA axis under chronic stress

Clinical Consequence

The disproportionate pain response explains why IBS patients experience intense pain despite the absence of structural intestinal damage.

Visceral hypersensitivity is considered the **central pathomechanism** of IBS and the primary target of pharmacotherapy.

Bristol Stool Chart

Type 1		Separate hard lumps, like nuts (hard to pass)
Type 2		Sausage-shaped but lumpy
Type 3		Like a sausage but with cracks on the surface
Type 4		Like a sausage or snake, smooth and soft
Type 5		Soft blobs with clear-cut edges
Type 6		Fluffy pieces with ragged edges, a mushy stool
Type 7		Watery, no solid pieces. **Entirely Liquid**

IBS Classification — Rome IV Criteria Criteria

IBS-C

Constipation-predominant type. Hard or lumpy stools predominate (Bristol type 1–2).

IBS-D

Diarrhea-predominant type. Loose or watery stools predominate (Bristol type 6–7).

IBS-M

Mixed type. Alternating constipation and diarrhea.

IBS-U

Unclassified type. Does not meet criteria for any of the above categories.

Classification by the predominant stool pattern is clinically relevant for selecting the appropriate treatment. Source: NIDDK / Rome IV Criteria.

Symptomatology

Cardinal (Main) Symptoms

- Recurrent abdominal pain (often relieved by defecation)
- Change in stool frequency and consistency
- Bloating and abdominal distension
- Excessive gas production (flatulence)
- Mucus in stool
- Sensation of incomplete evacuation

Extraintestinal Manifestations

- Fatigue and malaise
- Sleep disturbances
- Anxiety and depression (common comorbidities)

Symptom Triggers

- Stress and psychological burden
- Certain foods (e.g., FODMAPs)
- Hormonal changes (menstrual cycle)

Diagnosis of IBS

IBS is a **diagnosis per exclusionem**. The diagnosis is established based on the **Rome IV Criteria**: recurrent abdominal pain at least 1 day per week over the last 3 months, associated with a change in stool frequency or form. Organic diseases must be excluded before the diagnosis is confirmed.

Clinical Evaluation

Detailed history and physical examination; assessment of symptom pattern and duration.

Exclusion of Red Flags

Identify alarm symptoms that require further investigation to rule out serious pathology.

Laboratory & Imaging

CBC, CRP, calprotectin, celiac antibodies; colonoscopy if indicated.

Diagnosis Established

Rome IV criteria met; organic causes excluded; classification into IBS subtype.

Alarm Symptoms — "Red Flags"

The presence of the following symptoms requires **immediate further investigation** to exclude serious organic pathology — IBS does not explain them!

Rectal Bleeding

May indicate IBD, polyps, or colorectal carcinoma. Must be investigated promptly.

Unexplained Weight Loss

Typical for malignancy or malabsorption syndromes. Requires thorough workup.

Anemia

Requires exclusion of occult bleeding or celiac disease as an underlying cause.

Nocturnal Symptoms

Symptoms waking the patient from sleep are not typical of functional disorders.

Differential Diagnosis

Key distinction: IBS **does not cause structural damage** to the intestine and **does not increase the risk of carcinoma** — this differentiates it from organic GI diseases. Source: Mayo Clinic, Wikipedia.

Table 2. Differential Diagnosis of Irritable Bowel Syndrome Symptoms

- Carcinoid tumor
- Celiac disease
- Colorectal cancer
- Diverticular disease
- Drug use (opiate analgesics, calcium channel blockers, antidepressants)
- Gastrointestinal infection (e.g., *Giardia, Amoeba,* human immunodeficiency virus, bacterial overgrowth)
- Hyperthyroidism
- Hypothyroidism
- Inflammatory bowel disease (e.g., Crohn disease, ulcerative colitis)
- Ischemic colitis
- Lactose intolerance

Inflammatory Bowel Disease

Crohn's disease, ulcerative colitis — confirmed by colonoscopy with biopsy and elevated calprotectin/CRP.

Celiac Disease

Gluten-sensitive enteropathy — diagnosed by serological antibody testing and duodenal biopsy.

Colorectal Carcinoma

Excluded by colonoscopy; red flags such as bleeding, weight loss, and anemia must prompt investigation.

Microscopic Colitis

Normal endoscopic appearance but histological inflammation — requires biopsy for diagnosis.

Complications and Impact on Quality of Life

Direct Complications

- Chronic pain reducing work capacity and daily functioning
- Malnutrition from strict elimination diets
- Dehydration in severe IBS-D

Psychosocial Complications

- Development or worsening of anxiety disorders and depression
- Social isolation and avoidance of activities
- Reduced quality of life (QoL) comparable to chronic organic diseases

IBS does not lead to structural damage of the intestine nor to an increased risk of colorectal carcinoma.

Summary — Exam Review

01

Definition

Functional GI disorder (DGBI) — abdominal pain + change in bowel habits, **without organic pathology**.

02

Etiology

Multifactorial: gut–brain axis, motility disturbance, visceral hypersensitivity, dysbiosis, stress, genetics.

03

Classification

IBS-C (constipation), IBS-D (diarrhea), IBS-M (mixed), IBS-U (unclassified) — according to Rome IV Criteria.

04

Diagnosis

Diagnosis per exclusionem. Rome IV Criteria. Exclude "red flags" (bleeding, weight loss, anemia, nocturnal symptoms).

05

Key Point

IBS **does not cause structural damage** and **does not increase the risk of carcinoma**. Sources: Wikipedia, Mayo Clinic, NIDDK.

Irritable Bowel Syndrome (IBS): *Pathogenesis and clinical findings*

Authors: Ben Campbell
Reviewers:
Mehul Gupta
Kiana Hampton
Yan Yu*
Edwin Cheng*
** MD at time of publication*

Pathogenesis is multifactorial, not fully understood, and may arise from any one or a combination of these factors

Genetic factors are believed to influence many of these pathways

Environmental factors

Gut infection, ischemia, injury, chronic low-level inflammation, altered microbiome

Abnormal gut chemical milieu (cytokines, lymphocytes, mast cells, hormones)

One common pathway: altered quantity or activity of serotonin-releasing enterochromaffin cells and serotonin reuptake transporters in gut *

Serotonin is a key stimulator of gut muscle contraction and sensory signaling

Altered bowel motility
Bowel motility can ↑, ↓ or alternate ↑/↓

Psychosocial factors

Anxiety, depression, adverse experiences, dysregulated stress response

Brain can alter normal reflexes of enteric nervous system (e.g. via hypothalamic stress hormone release)

Complex mechanisms

↓ Brain's ability to modulate pain signals from gut; ↑ vigilance to pain

↑ Activity of sensory receptors in gut + recruitment of formerly "silent" receptors

Altered visceral bowel sensation
Visceral hypersensitivity

Note:
*Several signaling pathways in the gut are believed to play a role in IBS. The serotonin pathway is represented here for illustrative purposes, and it is a common target of therapy.

Irritable Bowel Syndrome

Disorder of brain-gut interaction with gastrointestinal manifestations

Not a predominantly inflammatory condition

- No lesions in gut mucosa → **Absence of bloody stool and anemia**
- **Absence of nocturnal symptoms and inflammatory markers**

(Absence of "red flags" for organic disease)

During times of ↓ bowel motility:

- ↑ Impacts of other factors that precipitate constipation → **Dietary sensitivity** (e.g. to inadequate intake of motility-promoting fibre)
- ↑ Time for water absorption from feces and bacterial fermentation → ↑ Hard stool and trapped gas in bowel → **Constipation**, **Bloating**

↑ Number and sensitivity of active pain receptors (nociceptors) in gut → **Hyperalgesia** ↑ Response to painful stimulus

Stretch receptors in gut stimulate pain in normally unconscious neural pathways → **Allodynia** Pain in response to normal stimulus

↑ Hard stool and trapped gas in bowel + **Hyperalgesia** + **Allodynia** → ↑ Stretch / stimulation of visceral afferent nerves in gut → **Diffuse abdominal pain**

During times of ↑ bowel motility:

- ↑ Peristalsis → ↑ Stretch / stimulation of visceral afferent nerves in gut
- ↓ Time for water absorption from feces → ↑ Water volume in gut lumen → **Bowel distension**, **Diarrhea**
  - ↑ Impacts of other factors that precipitate diarrhea → **Dietary sensitivity** (e.g. to intake of fermentable oligo/di/monosaccharides and polyols—FODMAPs—which draw water into gut lumen by osmosis)

All symptoms may exacerbate psychosocial factors

Legend: Pathophysiology | Mechanism | Sign/Symptom/Lab Finding | Complications

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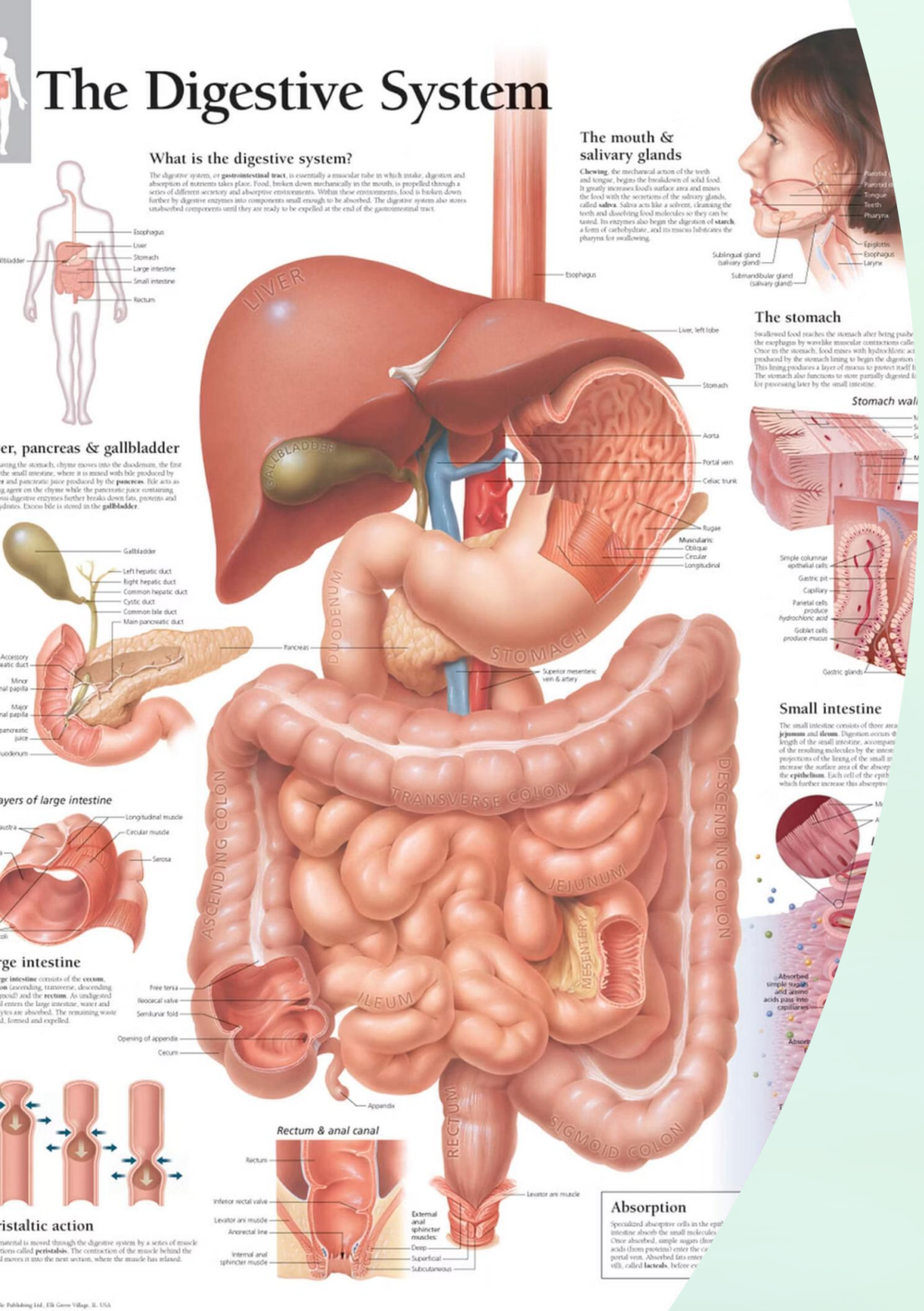


Inflammatory Bowel Disease

Crohn's Disease — Pathomechanism, Symptoms, Diagnosis & Complications

A comprehensive clinical review for medical students and healthcare professionals covering the spectrum of IBD, with a focus on Crohn's disease: its immunological basis, clinical presentation, diagnostic workup, and management of complications.

MEDICAL EDUCATION GASTROENTEROLOGY

IBD vs. IBS — Understanding the Distinction

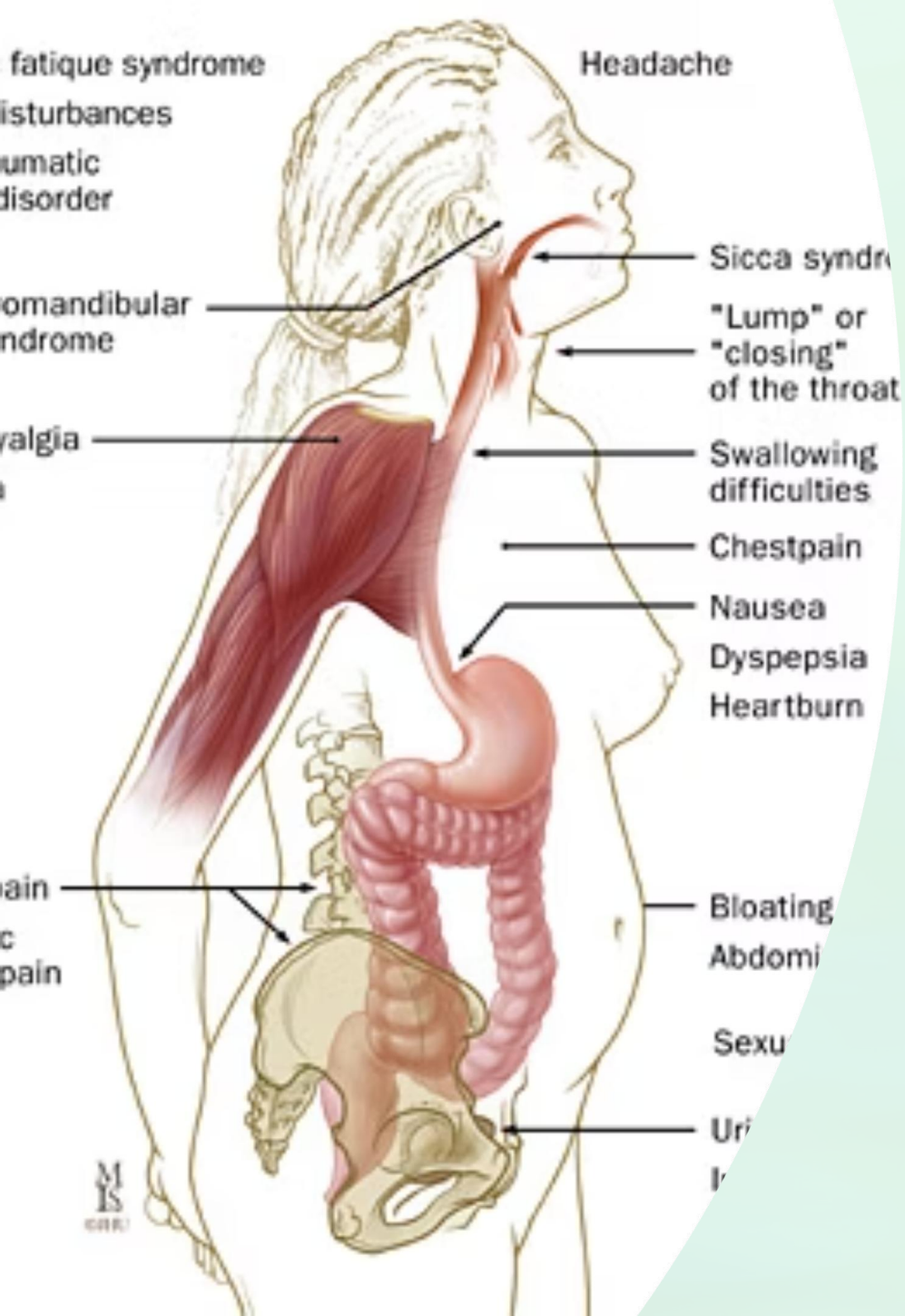
Inflammatory Bowel Disease (IBD) is frequently confused with Irritable Bowel Syndrome (IBS). IBD encompasses a group of **organic, immune-mediated inflammatory conditions** of the gastrointestinal tract confirmed by objective findings (endoscopy, histology, imaging), while IBS is a functional disorder with no structural or inflammatory basis. The two major forms of IBD are **Crohn's disease** and **ulcerative colitis**.

IBD (Inflammatory Bowel Disease)

- Structural, immune-mediated inflammation
- Detectable on endoscopy and histology
- Crohn's disease and ulcerative colitis
- Risk of serious complications

IBS (Irritable Bowel Syndrome)

- Functional disorder — no visible damage
- Diagnosis of exclusion
- Multisystemic symptoms (see image)
- No increased cancer risk



IBS — The Multisystemic Symptom Symptom Spectrum

IBS presents with a wide range of symptoms extending well beyond the gut. This diagram illustrates the breadth of manifestations: from headaches and sicca syndrome superiorly, to chest pain and heartburn in the thorax, to bloating, abdominal distension, and urinary urgency inferiorly. Understanding this spectrum helps clinicians differentiate IBS from organic IBD.

Source: Medical illustration of IBS symptom distribution. For educational reference only.

IBD — Crohn's vs. Ulcerative Colitis: Key Differences

The two major forms of IBD are distinguished primarily by the **location** and **depth** of inflammatory changes. Understanding these differences is essential for diagnosis and treatment planning.

Crohn's Disease

- Any part of GI tract: mouth to anus
- Most commonly: terminal ileum
- Skip lesions (non-continuous)
- Transmural inflammation (full bowel wall)
- Granuloma formation

Ulcerative Colitis

- Restricted to colon and rectum
- Begins at rectum, extends proximally
- Continuous lesions
- Mucosal inflammation only
- No granulomas

Both conditions can present with extra-intestinal manifestations affecting the liver, joints, skin, and eyes — though in different proportions.

Etiology of IBD — A Multifactorial Disease

The exact cause of IBD remains incompletely understood. Current research points to a complex interplay of **genetic susceptibility**, **immune dysregulation**, **environmental triggers**, and **gut microbiota imbalance**. No single factor is sufficient to cause disease; rather, multiple factors converge in a genetically predisposed individual.

Genetic Factors

Mutations in the **NOD2 gene** (chromosome 16) impair bacterial recognition. Association with cystic fibrosis and ankylosing spondylitis provides additional indirect genetic evidence.

Immune Dysregulation

A defective immune response leads to **uncontrolled inflammation** triggered by bacteria, viruses, or dietary proteins. The inflammatory cascade fails to switch off appropriately.

Environmental Triggers

Smoking significantly increases Crohn's risk. Oral contraceptive use, **westernized diet**, early antibiotic exposure, and urban living are recognized environmental contributors.

Pathomechanism of Crohn's Disease

Crohn's disease results from a dysregulated mucosal immune response to luminal antigens — particularly commensal bacteria — in a genetically susceptible host. The key steps are well-characterized at the molecular level.

Barrier Defect

Increased intestinal permeability allows translocation.

Immune Activation

Th1/Th17 response with TNF-α, IL-12, IL-23 release.

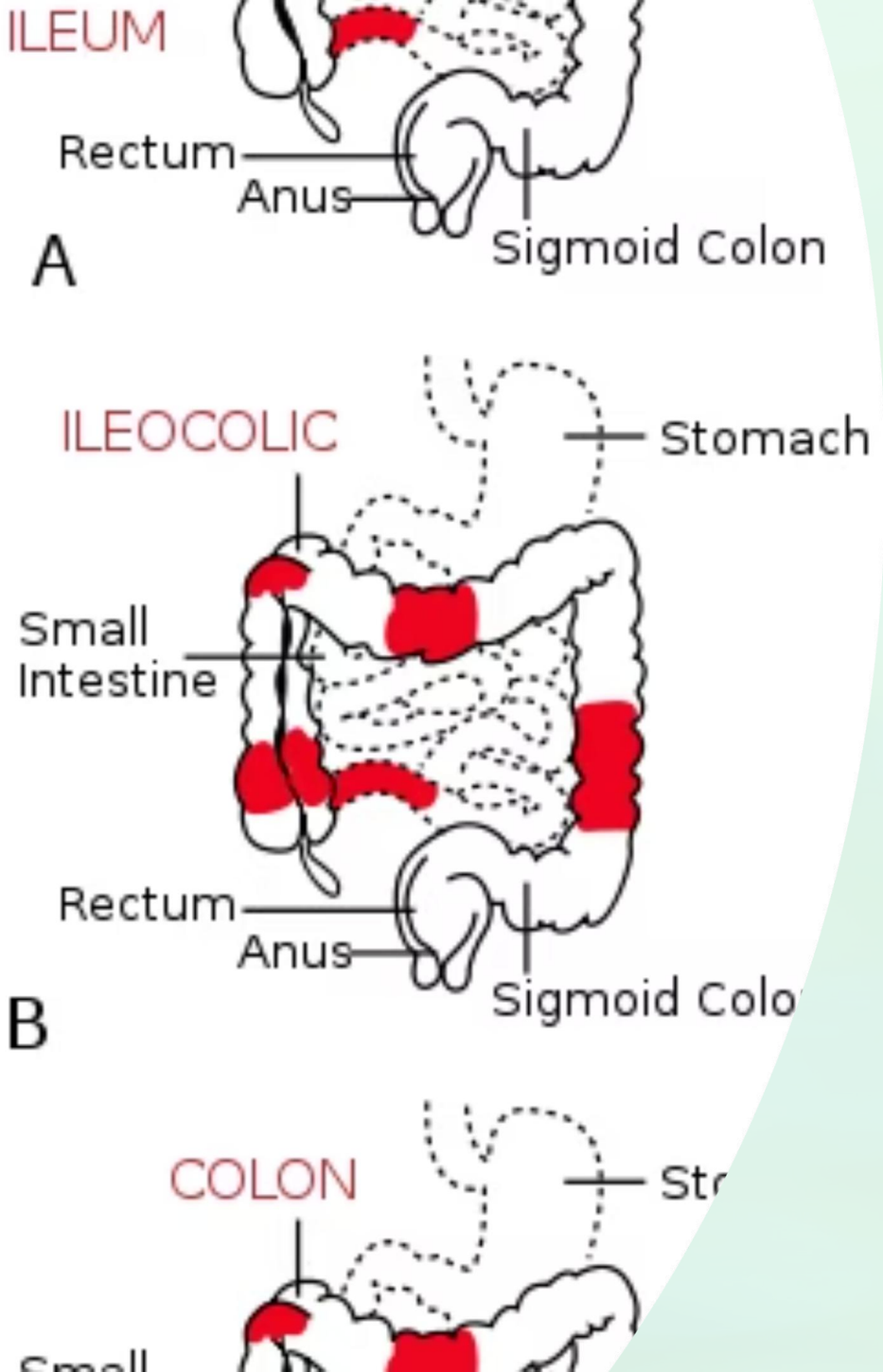
Genetic Susceptibility

NOD2 mutation impairs pathogen recognition.

Chronic Inflammation

Transmural inflammation with granulomas and fibrosis.

The dominant immune pathway involves **Th1 and Th17 lymphocyte activation**, driven by cytokines such as TNF-α, IL-12, and IL-23. This leads to macrophage recruitment, granuloma formation, and ultimately transmural tissue destruction — the hallmark of Crohn's pathology.



Crohn's Disease — Definition and and Distribution Patterns

Crohn's disease is a **chronic, transmural, granulomatous inflammatory disease** that can involve any segment of the gastrointestinal tract from the mouth to the anus. The image depicts the three most common anatomical patterns:

A — Ileal (30%)

Isolated terminal ileum involvement. Classic presentation with right lower quadrant pain.

B — Ileocolic (40%)

Most common pattern. Involves terminal ileum and ascending colon with skip lesions.

C — Colonic (25%)

Colon-predominant. May mimic ulcerative colitis but with transmural and skip features.

Source: Anatomical diagram of Crohn's disease distribution patterns. For educational reference only.

Gastrointestinal Symptoms of Crohn's Disease

Crohn's disease presents with a broad spectrum of GI symptoms reflecting its ability to involve any segment of the digestive tract. Symptoms vary based on the location and severity of inflammation.

Intestinal Symptoms

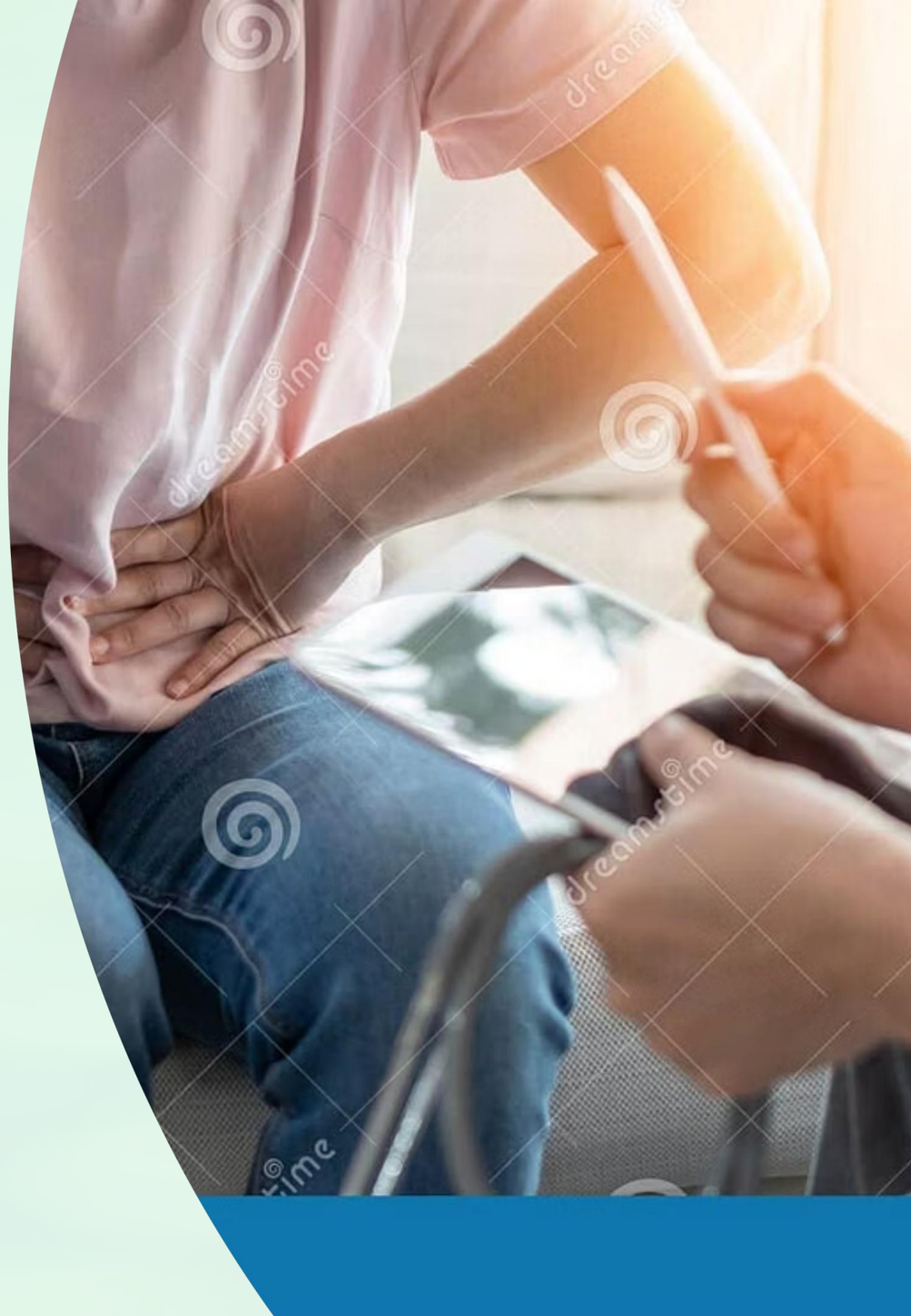
- Abdominal pain (most common — RLQ cramps)
- Chronic diarrhea, fecal incontinence
- Flatulence, bloating, distension
- Nausea and vomiting
- Perianal discomfort, fistulae, abscesses

Upper GI Involvement

- **Mouth:** Aphthous ulcers (10–20% of patients)
- **Esophagus:** Dysphagia (rare)
- **Stomach/duodenum:** Epigastric pain, early satiety

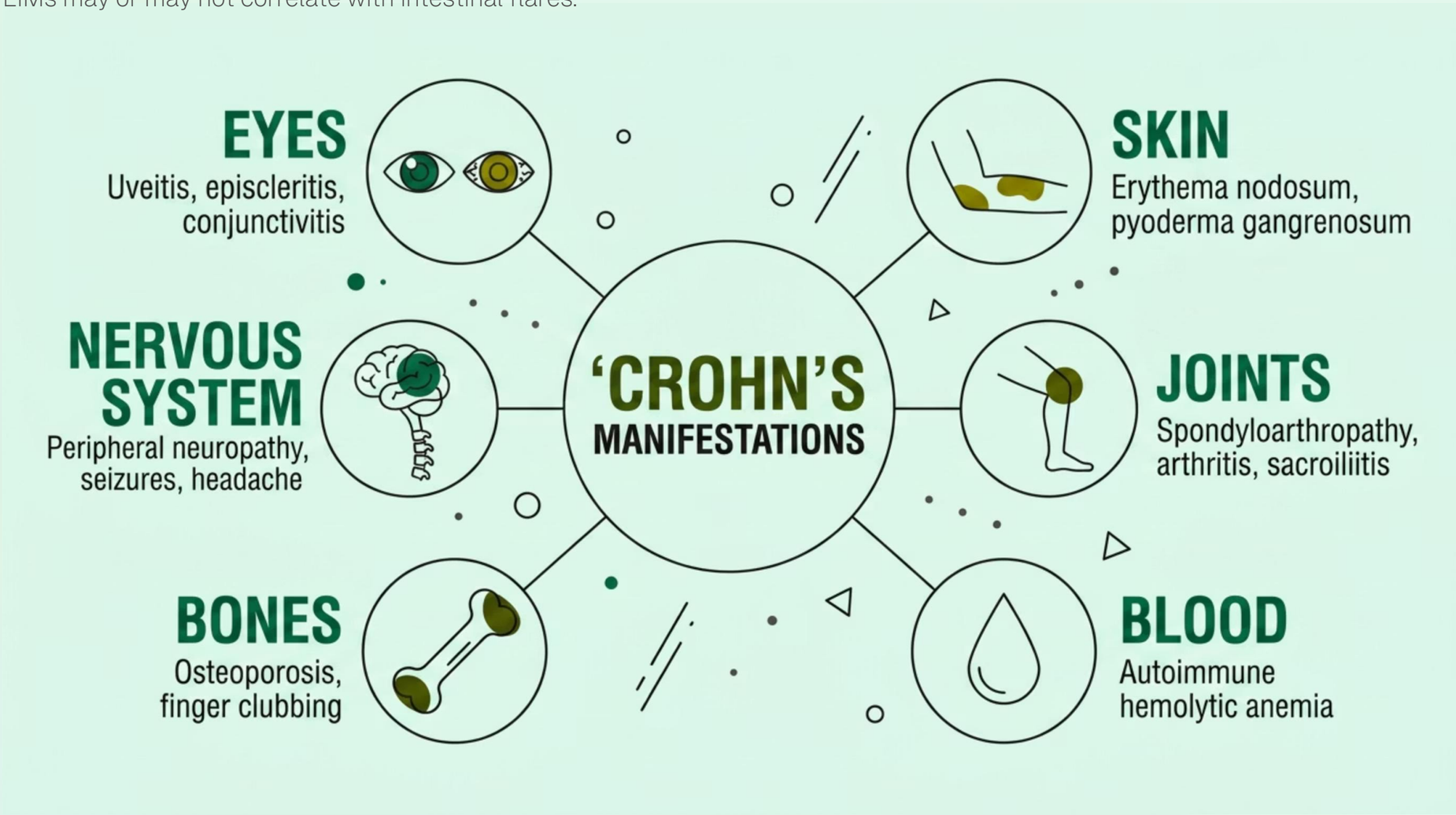
Systemic Symptoms

- Fever, fatigue, malaise
- Weight loss, anorexia
- Growth failure (children)



Extra-Intestinal Manifestations of Crohn's Disease

Up to **40% of Crohn's patients** develop extra-intestinal manifestations (EIMs), which can precede, coincide with, or follow bowel disease activity. EIMs may or may not correlate with intestinal flares.



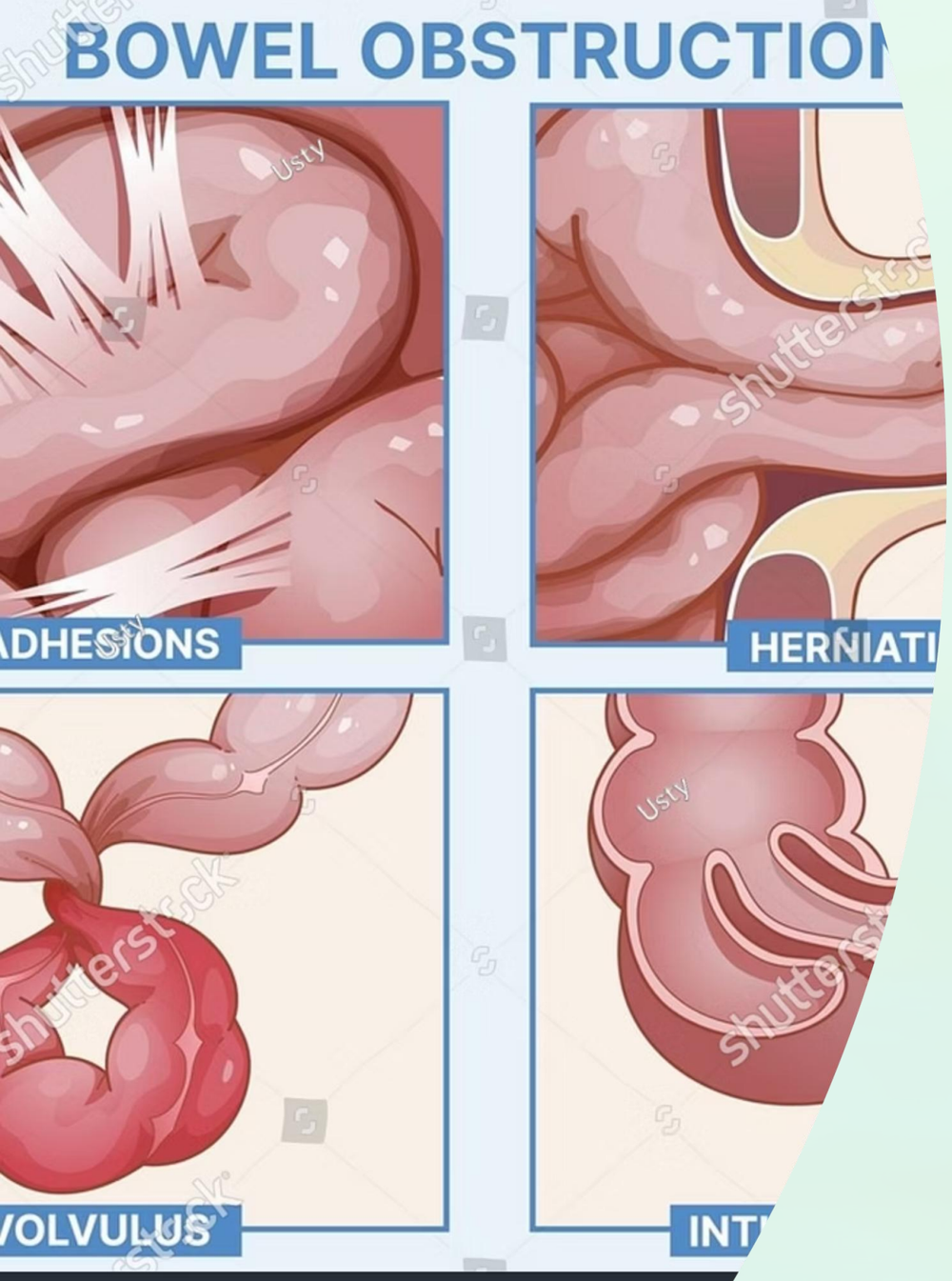
⚠ Uveitis and spondyloarthropathy may be independent of bowel disease activity and require separate monitoring and management.

Extra-Intestinal Manifestations — Clinical Images

Visual recognition of extra-intestinal manifestations is essential for early diagnosis. The following images depict the most clinically significant skin and perianal findings seen in Crohn's disease.

Perianal fistula (left) and anal fissure (right) — common perianal complications seen in Crohn's disease. Source: dermis.net, educational use.

Erythema nodosum (top left), Uveitis (top right), Pyoderma gangrenosum (bottom) — classical extra-intestinal skin and eye manifestations. Source: Clinical educational images.



Complications of Crohn's Disease

The transmural nature of Crohn's inflammation predisposes patients to a range of serious structural and metabolic complications. Early recognition is critical to prevent life-threatening outcomes.

Structural / Luminal

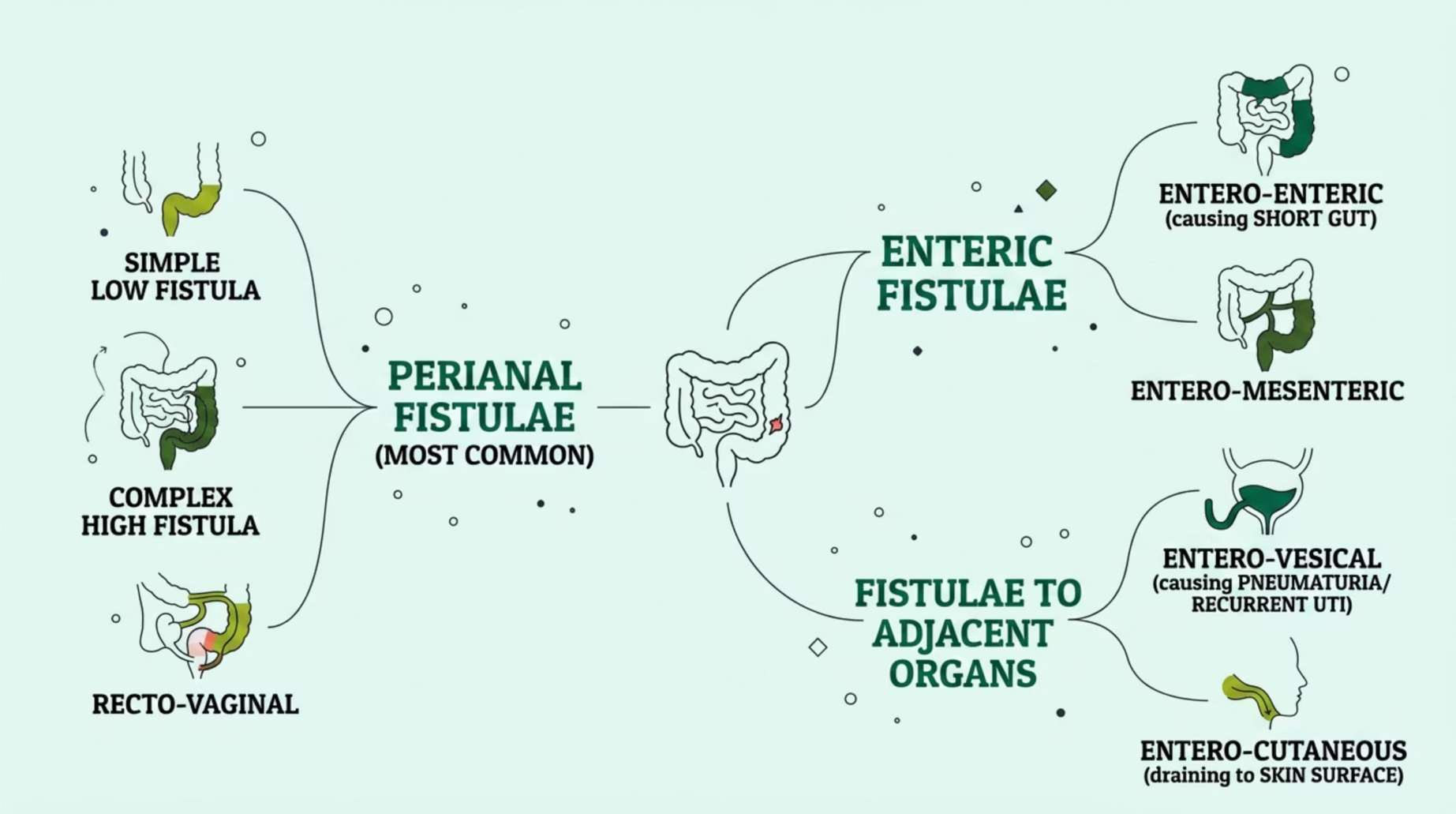
- **Bowel obstruction** — due to strictures, adhesions, or acute edema
- **Fistulae** — entero-enteric, entero-vesical, entero-cutaneous, recto-vaginal
- **Abscesses** — intra-abdominal or perianal collections
- **Perforation** and peritonitis (less common than in UC)
- **GI bleeding** — acute or chronic

Metabolic / Systemic

- **Malnutrition and malabsorption** — fat-soluble vitamins (A, D, E, K), B12, iron
- **Osteoporosis** — from chronic inflammation and steroid use
- **Small intestinal adenocarcinoma** — elevated risk after long disease duration
- **Infection** — from immunosuppressive therapy

Fistula Classification in Crohn's Disease

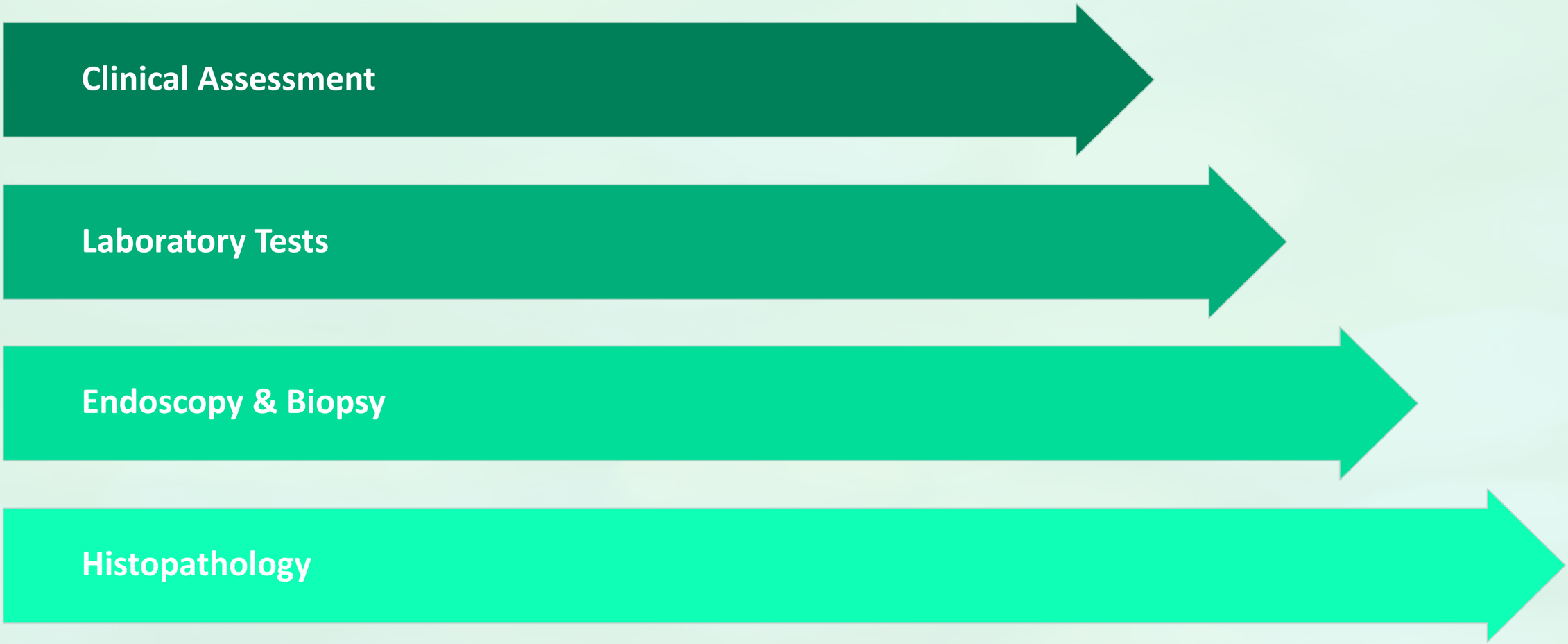
Fistulae are a hallmark complication of transmural Crohn's inflammation, occurring in up to **35% of patients** over a lifetime. They form when transmural ulceration penetrates adjacent structures. Classification guides surgical vs. medical management.



⚠ Complex fistulae with associated abscess require drainage before anti-TNF biological therapy can be initiated.

Diagnosis of Crohn's Disease — Overview

There is no single gold-standard test for Crohn's disease. Diagnosis requires **clinical, endoscopic, histological, radiological, and laboratory correlation**. The goal is to confirm inflammation, characterize extent, assess disease activity, and rule out infection or malignancy.



Fecal calprotectin is a highly sensitive non-invasive marker for mucosal inflammation, useful for distinguishing IBD from functional disorders and monitoring disease activity.

Laboratory Investigations

Inflammatory Markers

- CRP — elevated in active disease
- ESR — non-specific but useful
- Fecal calprotectin — mucosal inflammation marker (sensitive, non-invasive)
- Fecal lactoferrin — neutrophil activation marker

Serological Markers

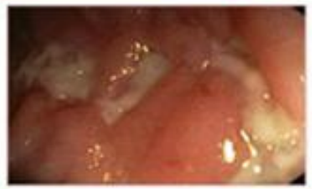
- ASCA (anti-Saccharomyces cerevisiae antibodies) — positive in ~60% of Crohn's
- pANCA — more associated with UC

Full Blood Count & Biochemistry

- Anemia (iron deficiency or B12/folate deficiency)
- Leukocytosis — active inflammation or infection
- Hypoalbuminemia — malnutrition indicator
- Elevated alkaline phosphatase — hepatobiliary involvement

Stool Culture

- Rule out infectious colitis (C. difficile, Salmonella, Campylobacter)
- Mandatory before immunosuppressive therapy

ENDOSCOPIC FINDING	ENDOSCOPY
No lesions	
<5 aphthous lesions	
i2a. Lesions confined to the anastomosis i2b. 5 aphthous lesions with normal mucosa between lesions; areas scattered with larger lesions	
Diffuse aphthous ileitis over inflamed mucosa	
Diffuse inflammation with large ulcers, nodules and/or strictures	

Endoscopy — The Diagnostic Cornerstone

Ileocolonoscopy with biopsy is the primary diagnostic tool for Crohn's disease. It allows direct visualization of the mucosa and targeted tissue sampling for histological confirmation.

Macroscopic Findings

- Deep longitudinal ulcers ("bear claw" pattern)
- Cobblestone mucosa
- Skip lesions
- Strictures and stenoses

Histological Findings

- Transmural inflammation
- Non-caseating granulomas (pathognomonic)
- Cryptitis and crypt abscesses
- Submucosal fibrosis

Capsule Endoscopy

- Detects small bowel lesions beyond reach of standard endoscope
- Risk of retention in strictures — contraindicated without patency capsule test

COBBLESTONE APPEARANCE

GI alternating normal and denuded mucosa from ulceration, esp in CROHN'S DISEASE OF COLON.

Crohn Disease

Regional enteritis

Clinical

- Chronic diarrhea
- Crampy abdominal pain
- Fever
- Weight loss
- Strictures
- Fistulization

Extraintestinal manifestations

- Pyoderma gangrenosum
- Erythema nodosum
- Ankylosing spondylitis or sacroiliitis
- Arthritis
- Uveitis
- Liver disease
- Renal stones

RoshReview

Cobble Stone Appearance - Crohn's Disease
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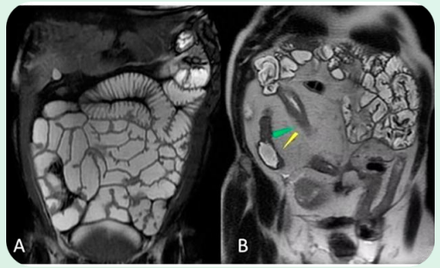
Imaging in Crohn's Disease

Cross-sectional imaging is indispensable for assessing **transmural disease extent**, detecting complications (strictures, fistulae, abscesses), and planning surgery. Different modalities offer complementary information.



MRI Enterography

Preferred modality for **small bowel Crohn's**. No radiation. Excellent for fistula mapping, perianal disease, and assessing wall edema vs. fibrosis. Used for monitoring and pre-surgical planning.

CT Enterography

Fast and widely available. Ideal in **acute complications** (obstruction, abscess, perforation). Higher radiation exposure — use judiciously, especially in young patients.

Bowel Ultrasound

Non-invasive and radiation-free. Detects bowel wall thickening, hyperemia (Doppler), and free fluid. Operator-dependent but increasingly used for **follow-up** in experienced centers.

Disease Activity Scoring — CDAI

The Crohn's Disease Activity Index (CDAI) is the most widely used tool for quantifying disease severity in clinical trials and practice. It integrates patient-reported outcomes with objective clinical parameters over an 8-day diary period.

1. **Number of liquid stools/day**
   Weighted ×2 — the most heavily scored parameter
2. **Abdominal pain (0–3)**
   Averaged daily over 7 days, weighted ×5
3. **General wellbeing (0–4 scale)**
   Averaged daily, weighted ×7
4. **Extra-intestinal features + labs**
   Arthritis, uveitis, skin lesions, fever, anemia, fistulae

Interpreting the CDAI Score

Score	Disease Activity
< 150	Remission
150–219	Mild disease
220–450	Moderate disease
> 450	Severe disease

A decrease of ≥100 points = clinical response. CDAI <150 = clinical remission.

Surgical Management of Crohn's Disease

Surgery is required in approximately **70–80% of Crohn's patients** within 10 years of diagnosis, typically for complications rather than failure of medical therapy alone. Surgery is not curative — disease recurrence at anastomotic sites is common.

Indications for Surgery

- Bowel obstruction unresponsive to medical therapy
- Perforation or peritonitis
- Uncontrolled hemorrhage
- Abscesses not amenable to percutaneous drainage
- Complex fistulae refractory to medical therapy
- Dysplasia or malignancy

Surgical Procedures

- **Strictureplasty** — bowel-sparing; widens stricture without resection
- **Segmental resection** — limited resection with anastomosis
- **Ileocecal resection** — most common procedure
- **Proctocolectomy** — reserved for severe colorectal involvement

⚠ Bowel-conserving surgery is preferred to avoid short bowel syndrome from repeated resections.

Key Takeaways — Crohn's Disease Summary

01

Definition & Distribution

Chronic transmural granulomatous inflammation; any segment from mouth to anus; most commonly terminal ileum; skip lesions are hallmark.

02

Pathomechanism

Genetic susceptibility (NOD2) + environmental triggers → dysregulated Th1/Th17 immune response → chronic transmural inflammation and granuloma formation.

03

Symptoms

RLQ pain, chronic diarrhea, perianal disease, weight loss, fever, plus extra-intestinal manifestations (uveitis, erythema nodosum, spondyloarthropathy).

04

Diagnosis

Ileocolonoscopy + biopsy (non-caseating granulomas), MRI enterography, CRP, fecal calprotectin, CDAI scoring for disease activity.

05

Complications & Treatment

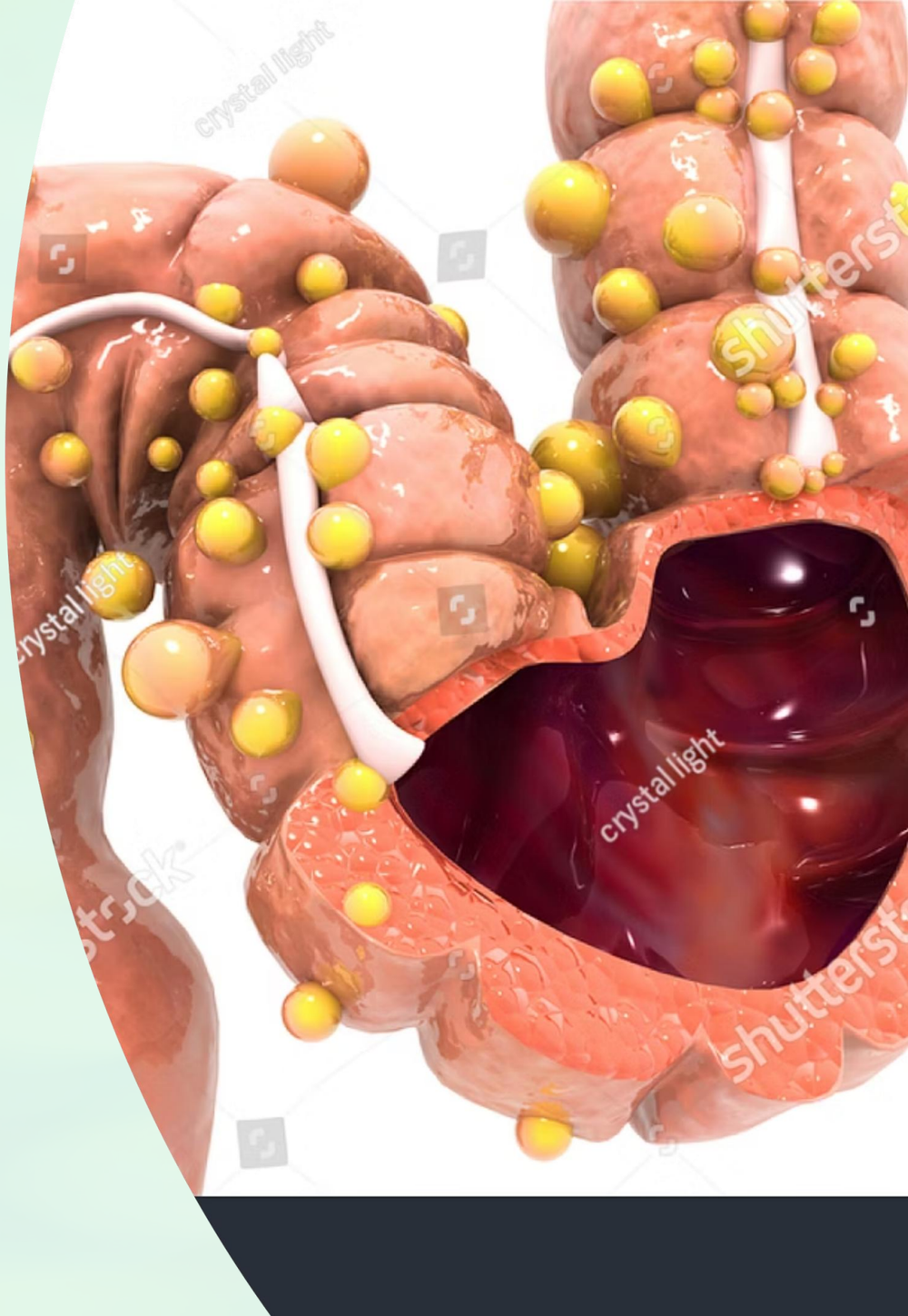
Strictures, fistulae, abscesses, malnutrition, cancer risk. Medical: corticosteroids, immunomodulators, biologics. Surgical in 70–80% within 10 years.

Ulcerative Colitis

A comprehensive clinical overview covering etiology, pathomechanism, symptoms, diagnostics, and complications — for medical students and healthcare professionals.

INFLAMMATORY BOWEL DISEASE

CLINICAL SUMMARY



Definition & Overview

What is Ulcerative Colitis?

Ulcerative colitis (UC) is a chronic inflammatory bowel disease characterized by diffuse mucosal inflammation of the colon and rectum. The inner lining — the **mucosa** — becomes inflamed and develops ulcers, most severely in the rectal area.

Key Classification by Extent

40–50%

Proctitis / rectosigmoid disease only

30–40%

Disease extending beyond the sigmoid

20%

Total colitis (pancolitis)

Proximal spread occurs **in continuity** — there are no skip lesions or areas of uninvolved mucosa, distinguishing UC from Crohn's disease.

Etiology & Risk Factors

Genetic Predisposition

Multiple susceptibility loci identified. First-degree relatives have a 10–15× higher risk. Concordance in monozygotic twins ~10–15%, suggesting environmental co-factors are essential.

Autoimmune Dysregulation

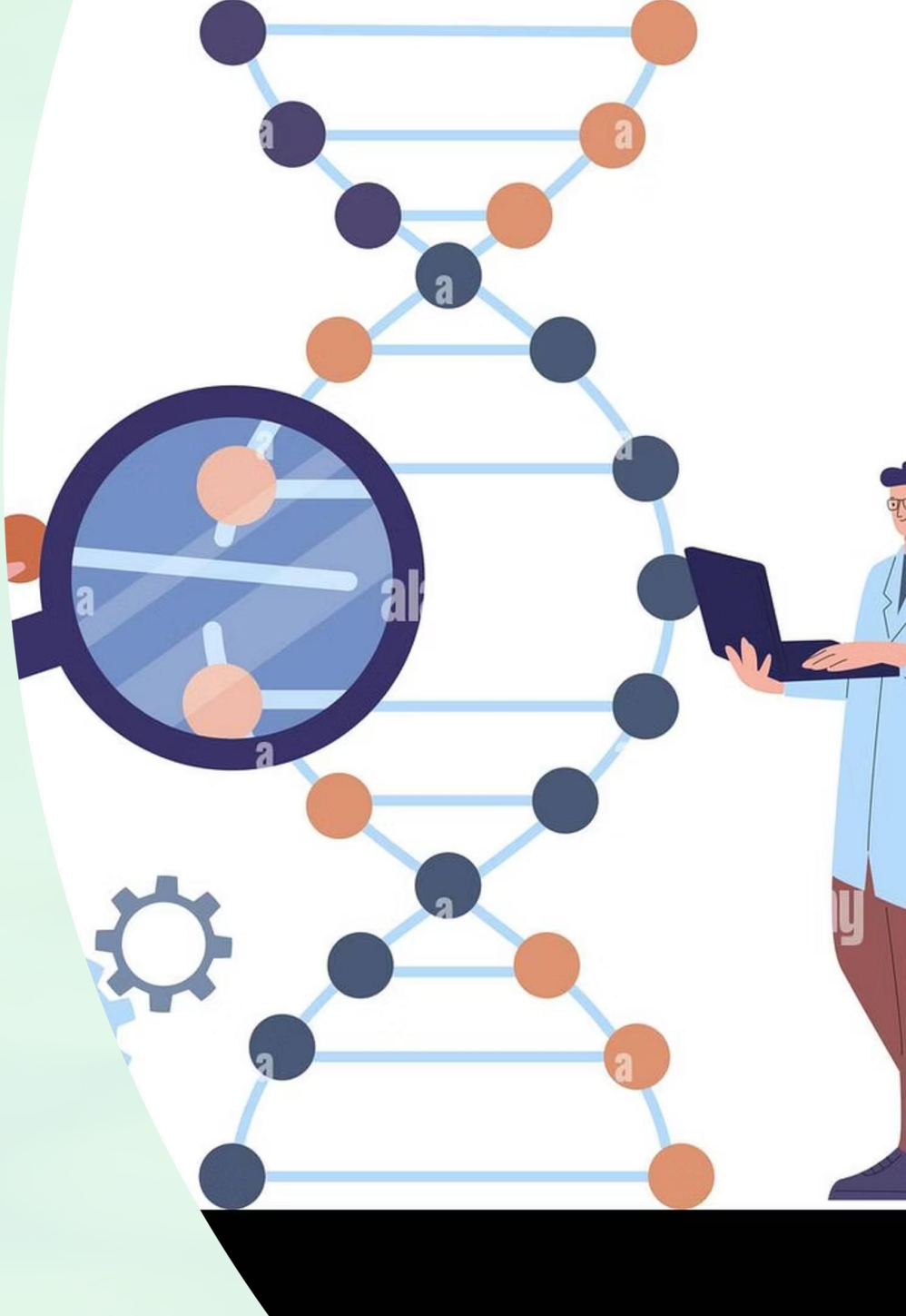
Aberrant mucosal immune response to luminal antigens. T-helper cell imbalance (Th2-predominant in UC) leads to cytokine-driven mucosal damage and loss of epithelial barrier integrity.

Environmental Factors

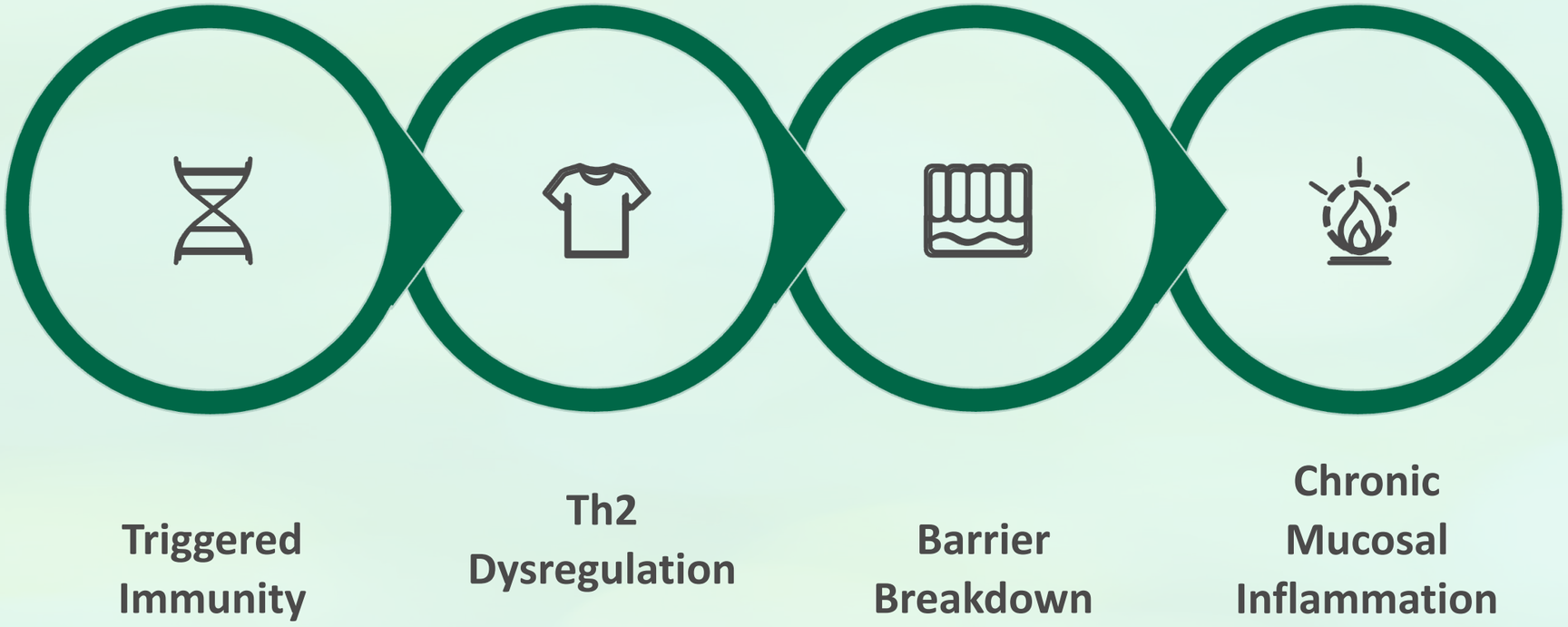
Low dietary fiber, Western diet patterns, antibiotic use, and stress are implicated triggers. Breastfeeding is a recognized protective factor. Appendectomy before age 20 may also be protective.

Gut Microbiome Dysbiosis

Disrupted composition of intestinal microbiota reduces mucosal tolerance. Decreased diversity and reduced short-chain fatty acid–producing bacteria contribute to chronic mucosal inflammation.



Pathomechanism of UC



The core mechanism involves a failure of immune tolerance to commensal bacteria in genetically susceptible individuals. Unlike Crohn's disease, the inflammatory process in UC is confined to the **mucosa and submucosa**, never extending transmurally. This distinction has major implications for disease complications and surgical management.

Microscopic Pathology

Histologic Features

The two hallmark microscopic findings that distinguish UC from infectious or other colitides are:

Crypt Architectural Distortion

Branching, shortening, and irregular spacing of crypts. Crypt atrophy and loss of goblet cells (mucin depletion) indicate chronicity. This is the most reliable diagnostic marker on biopsy.

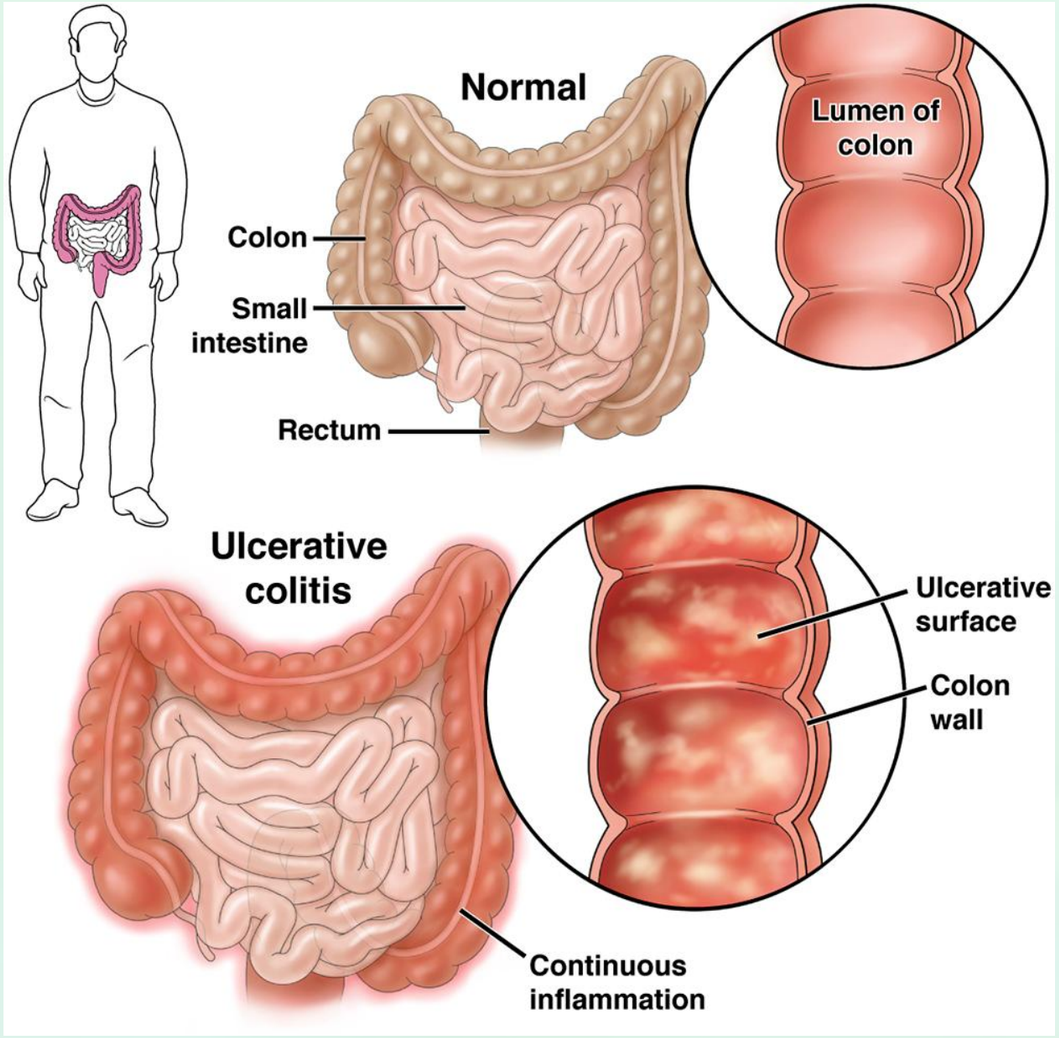
Basal Plasmacytosis & Lymphoid Aggregates

Basal plasma cells and multiple basal lymphoid aggregates beneath the crypts are early and specific features of IBD, often present before crypt distortion becomes obvious.

Active Inflammation Markers

- Cryptitis — neutrophilic infiltration of crypt epithelium
- Crypt abscesses — neutrophil accumulation within crypt lumina
- Lamina propria edema and congestion
- Increased eosinophils and mast cells

Biopsy remains essential for diagnosis. Multiple biopsies from different colonic segments are required to assess extent and exclude Crohn's disease.



Macroscopic Pathology

1

Mild Disease

Mucosa is **erythematous** with a granular, sandpaper-like surface. Loss of normal vascular pattern. Fine mucosal granularity on endoscopy.

2

Moderate Disease

Mucosa becomes thickened. **Superficial collar-button ulcers** appear. Loss of haustration is visible on imaging. Contact bleeding on endoscopy.

3

Severe Disease

Mucosa is **hemorrhagic, edematous, and deeply ulcerated**. Pseudopolyps may form from regenerating mucosa surrounded by ulceration.

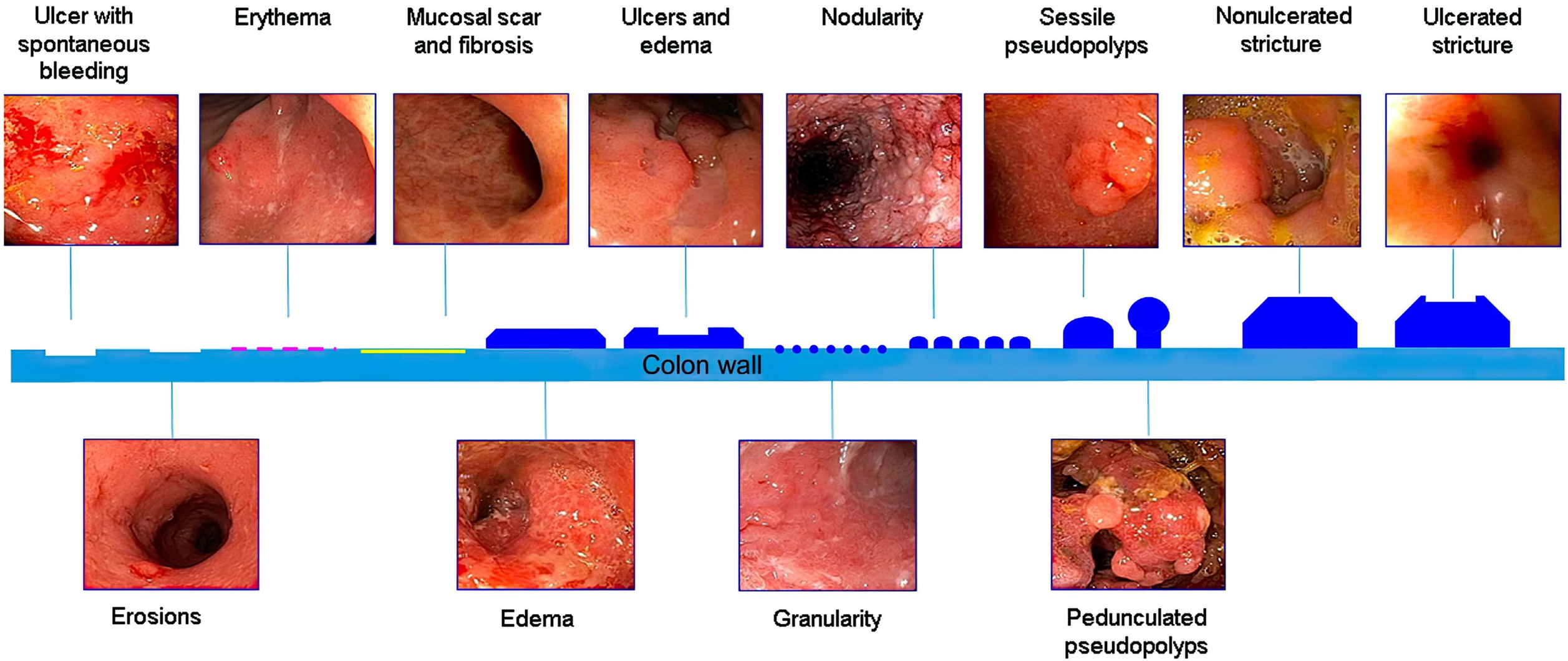
4

Fulminant Disease

Toxic colitis or **toxic megacolon**. The bowel wall becomes very thin, mucosa is severely ulcerated, and the colon is at risk of perforation.

Ulcerative Colitis Colonoscopy

Endoscopic Findings in Active Ulcerative Colitis



[Ulcerative Colitis Colonoscopy]

Clinical Symptoms

Core Gastrointestinal Symptoms

→ **Diarrhea with Blood or Mucus**

Blood is almost always present — either visibly mixed with stool or detected as occult blood. Patients with proctitis pass fresh blood or blood-stained mucus streaked onto stool.

→ **Tenesmus & Urgency**

Painful straining with defecation and a constant sensation of incomplete evacuation — characteristic of rectal involvement.

→ **Crampy Abdominal Pain**

Vague lower abdominal discomfort or cramping, often relieved by defecation. Severe pain raises concern for complications.

Systemic Symptoms (Moderate–Severe)

- Fever and malaise
- Anorexia, nausea, vomiting
- Significant weight loss
- Impaired growth in children and adolescents

Oral Manifestation

Aphthous oral ulcers occur as an extraintestinal manifestation and may precede or accompany intestinal flares.

UC vs. Crohn's Disease — Symptom Comparison

Symptom	Ulcerative Colitis	Crohn's Disease
Diarrhea	Very common; may contain blood and mucus	Common; usually without visible blood
Rectal bleeding	Almost always present	Less common
Abdominal pain	Lower abdomen; relieved by defecation	Lower right abdomen; recurrent episodes
Constipation	Possible (rectal spasm reflex)	Possible (small bowel obstruction)
Weight loss	Moderate	Significant (10–20% of body weight)
Anal fistulas/ulcers	Almost never	Common early feature
Fever	With severe attacks	Low-grade; spiking with complications
Neurologic symptoms	No	May occur with GI symptoms

Extraintestinal Manifestations — Dermatologic

Erythema Nodosum (EN)

Occurs in **up to 15% of Crohn's disease** patients and **10% of UC** patients. Lesions are hot, red, tender nodules up to 5 cm in diameter, located on the anterior legs, ankles, calves, thighs, and arms. Activity typically correlates with bowel disease activity.

Pyoderma Gangrenosum (PG)

Seen in **1–12% of UC patients** and less commonly in Crohn's colitis. PG may appear **years before** onset of bowel symptoms, making it a potential early marker. Lesions typically occur on the dorsal surface of feet and legs, but may affect arms, chest, and face. Lesions begin as pustules that rapidly enlarge into painful, necrotic ulcers.

⚠ Skin manifestations may precede intestinal symptoms. A diagnosis of PG or EN should prompt evaluation for underlying IBD.

Extraintestinal Manifestations — Rheumatologic & Ocular

Ocular

Peripheral Arthritis

Develops in **15–20% of IBD patients**; more common in Crohn's. It is **asymmetric, polyarticular, and migratory**, most often affecting large joints of the upper and lower extremities. Activity typically parallels bowel inflammation.

Ankylosing Spondylitis (AS)

Occurs in **~10% of IBD patients**. Characterized by sacroiliac joint inflammation progressing to spinal fusion. Course is **independent** of bowel disease activity. HLA-B27 positive in most cases.

Sacroiliitis

Symmetric inflammation of sacroiliac joints. Occurs equally in UC and CD. Often **asymptomatic** and detected incidentally on imaging. Does not necessarily progress to AS.

Ocular Complications

Incidence **1–10%** in IBD. Most common: **conjunctivitis, anterior uveitis, episcleritis**. Symptoms include ocular pain, photophobia, blurred vision, and headache. Uveitis may be vision-threatening if untreated.

Extraintestinal Manifestations — Systemic

Urologic Complications

Most frequent genitourinary complications: **nephrolithiasis, ureteral obstruction, and fistulas**. Nephrolithiasis occurs in **10–20%** of Crohn's disease patients (oxalate stones from fat malabsorption). Ureteral obstruction may result from adjacent bowel inflammation or abscess.

Hepatobiliary

Pericholangitis, primary sclerosing cholangitis (PSC — more common in UC), and fatty liver. PSC is the most serious hepatic complication and increases cholangiocarcinoma risk.

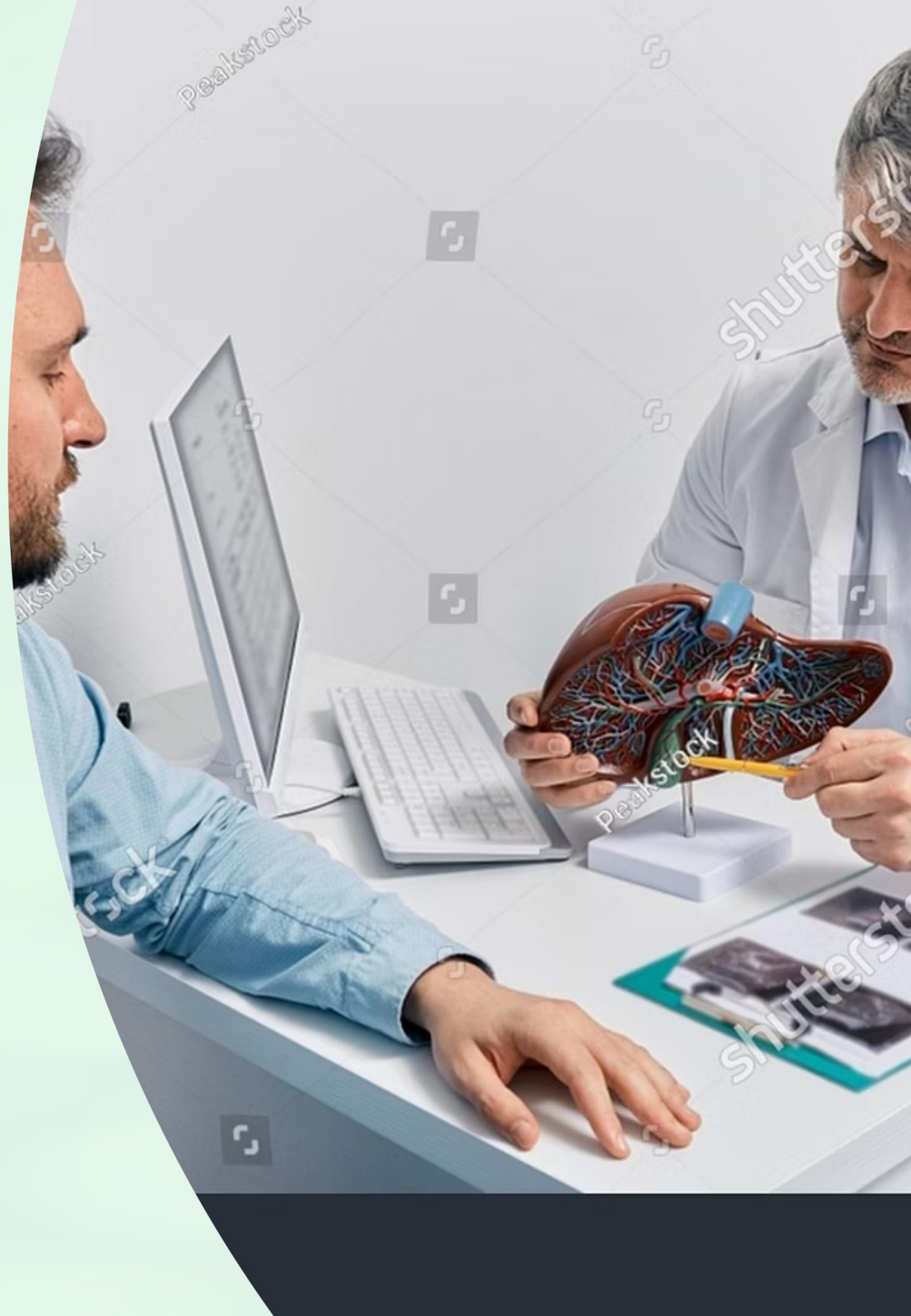
Metabolic Bone Disease

Increased prevalence of **osteoporosis** secondary to:

- Vitamin D deficiency
- Calcium malabsorption
- Chronic malnutrition
- Long-term corticosteroid use

Cardiopulmonary

Less common manifestations include **endocarditis, myocarditis, pleuropericarditis**, and interstitial lung disease. Thromboembolic disease (DVT, PE) is increased due to a hypercoagulable state during active disease.



Complications of Ulcerative Colitis

Hemorrhage

Severe lower GI bleeding requiring transfusion or emergency colectomy. Occurs in fulminant disease.

Perforation

Life-threatening surgical emergency. Risk is highest in toxic megacolon. Requires immediate colectomy.

Stricture

Fibrotic narrowing after chronic inflammation. Any stricture in UC must be evaluated to exclude dysplasia or carcinoma.

Toxic Megacolon

Transverse colon diameter >5–6 cm with loss of haustration. Bowel wall thins severely; mucosa is extensively ulcerated. Risk of perforation and sepsis is high.

Colorectal Cancer

Risk increases with disease duration and extent. After 10 years of pancolitis, CRC risk rises significantly. Annual surveillance colonoscopy is recommended.

Toxic Megacolon — A Critical Complication

Definition

Toxic megacolon is defined as acute non-obstructive colonic dilatation with a **transverse colon diameter >5–6 cm** combined with systemic toxicity. It is the most feared acute complication of UC.

Precipitating Factors

- Hypokalemia and hypomagnesemia
- Anticholinergic or opioid drugs
- Colonoscopy or barium enema during active disease
- Sudden cessation of therapy

Clinical Presentation

- High fever (>38.6°C), tachycardia
- Abdominal distension and tenderness
- Leukocytosis (>10,500/mm³)
- Anemia requiring transfusion

Management Principles

- IV corticosteroids (first-line)
- IV cyclosporine or infliximab rescue therapy
- Bowel rest, nasogastric decompression
- Emergency colectomy if no improvement in 48–72 hours

⊗ Perforation in the setting of toxic megacolon carries a mortality rate exceeding 40%. Early surgical consultation is mandatory.

Diagnostic Approach

Laboratory Tests

CBC, CRP, ESR, calprotectin, stool culture.

Endoscopy & Biopsy

Colonoscopy with targeted histologic sampling.

Clinical Assessment

History, symptoms, and physical exam.

Imaging

Abdominal X-ray or CT for complications.

Diagnosis of UC requires integration of clinical findings, laboratory results, endoscopic appearances, and histopathology. Stool cultures must always be obtained to exclude infectious colitis before commencing immunosuppressive therapy. No single test is pathognomonic for UC.

Laboratory Investigations

1

Complete Blood Count (CBC)

Anemia (iron deficiency from chronic bleeding), leukocytosis in active or complicated disease, thrombocytosis as an acute-phase response marker.

2

Inflammatory Markers

CRP and ESR correlate with disease activity. Fecal calprotectin is a sensitive, non-invasive marker of mucosal inflammation — useful for monitoring and predicting relapse.

3

Stool Studies

Stool culture, C. difficile toxin assay, and microscopy must be performed to exclude infectious etiology. CMV colitis must be excluded in steroid-refractory disease.

4

Serology

pANCA (perinuclear anti-neutrophil cytoplasmic antibody) is positive in ~60–70% of UC patients. ASCA (anti-Saccharomyces cerevisiae antibody) is more associated with Crohn's disease. Useful adjuncts but not diagnostic alone.

Endoscopy & Histology

Colonoscopic Findings by Severity Severity

Mild

Mucosal erythema, fine granularity, loss of vascular pattern, mild friability

Moderate

Marked erythema, coarse granularity, contact bleeding, superficial ulcerations (collar-button ulcers)

Severe

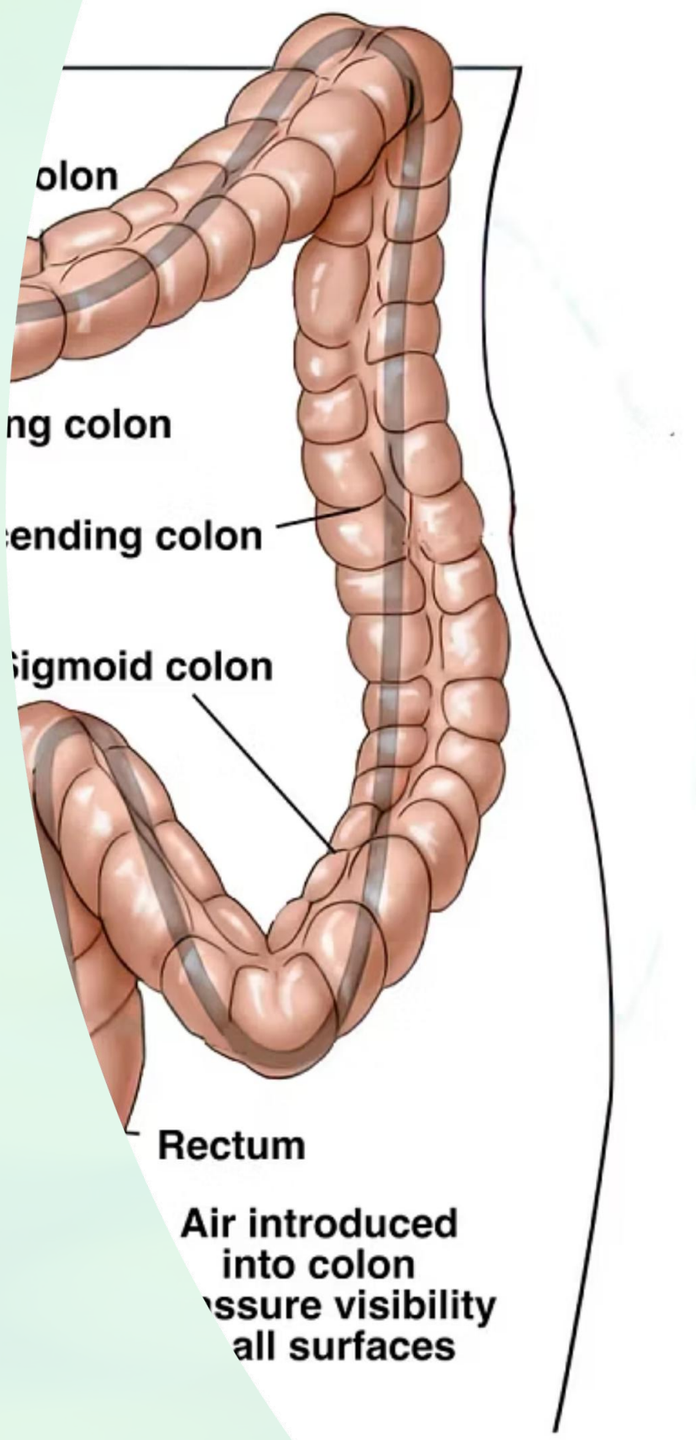
Spontaneous bleeding, deep ulcerations, exudate, pseudopolyps (inflammatory polyps)

Biopsy Protocol

Biopsies from at least 5 colorectal segments including the terminal ileum are recommended to map disease extent and exclude Crohn's disease.

Key Histologic Criteria for Diagnosis

- Crypt architectural distortion (branching, shortening)
- Basal plasmacytosis
- Cryptitis and crypt abscesses
- Mucin depletion from goblet cells
- Inflammation confined to mucosa/submucosa



Imaging in Ulcerative Colitis

Plain Abdominal X-Ray

First-line imaging in acute severe UC. Key findings:

- Loss of haustration — "lead pipe" appearance of the colon
- Mucosal thickening and thumbprinting
- Colonic dilatation — screen for toxic megacolon (>5–6 cm transverse colon)
- Free air under diaphragm — indicates perforation

CT Abdomen & Pelvis (with contrast)

Used in moderate-to-severe or complicated disease. Findings include:

- Mural thickening of the colon
- Pericolonic fat stranding
- Accordion sign in severe colitis
- Assessment for abscess, perforation, or malignancy

MRI Enterography

Preferred for evaluating extent of disease without radiation. Especially useful in pediatric patients and for differentiating UC from Crohn's disease by identifying transmural involvement or fistulas.

Barium enema and colonoscopy are **contraindicated** during suspected toxic megacolon due to the risk of perforation.

Disease Activity Assessment

≤2

Remission

Mayo score ≤2; no blood in stool; normal endoscopy

3–5

Mild–Moderate

Mild rectal bleeding, stool frequency <4/day, normal CRP

6–10

Moderate–Severe

Stool frequency >6/day, visible blood, elevated CRP, anemia

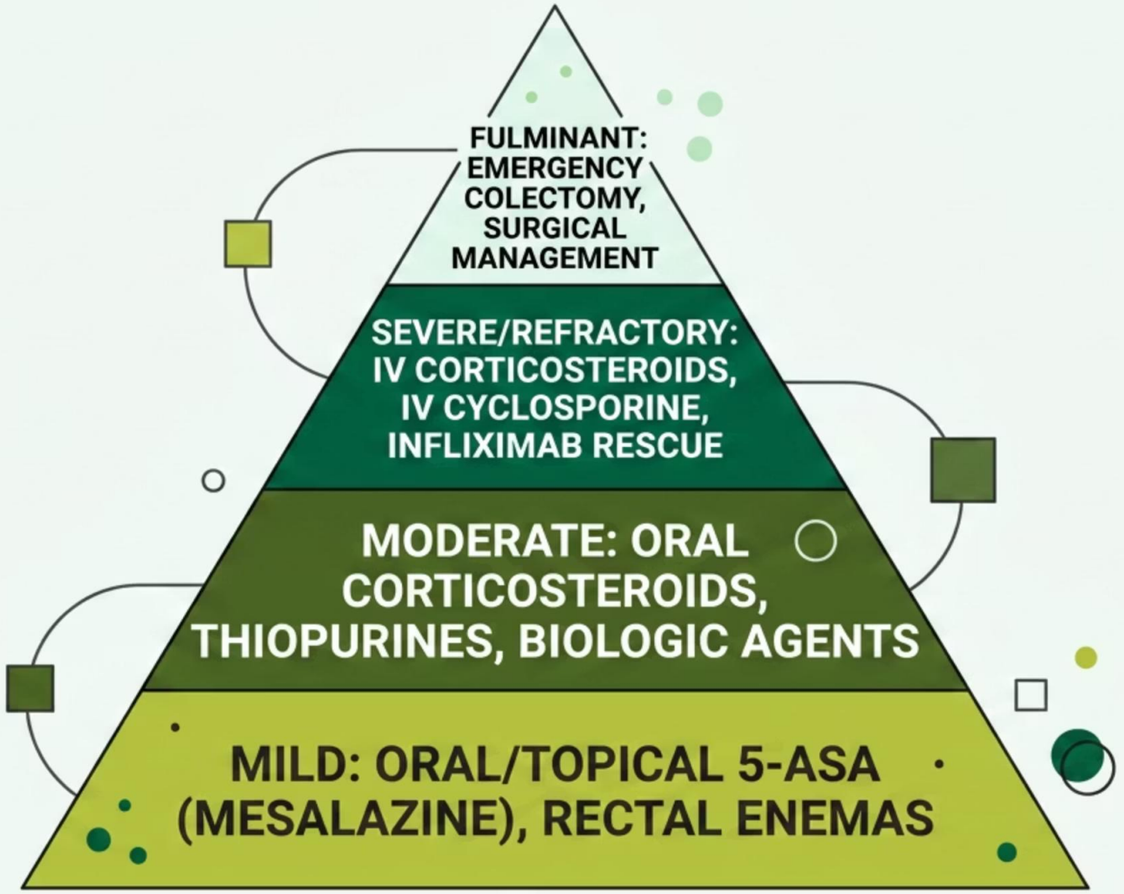
≥10

Fulminant

>10 bloody stools/day, fever, tachycardia, requires hospitalization

The Mayo Scoring System (0–12 points) integrates stool frequency, rectal bleeding, endoscopic findings, and physician global assessment. It is the most widely used index for clinical trials and routine practice. The Truelove and Witts criteria remain the gold standard for classifying acute severe UC requiring hospitalization.

Treatment Overview



Treatment strategy is guided by disease extent, severity, and prior response. The goal is to achieve and maintain mucosal healing. Biologics (anti-TNF agents, anti-integrins) have transformed the management of moderate-to-severe UC. Colectomy is curative in UC — unlike Crohn's disease.

Clinical Summary & Key Takeaways

Etiology

Multifactorial: genetic predisposition, autoimmune dysregulation, gut microbiome dysbiosis, and environmental triggers. Exact cause remains unknown.

Pathology

Continuous mucosal and submucosal inflammation from rectum proximally. No skip lesions. Crypt distortion and basal plasmacytosis are hallmark histologic features.

Symptoms

Bloody diarrhea, tenesmus, urgency, crampy pain. Systemic features in moderate-to-severe disease. Rich extraintestinal manifestations involving skin, joints, eyes, and liver.

Diagnostics

Colonoscopy with biopsy is gold standard. Supported by labs (CRP, calprotectin, pANCA), plain X-ray, and CT for complications. Exclude infection first.

Complications

Hemorrhage, perforation, stricture, toxic megacolon (>5–6 cm), and colorectal cancer (long-standing pancolitis). Surveillance colonoscopy is mandatory.

Treatment

5-ASA for mild disease; corticosteroids, immunomodulators, biologics for moderate-to-severe. IV therapy and surgery for fulminant disease. Colectomy is curative.

Image sources: Clinical endoscopic and dermatologic images used for educational purposes. Oral ulcer image sourced from course materials. All diagrams are original educational constructs.