

SUBJECT: PATHOLOGY

SYSTEM: GENERAL PATHOLOGY

TOPIC: INFLAMMATION

YEAR: 2

DOWNLOADABLE NOTES ON INFLAMMATION
For 2nd Year Dental Students

I. OVERVIEW OF INFLAMMATION

Definition

- Protective response to **eliminate injurious agents** and **remove damaged tissue**
- Involves **vascular, cellular, and systemic components**

Cardinal Signs

Sign	Latin Term	Cause
Redness	Rubor	Vasodilation ↑ blood flow
Heat	Calor	Vasodilation ↑ blood flow
Swelling	Tumor	Increased vascular permeability → edema
Pain	Dolor	Chemical mediators (bradykinin, PGs) + pressure on nerves
Loss of Function	Functio laesa	Pain + swelling

II. ACUTE vs CHRONIC INFLAMMATION

Feature	Acute	Chronic
Duration	Short (minutes-days)	Long (weeks-years)
Onset	Rapid	Insidious
Primary Cells	Neutrophils	Macrophages, Lymphocytes, Plasma cells
Tissue Injury	Usually mild, reversible	Often severe, progressive
Fibrosis	Minimal	Prominent (scarring)
Local Signs	Prominent	Less prominent

III. ACUTE INFLAMMATION - VASCULAR EVENTS

Triple Response

1. **Vasoconstriction** (transient, seconds)
2. **Vasodilation** → ↑ blood flow (rubor, calor)
3. **Increased Permeability** → edema (tumor)

Patterns of Exudate

Type	Character	Clinical Example
Serous	Clear, watery fluid	Blister, viral infections

Type	Character	Clinical Example
Fibrinous	Rich in fibrin	Pericarditis, pneumonia
Purulent	Pus (neutrophils + debris)	Abscess, bacterial infection
Hemorrhagic	Blood + inflammatory cells	Severe bacterial infections

IV. ACUTE INFLAMMATION - CELLULAR EVENTS

Leukocyte Recruitment Steps

1. **Margination** → move to periphery
2. **Rolling** → selectins (P/E-selectin)
3. **Adhesion** → integrins (LFA-1 → ICAM-1)
4. **Transmigration** → PECAM-1 (CD31)
5. **Chemotaxis** → follow chemical gradient
6. **Phagocytosis** → engulfment + destruction

Key Chemical Mediators

Mediator	Main Actions
Histamine	Vasodilation, ↑ permeability (immediate)
Prostaglandins	Vasodilation, pain, fever
Leukotrienes	↑ permeability, chemotaxis
Cytokines (IL-1, TNF)	Fever, systemic effects
Complement (C5a)	Chemotaxis, ↑ permeability

V. CHRONIC INFLAMMATION

Causes

- Persistent infections (TB, fungi)
- Autoimmune diseases
- Prolonged exposure to toxins
- Foreign bodies

Key Cells & Functions

Cell Type	Primary Function
Macrophages	Phagocytosis, antigen presentation, cytokine release
Lymphocytes	T-cells (cellular immunity), B-cells (antibodies)
Plasma Cells	Antibody production
Giant Cells	Form when macrophages fuse around foreign material

VI. GRANULOMATOUS INFLAMMATION

Definition

- Special type of chronic inflammation
- Focal collections of **epithelioid macrophages** ± **giant cells**

Types of Giant Cells

- **Foreign Body:** Haphazard nuclei
- **Langhans:** Horseshoe-shaped nuclei (TB, sarcoid)
- **Touton:** Lipid-laden, ring of nuclei

Causes

- **Infectious:** TB, leprosy, syphilis, fungi
 - **Non-infectious:** Sarcoidosis, Crohn's, foreign bodies
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VII. CLINICAL PATTERNS & DENTAL RELEVANCE

Abscess Formation

- **Definition:** Localized collection of pus
- **Dental Example:** Periapical abscess from necrotic pulp
- **Management:** Drainage + eliminate source

Ulcer

- **Definition:** Local defect of epithelium
- **Dental Example:** Aphthous ulcer
- **Features:** Loss of surface epithelium, fibrinopurulent exudate

Cellulitis

- **Definition:** Spreading infection in connective tissue
 - **Dental Example:** Ludwig's angina
 - **Risk:** Can spread to deep neck spaces
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VIII. SYSTEMIC EFFECTS

Manifestations

- **Fever** (pyrogens → hypothalamus)
 - **Leukocytosis** ↑ WBC count
 - **Increased ESR/CRP**
 - **Malaise, anorexia, weight loss**
 - **Sepsis** (systemic inflammatory response)
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IX. MINI CLINICAL CASES

Case 1: Acute Pulpitis → Abscess

- **Presentation:** 25F with severe, throbbing tooth pain #30, sensitive to percussion, buccal swelling.
- **Diagnosis:** Acute periapical abscess from necrotic pulp.
- **Management:** Incision & drainage, RCT or extraction, antibiotics if systemic signs.

Case 2: Chronic Sinus Tract

- **Presentation:** 40M with recurrent "gum pimple" near #9 that drains pus periodically. Tooth non-vital.
- **Diagnosis:** Chronic periapical inflammation with draining sinus tract.
- **Management:** Root canal treatment to eliminate source.

Case 3: Foreign Body Reaction

- **Presentation:** 60F with non-healing extraction socket, tender nodule. History of tooth extraction 6 weeks ago.
- **Diagnosis:** Foreign body granuloma (suture material/bone chip).
- **Management:** Excision + remove foreign material.

Case 4: Pyogenic Granuloma

- **Presentation:** 30F with rapidly growing, red, bleeding gingival mass between #8-9. 16 weeks pregnant.
 - **Diagnosis:** Pyogenic granuloma (exaggerated healing response).
 - **Management:** Excision (often recurs during pregnancy).
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X. HEALING & REPAIR

Outcomes of Inflammation

1. **Complete Resolution** (minimal damage, capable of regeneration)
2. **Healing by Scarring** (substantial damage, fibrosis)
3. **Chronic Inflammation** (persistent stimulus)

Factors Affecting Healing

- **Local:** Blood supply, infection, foreign bodies
 - **Systemic:** Age, nutrition, diabetes, medications
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XI. PHARMACOLOGY CONNECTION

Anti-inflammatory Drugs

Drug Class	Mechanism	Dental Use
NSAIDs	Inhibit COX → ↓ prostaglandins	Pain, inflammation (ibuprofen)
Corticosteroids	Inhibit phospholipase A2 → ↓ all mediators	Severe inflammation, oral lesions

Drug Class	Mechanism	Dental Use
Antihistamines	Block H1 receptors	Allergies, pre-op anxiety

Revision Tips:

- Focus on distinguishing acute vs chronic inflammation
- Understand sequence from injury to healing
- Relate every concept to dental clinical scenarios
- Practice identifying cell types from descriptions

This guide covers key pathology concepts—use as foundation for clinical years.