GINGIVAL INFLAMMATION

- ***Gingivitis is the inflammation of gingiva***

- Pathologic changes of gingivitis
  - Associated with presence of oral microorganisms in the sulcus

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These organisms are capable of synthesizing products

↓ ***Causes***

Damage to:

- Epithelial & connective tissue cells
- Intercellular components
  - Collagen
  - Ground substance
  - Glycocalyx (cell coat)

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Resultant widening of the spaces b/w junctional epithelial cells during early gingivitis

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Permit injurious agents derived from:
Bacteria or Bacteria themselves

↓

Gain access to connective tissue

↓

Microbial products → Activate monocytes & Macrophages

↓

Produce Vasoactive substances

- Prostaglandin E_2
- Interferon
- Tumor necrosis factor
- Interleukin-1

SEQUENCE OF EVENTS IN GINGIVITIS:

1. Initial lesion
2. Early lesion
3. Established lesion
4. Periodontitis designated as the advanced lesion

STAGE I GINGIVITIS: THE INITIAL LESION

- Features characterizing the initial lesion merely reflect the enhanced levels of activity of the mechanisms of host defense that normally are operative within the gingival tissues
- This initial response of the gingiva is also called subclinical gingivitis
- The initial lesion is localized to the region of the gingival sulcus

Features

1.	**Classic vasculitis of vessels**
2.	Exudation of fluid from gingival sulcus
3.	Increased migration of leukocytes into the junctional epithelium & gingival sulcus
4.	Presence of serum proteins (especially fibrin extravascularly)
5.	Alteration of most coronal portion of junctional epithelium
6.	Loss of perivascular collagen

- Within the deeper regions of the junctional epithelium:
  - Numerous intact neutrophils
  - Other leukocytes may be present
- **Initial lesion is a response to generation of:**
  - Chemotactic substances
  - Antigenic substances in the region of the gingival sulcus

- Initial lesion emerges within **2 to 4 days**, when a previously normal, infiltrate-free gingiva is subjected to the accumulation of microbial plaque

STAGE II GINGIVITIS: THE EARLY LESION

- Early lesion overlaps & evolves from the initial lesion with no clear cut dividing line

Clinical signs of:

- Erythema may appear due to:
  - Proliferation of capillaries
  - Increased formation of capillary loops between rete pegs & ridges
- Bleeding on probing may also be evident

Features

1.	**Accentuation of features of initial lesion**
2.	Accumulation of lymphoid cells immediately subjacent to the junctional epithelium at the site of acute inflammation
3.	Cytopathic alterations in resident fibroblasts, possibly associated with interactions with lymphoid cells
4.	Further loss of the collagen fiber network supporting the marginal gingiva
5.	Beginning proliferation of the basal cells of the junctional epithelium

STAGE III GINGIVITIS: THE ESTABLISHED LESION

- Blood vessels - engorged & congested
- Venous return is impaired
- Blood flow becomes sluggish

Results → Localized gingival anoxemia → Superimposes a bluish hue on the reddened gingiva

- Distinguishing feature of the established lesion is:

Predominance of:

- B-lymphocytes
- Plasma cells

Within the affected connective tissues at a stage prior to extensive bone loss

Features

1. Persistence of the manifestations of acute inflammation
2. Predominance of plasma cells but without appreciable bone loss
3. ***Presence of immunoglobulins extravascularly in the connective tissues and in junctional epithelium***
4. Continuing loss of connective tissue substance noted in the early lesion
5. Proliferation, apical migration, & lateral extension of the junctional epithelium
6. Early pocket formation may or may not be present

- Lesion apparently has a high degree of organization, and plasma cells are located in its periphery.
- **Increased levels of:**
  - Acid phosphatase
  - Alkaline phosphatase
  - β-glucuronidase
  - β-glucosidase
  - β-galactosidase
  - Esterases
  - Aminopeptidase
  - Cytochrome oxidase

- **Decreased levels of** mucopolysaccharide levels as a result of degradation of the ground substance

- **Established lesions - Two types** apparently exist:

Stable lesions (Do not progress for months /years)

Active Lesions → ***convert to*** → **Progressive destructive lesions**

- Proportion of T cells decreases
- B cells & plasma cells increases

As

Gingivitis appears & increases in severity

- **Connective tissue infiltrate, which may eventually form consists:**
  - Almost entirely of lymphocytes
    - Almost all of which are T cells
    - Few B-lymphocytes
  - Macrophages are present
  - Very few neutrophils
  - Fewer plasma cells are apparent

- Proportion of the inflammatory cell infiltrate accounted for by B cells & plasma cells increases:
  - With time
  - With the increasing degree of inflammation in adults
  - This relationship does not seem to occur in children

- The lack of B-lymphocytes & plasma cells in the gingival tissues in children may be related to the absence of a gingival pocket

STAGE IV GINGIVITIS: THE ADVANCED LESION

- Persistence of features described for the established lesion
- Extension of the lesion into the alveolar bone & periodontal ligament with significant bone loss
- Continued loss of collagen subjacent to the pocket epithelium with fibrosis at more distant sites
- Presence of cytopathically altered plasma cells in the absence of altered fibroblasts
- Formation of periodontal pockets
- Periods of quiescence & exacerbation
- Advanced lesion represents frank & overt periodontitis
- Conversion of the bone marrow distant from the lesion into fibrous connective tissue
- Widespread manifestations of inflammatory & immunopathologic tissue reactions
- Plasma cells predominate in the lesion, although lymphocytes and macrophages are also present
- Signs of acute vasculitis persist in the presence of chronic fibrotic inflammation
- Lesion is no longer localized
  - May extend apically
  - As well as laterally

  To form a variably broad band around the necks & roots of the teeth

STAGES OF GINGIVITIS

Stage	Time (Days)	Blood Vessels	Junctional and Sulcular Epithelia	Predominant Immune Cells	Collagen	Clinical Findings
I. Initial lesion	2–4	Vascular dilation Vasculitis	Infiltration by PMNs	PMNs	Perivascular loss	Gingival fluid flow
II. Early lesion	4–7	Vascular proliferation	Same as stage I Rete pegs Atrophic areas	Lymphocytes	Increased loss around infiltrate	Erythema Bleeding on probing
III. Established lesion	14–21	Same as stage II, plus blood stasis	Same as stage II but more advanced	Plasma cells	Continued loss	Changes in color, size, texture, etc.