Lecture of

Dentin Caries

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Preface:

- Dentin caries is a critical stage in tooth decay, occurring once the lesion extends beyond the DEJ.
- Dentin caries cause sensitivity, pain, and increase the risk of pulpal involvement.
- Dentin can perform defense mechanism by deposition of tertiary and sclerotic dentin.

Satges of Enamel Caries

1. **The early lesion**
(Submicroscopic)

2. **Non-bacterial**
(Enamel Caries)

3. **Cavitation**
(Enamel Caries)

4. **Bacterial**
(Enamel caries)

5. **Bacterial**
(dentin caries)

6. **Undermining enamel**
(dentin caries)

Factors Affecting The Spread of Dentin Caries

1. Dentin is vital connective tissue. **Can perform defense mechanism.**

2. Dentin is less mineralized and higher organic than enamel.

65% inorganic + 35% organic (mainly collagen).

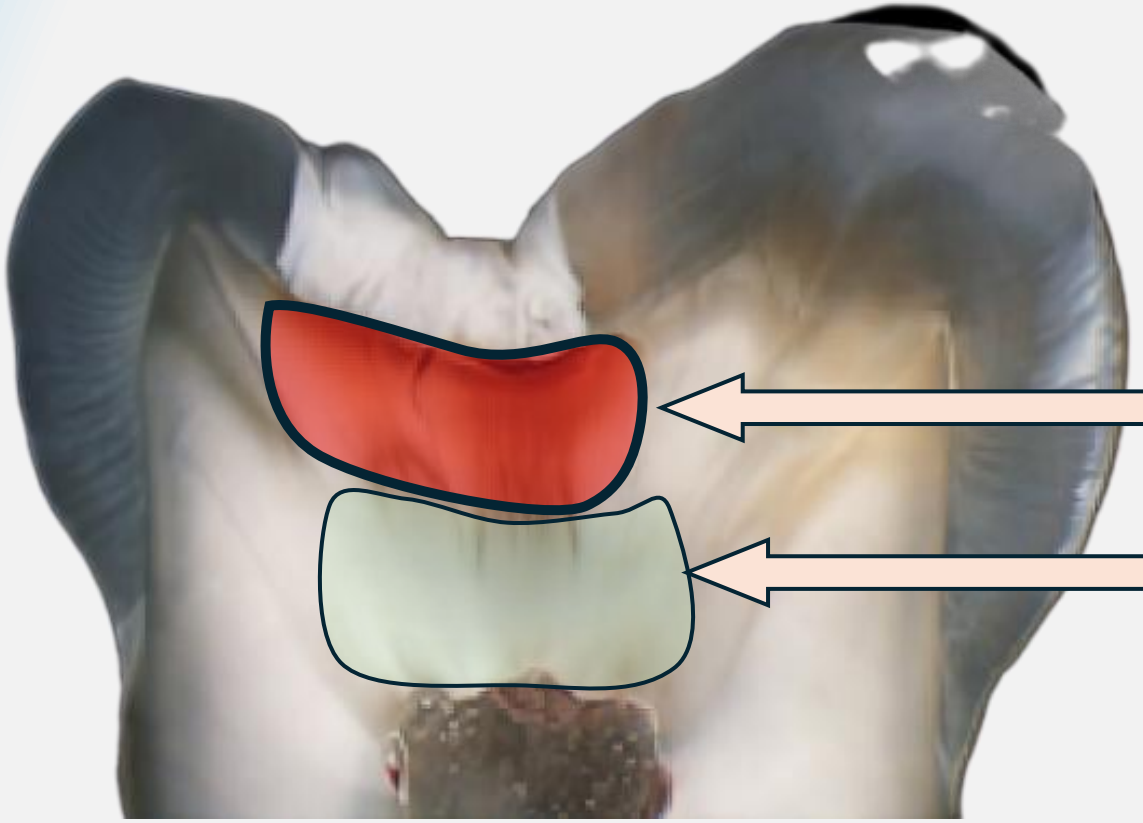
3. Dentin is highly permeable by the hollow dentinal tubules.

Increase the chance of bacterial invasion and colonization.

4. Dentin is a sensitive tissue. **Pain is an alarm defense mechanism**

5. Dentin is supported by the pulp. **Easy to do the protective action.**

Zones of Carious Dentin



Destructive dentin by the action of caries

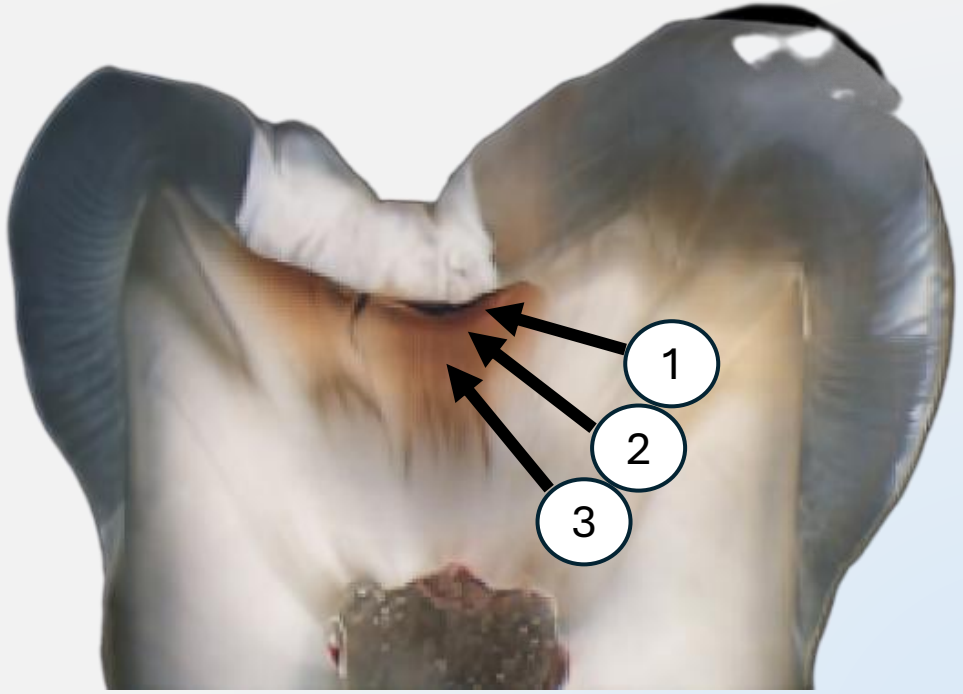
Dentin protection by the action of pulp

I. Zones of Dentin Disrtuction

- These zones represent the progression of the caries toward the pulp.
- Characterized by destruction of dentin structure.
- Changes are mainly pathological and irreversible (especially outer layers).

These zones include:

1. Zone of demineralization.
2. Zone of decomposition (necrotic dentin).
3. Zone of bacterial invasion.
4. Next zone of demineralization.

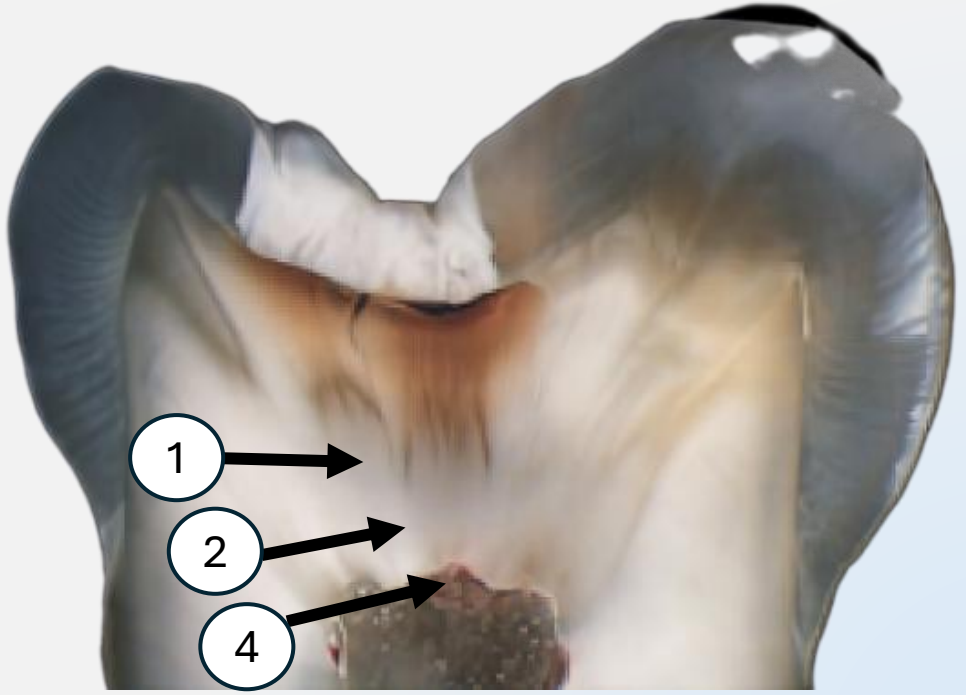


II. Zones of Dentin Protection

- These zones represent the tooth's response to irritation.
- Aims to protect the pulp and slow caries progression.
- Changes are protective or degenerative in nature.

These zones include:

1. Zone of dead tracts.
2. Zone of dentinal sclerosis.
3. Zone of fatty degeneration of Tomes' fibers.
4. Zone of tertiary dentin.



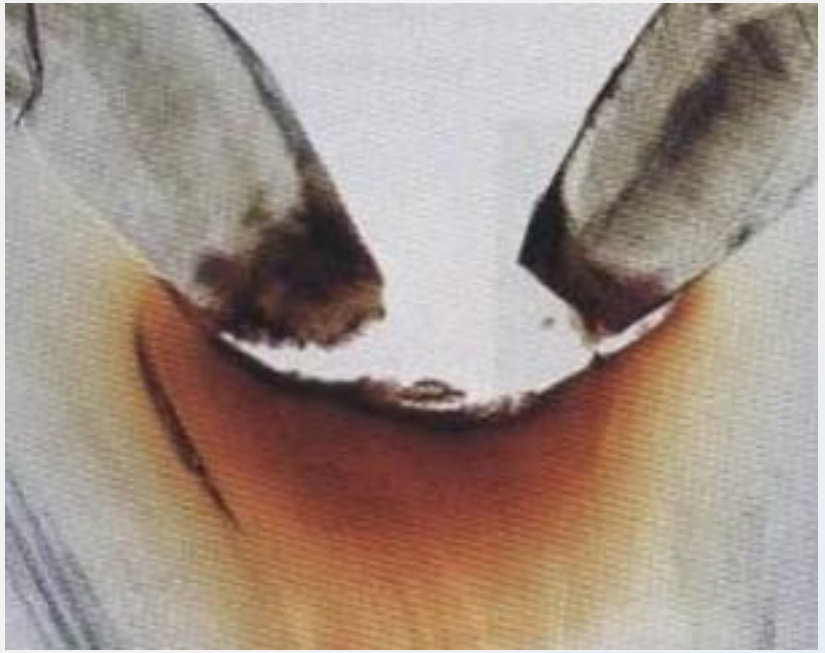
Early stage of dentin caries (Necrotic Dentin)

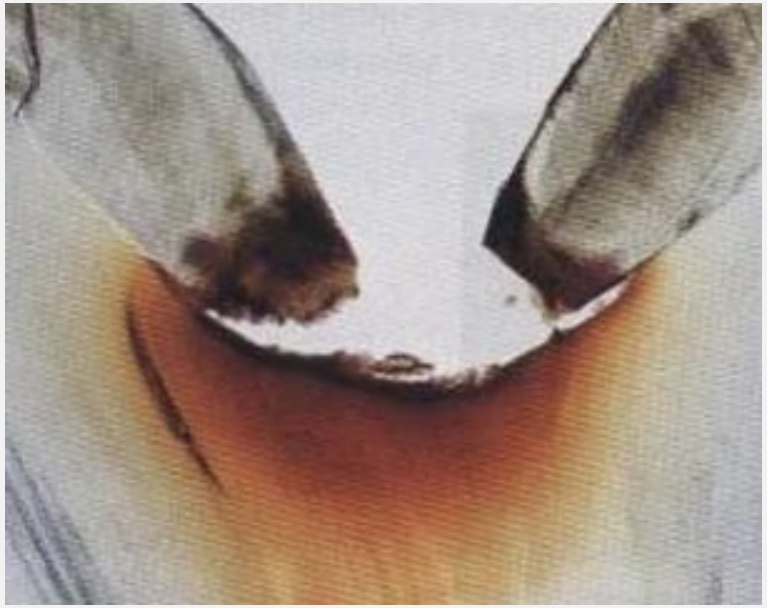
1. **Spread of enamel caries into dentin:**
   - Caries penetrate enamel and reach the dentinoenamel junction.
   - The dentin is less calcified and high organic, leads to rapid spread of the caries into:
     a) Inward spread toward the pulp.
     b) Lateral spread under the unaffected enamel.
   - The enamel overlying the laterally spreading dentin caries become **undermined**.

2. **Early dentin caries:**
   - The initial layer of dentin caries is considered a mild (weak) irritant lesion.
   - Initially, the superficial dentin undergoes demineralization without bacterial invasion.
   - Then, fatty degeneration of Tomes' fibers occurred.
   - This layer of non-bacterial dentin caries is termed necrotic dentin.
   - Followed by sclerosis of DT by mineral deposition until complete occlusion.

Undermined Enamel

- Undermined enamel is enamel that has lost its underlying dentinal support due to carious destruction of dentin
- The surface enamel may appear intact initially.
- Loss of support makes the enamel structurally weak and able to fracture.





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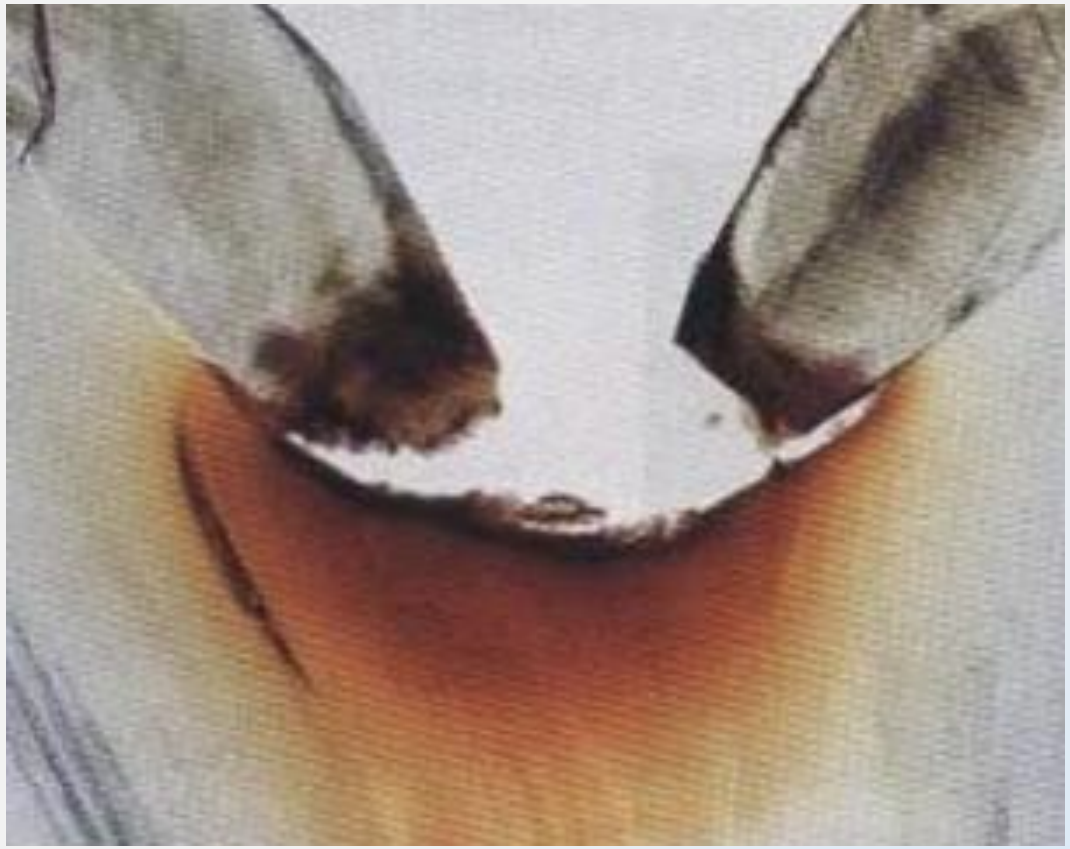
4. Next zone of demineralization.

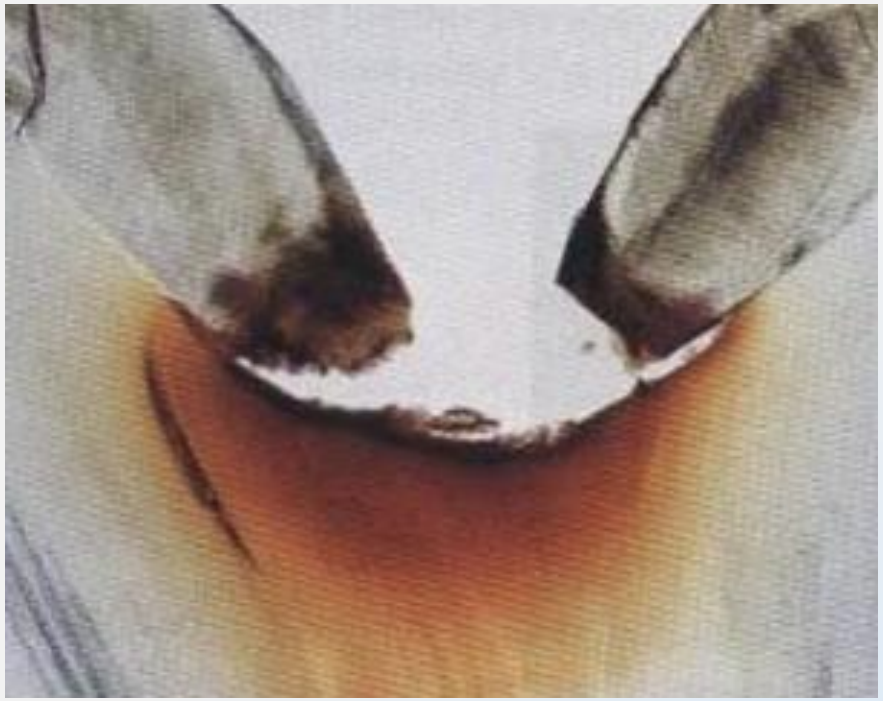
1. Zone of demineralization.

- Located just beneath the carious enamel.
- Characterized by loss of inorganic mineral (hydroxyapatite) by acids with no or minimal bacterial presence.
- Initially, the collagen fibers remains intact , providing a scaffold for possible remineralization.
- Dentinal tubules become widened due to mineral loss, increasing permeability for passage of microorganisms.
- Progression beyond this zone leads to bacterial invasion and destruction of the organic matrix.

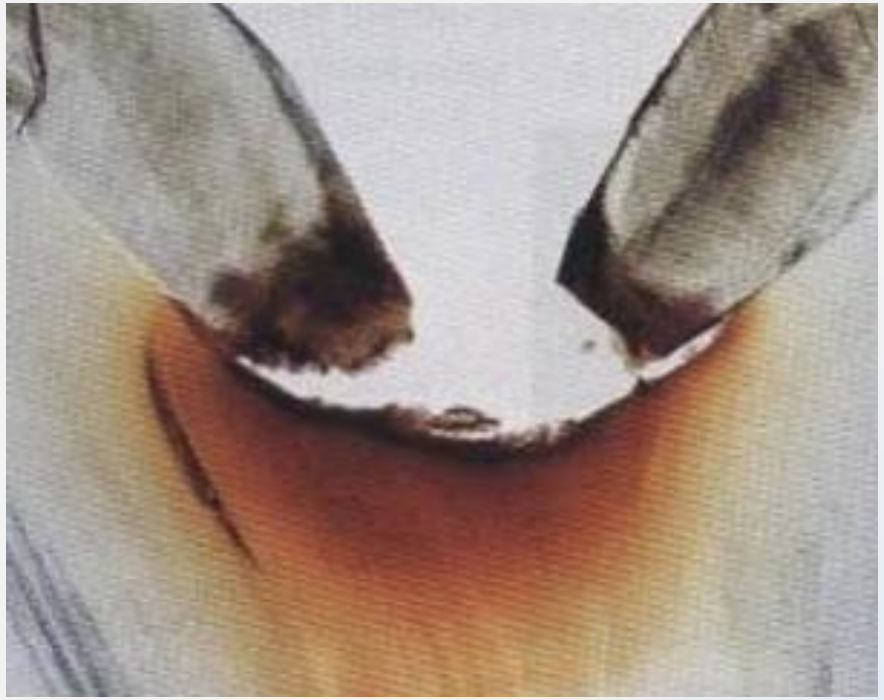


2. Zone of decomposition (necrotic dentin).
3. Zone of bacterial invasion.

- The bacteria inter the widened dentinal tubules.
- **Team of bacteria needed for dentin caries:**
    1) **Cariogenic bacteria (Lactobacilli)** for decalcification.
    2) **Proteolytic bacteria** for degradation of organic matrix and collagen fibers.
- The result is the necrotic organic material and cavitation.
- This cavitation allow more invasion of bacteria.
- The carious area become infected.



4. Next zone of demineralization.

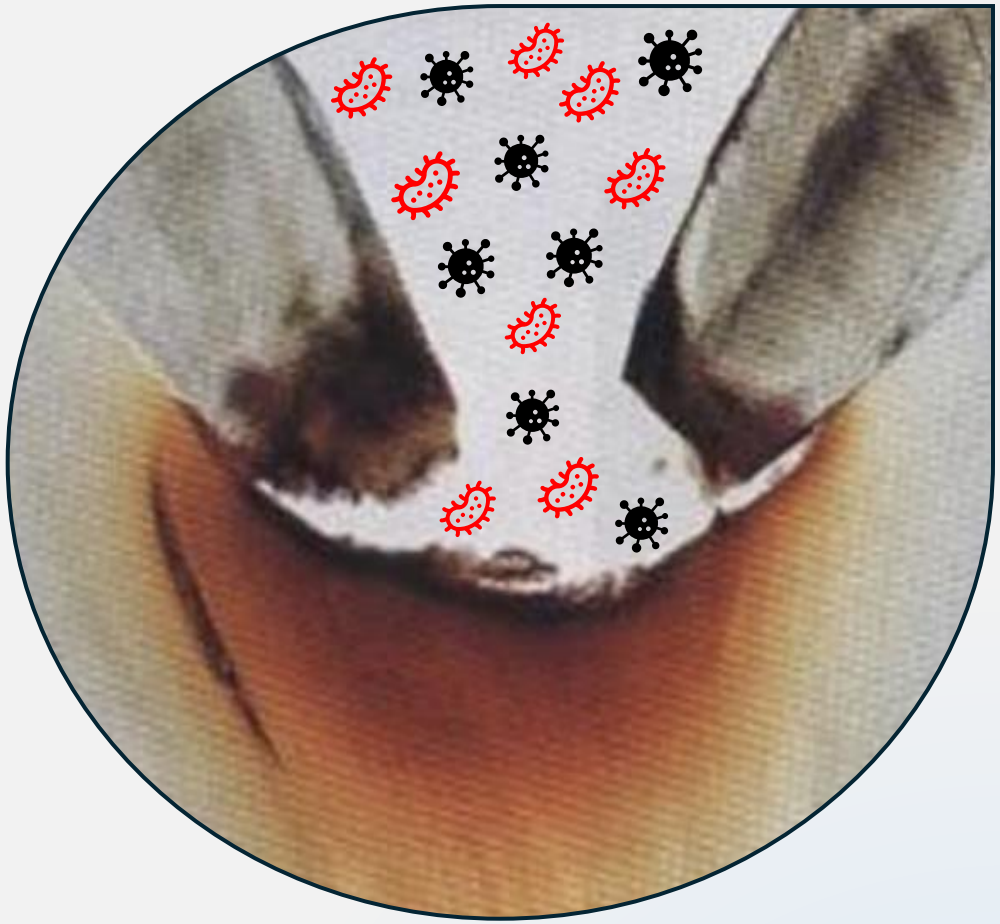


Progressive Destructive Actions of Bacteria in The Carious Dentin

1. Invasion of bacteria.
2. Bacterial proliferation.
3. Longitudinal Beading formation.
4. Liquefaction foci.
5. Transverse cleft formation.
6. ???????

1. **Bacterial invasion :**
    - Microorganisms penetrate dentinal tubules after enamel breakdown.
    - Bacteria follow the direction of dentinal tubules toward the pulp.

2. **Bacterial proliferation**
    - Rapid multiplication of bacteria within tubules.
    - Tubules become distended and packed with microorganisms.
    - Proteolytic activity begins to destroy the organic matrix of dentin.



3. Beading formation:

- Appears as bead-like area within each dentinal tubule.
- Caused by bacterial invasion and multiplication within the necrotic tubules.
- Contain accumulation of bacteria, toxins, and degraded organic components.
- Leads to irregular swelling of dead dentinal tubules.

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❖ **Indicates:**

1. Active progression of bacterial destruction.
2. Spread of infected caries toward the pulp.

4. Liquefaction Foci Formation

- Represents an advanced stage of dentin destruction by bacteria.
- Proteolytic bacteria break down the organic matrix of dentin.
- Infected dentinal tubules undergo degeneration and begin to **coalesce**.
- Formation of large cavities within dentin (liquefaction foci).
- These spaces are filled with bacteria, necrotic debris, and degraded organic components.

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Indicates:

- Weakens the structural integrity of dentin significantly.
- Further lateral and deeper spread of infection.

5. Transverse clefts formation

- Represents the more advanced stage of dentin caries.
- Caused by lateral spread of bacteria across dentinal tubules.
- Leading to coalescence of adjacent liquefaction foci.
- Results in clefts or cracks that run perpendicular to dentinal tubules.

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Leads to:

1. marked weakening of dentin structure.
2. Indicates severe and advanced carious involvement.