Oral pathology
Dental Caries (Tooth Decay)

• Dental caries is a chronic infectious disease of tooth enamel, dentin and cementum.
• It is a local process manifesting with demineralization and progressive destruction of dental tissues with the formation of defect (cavity).
Dental Caries (Tooth Decay)

• Caries is the most prevalent chronic disease of the calcified tissues of teeth, affecting both sexes and every age group.

• Its incidence has markedly increased with modern civilization.
Dental Caries (Tooth Decay)

• Teeth of upper jaw are most often affected:
  – first molars,
  – second molars,
  – premolars and upper incisors,
  – canines.
Dental Caries, etiologic factors

• Bacteria
• Saliva
• Dietary factors
• Fluoride
• Other factors:
  – general state of the organism,
  – genetic predisposition,
  – age,
  – violation of mineral and carbohydrate metabolism,
  – malnutrition, lack of vitamins.
Dental Caries, etiologic factors: bacteria

• Tooth surfaces are normally colonized by many microorganisms.
• Unless the surface is cleaned thoroughly and frequently, bacterial colonies coalesce into a soft mass known as dental plaque.
• *Streptococcus mutans* is the primary etiologic agent that initiates caries.
• Carious lesions result primarily from leaching of mineral in dental tissues by acids produced from food residues by microorganisms on tooth surfaces.
Dental Caries, etiologic factors: saliva

- Normally, saliva neutralizes microbially produced acids in the mouth.
- It contains bacteriostatic factors (lysozyme, lactoferrin, lactoperoxidases and secretory immunoglobulins).
- Xerostomia (chronic dryness of the mouth from lack of saliva) results in caries.
Dental Caries, etiologic factors: dietary factors

- One of the most important factors in the pathogenesis of caries is a high-carbohydrate diet.
- Roughage in raw and unrefined foods cleanses the teeth and necessitates more mastication, which further contributes to cleansing of the teeth.
- Soft and refined foods tend to stick to the teeth and also require less chewing.
Dental Caries, etiologic factors: fluoride

• Fluoride protects against dental caries.
• It is incorporated into the crystal lattice structure of enamel, where it forms fluorapatite, which is less acid soluble than is the apatite of enamel.
• Fluoridation of drinking water leads to reductions in dental caries.
Dental Caries, classification: ICD-10

<table>
<thead>
<tr>
<th>K02</th>
<th>Dental caries</th>
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<tbody>
<tr>
<td>K02.0</td>
<td>Caries limited to enamel</td>
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<tr>
<td>K02.1</td>
<td>Caries of dentin</td>
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<td>K02.2</td>
<td>Caries of cementum</td>
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<td>K02.3</td>
<td>Arrested dental caries</td>
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<td>K02.4</td>
<td>Odontoclasia</td>
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<tr>
<td>K02.8</td>
<td>Other dental caries</td>
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<tr>
<td>K02.9</td>
<td>Dental caries, unspecified</td>
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</tbody>
</table>
Dental Caries, anatomical classification

• Caries of enamel
• Caries of dentine
• Caries of cement
Dental Caries, clinico-topographical classification

- Initial (white, pigmented stain) – macula cariosa.
- Superficial caries – defect in the enamel.
- Middle caries – the lesion extends beyond the enamel-dentine connection and is located in the superficial layers of dentin.
- Deep caries – damage of deep layers of dentin (1.5 mm or less of dentin layer remains intact).
Initial caries

- is characterized by demineralization of sub-superficial layer of enamel without formation of defect in form of cavity.
- whitish or dark, opaque macula with accurate borders.
Initial caries
Initial caries

Enamel

Subsurface carious lesion

Striae of Retzius
(regions of demineralization)
Superficial caries

• is characterized by demineralization of all layer of enamel with formation of defect in form of a cavity within enamel.
• Disorientation of hydroxiapatite crystals, change of their shape. Defect is found in enamel-dentin border
• Changes in pulp are not observed.
Superficial caries
Middle caries

- is characterized by demineralization of enamel and a cloak layer of dentine with formation of defect within a cloak dentine in form of a cavity.
Middle caries
Middle caries
Middle caries, pathomorphology

At light microscopy four layers are seen:

1. Decay and demineralization of enamel and cloak dentine ("body of the lesion");
2. Translucent (calcified) dentine;
3. Intact dentine;
4. Replacement (substitutionary) dentine and changes in pulp.
Middle caries, layer of decay and demineralization (1)

• Remnants of destroyed enamel and dentine with large number of microorganisms are visible.

• The cloak dentine is softened, dentinal tubules are dilated or merged, forming microcavities filled with bacteria.
Middle caries, layer of translucent dentine (2)

- Layer of compacted dentine with considerably reduced dentinal canaliculi.
- This region is characteristic only for chronic caries.
- This region is named because of optical effect: due to calcification of the dentinal tubules dentin becomes translucent.
Layers of intact (3) and replacement dentine (4)

• At chronic middle caries the layer of near-pulpar dentine is intact.

• In the forth zone dentinal tubuli lack or misoriented. This zone is characteristic only for chronic current caries.
Deep caries

Three zones are characteristic:
1. Disintegration and demineralization;
2. The thin zone of intact dentine (sometimes is absent);
3. Changes in pulp.
Deep caries
Deep caries
Dental Caries, complications

Destruction of enamel and dentin allows the bacteria to enter the pulp and extend into the bone at the tip of the tooth. Typical complications:

• **Acute pulpitis:** infection in the central cavity of the tooth.
• **Apical abscess:** Bacteria extend from the pulp into the bone surrounding the root of the tooth. Pus may drain into the mouth along the lateral sides of the infected tooth.
• **Periapical granuloma:** granulation tissue that develops inside the healing periapical abscess.
• **Radicular cyst:** If the pus from an abscess is resorbed, a cavity remains. This initial pseudocyst (no epithelial lining) may be partially covered by ingrowths of gingival epithelium.
Non-carious lesions

- **Erosion** – irreversible loss of tooth structure due to chemical dissolution by acids not of bacterial origin.

- The most common cause – acidic foods and drinks.

- Foods and drinks with a pH below 5.0-5.7 have been known to trigger dental erosion effects.
Non-caries lesions, erosion
Non-curious lesions, erosion

• Intrinsic dental erosion (perimolysis) is due to contact of teeth with gastric acid from the stomach.
• Erosion can develop in persons who work in the production of inorganic acids (professional pathology).
Non-carious lesions

• **Abfraction** – non-carious cervical lesions (NCCL) caused by flexural forces, usually from cyclic loading.

As teeth flex under pressure, the arrangement of teeth touching each other causes tension on one side of the tooth and compression on the other side of the tooth. This is believed to cause V-shaped depressions on the side under tension and C-shaped depressions on the side under compression.
Non-carious lesions, abfraction
Non-carious lesions

- **Abrasion** – loss of tooth structure by mechanical forces from a foreign element.
- Possible sources of this wearing of tooth are toothbrushes, toothpicks, floss, and any dental appliance frequently set in and removed from the mouth.
- The appearance is V-shaped when caused by excessive pressure during tooth brushing.
- The surface is shiny rather than carious, and sometimes the ridge is deep enough to see the pulp chamber within the tooth itself.
- The teeth most commonly affected are premolars and canines.
Non-carious lesions, abrasion
Non-carious lesions

• **Dental fluorosis**, aka. *mottling of tooth enamel*, is a developmental disturbance of dental enamel caused by excessive exposure to high concentrations of fluoride during tooth development.

• Develops if fluoride concentration in food and water exceeds 2 mg/l (normal is 0.7-1.2 mg/l).
Non-carious lesions, dental fluorosis

- Occurs in children who are excessively exposed to fluoride between 20 and 30 months of age.
- The critical period of exposure is between 1 and 4 years old, and the child is no longer at risk after 8 years of age.
Non-carious lesions, dental fluorosis

• The severity depends on:
  – amount of fluoride exposure,
  – age of the child,
  – individual response,
  – weight,
  – degree of physical activity,
  – nutrition,
  – bone growth
Non-carious lesions, dental fluorosis

- Sources of fluoride:
  - dentifrice/fluoridated mouthrinse (which young children may swallow),
  - bottled waters which are not tested for their fluoride content,
  - public water fluoridation.
### Non-carious lesions, dental fluorosis: classification

<table>
<thead>
<tr>
<th>Dean's Index</th>
<th>Criteria – description of enamel</th>
<th>In former USSR</th>
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</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Smooth, glossy, pale creamy-white translucent surface</td>
<td></td>
</tr>
<tr>
<td>Questionable</td>
<td>A few white flecks or white spots</td>
<td></td>
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<tr>
<td>Very Mild</td>
<td>Small opaque, paper white areas covering less than 25% of the tooth surface</td>
<td>I stage - affection of less than 1/3 of the tooth surface</td>
</tr>
<tr>
<td>Mild</td>
<td>Opaque white areas covering less than 50% of the tooth surface</td>
<td>II stage - enamel affection (not dentine)</td>
</tr>
<tr>
<td>Moderate</td>
<td>All tooth surfaces affected; marked wear on biting surfaces; brown stain may be present</td>
<td>III stage - affection of more than 50% of the tooth surface</td>
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<tr>
<td>Severe</td>
<td>All tooth surfaces affected; discrete or confluent pitting; brown stain present</td>
<td>IV stage</td>
</tr>
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</table>
Non-carious lesions, dental fluorosis
Pulpitis

• **Pulpitis** is inflammation of dental pulp tissue.
• Etiology:
  – infection due to dental caries that penetrate through the enamel and dentin;
  – (very rare) infection due to lympho- or hematogenous dissemination
  – trauma;
  – chemical irritation;
  – thermal changes.
Pulpitis

• On clinical course:
  – acute (serous, focal purulent, diffuse purulent);
  – chronic (gangrenous, granulating, fibrous);
  – chronic with acute exacerbation.

• On localization:
  – crown;
  – root;
  – total.
Acute pulpitis

- **Serous pulpitis:** pulp is swollen, hyperemic, with slight leukocyte infiltration, hemorrhage, mild degenerative changes in the nerve cells.
- **Focal purulent pulpitis:** marked leukocyte infiltration with the formation of cavity filled with pus (abscess).
- **Diffuse purulent pulpitis:** exudate fills both crown and root pulp (phlegmon).

In case of carious destruction anaerobic infection can penetrate the pulp and **gangrene** develops:

- Grossly, pulp is gray-black with a putrid odor. Microscopically, pulp is structureless, sometimes contains fatty acids and microbes.
Acute pulpitis
Chronic pulpitis

Sometimes develops independently, but can result from acute pulpitis.

- **Gangrenous pulpitis** can arise from acute after the partial destruction of the pulp.

- **Granulating (hypertrophic)** is characterized by a chronic proliferative inflammation. Tooth cavity is replaced by granulation tissue, which can protrude to carious cavity. In such cases, the *pulp polyp* is formed. There may be a lacunary resorption of dentin. Maturation of granulation tissue leads to sclerosis; petrification leads to denticles formation.

- **Fibrous pulpitis** - most of the tooth cavity is filled with connective tissue with lympho-plasmocytic infiltration. Then hyalinosis develops, denticles are formed.
Chronic pulpitis, pulp polyp
Pulpitis, complications

- **Apical (or periapical) granuloma**, the most common sequel of pulpitis, is chronically inflamed periapical granulation tissue. The inflammation gradually becomes surrounded by a fibrous capsule, which, on extraction, may be seen attached to the root of the tooth.
Pulpitis, complications
Pulpitis, complications

- **Radicular cyst** (apical periodontal cyst) occurs when the squamous epithelium of an apical granuloma proliferates, forming a cavity or cyst.
Pulpitis, complications

- **Periapical abscess** may follow pulpitis.
- **Osteomyelitis** may complicate a periapical abscess, and is usually caused by *S. aureus*, *S. epiermidis*, various streptococci or mixed organisms. Infection may traverse the cortical bone and spread to tissue spaces of the head and neck or, rarely, mediastinum.
Periodontitis

- **Periodontitis** is a set of inflammatory diseases affecting the periodontium.

- **Etiology:**
  - trauma or chemical affection leading to infection

- **Ways of infection:**
  - intradental (after pulpitis);
  - extradental (from neighbour teeth);
  - lympho- or hematogenous.
Periodontitis, classification

• On localization:
  – apical
  – marginal.

• On clinical course:
  – acute;
  – chronic;
  – chronic with exacerbation.
Acute apical periodontitis

- serous
- purulent – usually with abscess formation
Chronic apical periodontitis

- **granulating** - formation of granulation tissue, osteoclastic resorption of bone, cement, sometimes dentin; fistulas may form;
Chronic apical periodontitis

• granulomatous:
  – simple granuloma: granulation tissue is surrounded by fibrous tissue;
  – complex (epithelial) granuloma: + strands of stratified squamous epithelium, penetrating the granulation tissue;
  – cystogranuloma: cavity is formed, lined with epithelium.

• fibrous.
Pulp stones, or denticles

- are nodular, calcified masses appearing in either or both in coronal and root portion of the pulp.
Pulp stones, or denticles

On the structure:

• true denticles (formed by odontoblasts).
• false denticles (formed by an accumulation of mineral around debris, often in a concentric manner).
Pulp stones, or denticles

On location

• free - entirely surrounded by pulp tissue;
• attached - partly fused with dentin;
• embedded - entirely surrounded by dentin.
Pulp stones, or denticles
Pulp stones, or denticles