Cariology in deciduous dentition.

5.DM - Pedo
Dental caries (in general MT, PT)

- Progressive demineralization and dissintegration of the calcified dental tissues, that occurs underneath a layer of bacteria on the tooth surface

- Chronic infectious non-transmittable disease

- Affects 80-90% of young population
Dental caries

- caused by acids formed by plaque bacteria through metabolism of dietary sugars

- Most dramatic increase of dental caries – end of 19th, 20th century – coincides with increase in sugar consumption – industrialisation, urbanisation
Caries etiology

- Susceptible tooth surface
- Carbohydrates
- Cariogenic bacteria
- Time

*Fermentable Carbohydrate
**Particularly *Streptococcus mutans*
External factors

- oral hygiene
- mechanical impairment of HDT
- chemical impairment of HDT
- saliva secretion
- dietary habits

Sugar consumption $\Rightarrow$ Dental caries
Caries etiology - Sugar

A large number of other epidemiologic studies clearly confirm the correlation between sugar consumption and decay.

*Positive correlation between sugar intake and dental caries has changed after introduction of fluoride as a preventive agent*

People of an isolated island in Atlantic had low caries rate due to low sugar intake. After introduction of sugar the prevalence of caries has increased from 5 to 30%

Extremely low caries experience in patients with hereditary fructose intolerance
Caries etiology - Bacteria

- Orland et al., 1950, the significance of the infective agent
- No cavities developed on high sugar diets, until cariogenic MIO were introduced
- Most cariogenic bacteria Str. mutans
Caries etiology - Bacteria

Str. mutans in the presence of sugar

extracellular glucan (glucose polymer)

establishing on tooth surface, producing highly cariogenic plaque
Mechanism of dental caries formation

Destruction of HDT by organic acids that are produced in carbohydrate metabolism done by oral MIO (SM, LA)

Desintegration of organic substance of HDT induced by direct proteolytic activity of oral MIO

After sugar intake and synchronous MIO activity followed by acids creation resulting in fall of pH level of saliva below 5.5 leading to the process of demineralisation in subsuperficial layer of enamel

Enamel becomes porous leading to loss of enamel translucency (white spot)
Mechanism of dental caries formation

Changes in plaque pH following a glucose rinse ("Stephan curve").
Mechanism of dental caries formation

- Diffusion of acids from plaque to subsuperficial layer of enamel, where hydroxyapatite is dissolved and Ca, PO₄, F ions pass into saliva and plaque.
- When pH rises above 5.5 and sufficient amount of ions in saliva is present – reverse diffusion of ions begins – remineralisation.
- Cavity formation – when the process of demineralisation is greater remineralisation.
DEMINERALISATION AND REMINERALISATION

- Plaque bacteria produce acids by fermentation of sugars, decreasing the pH at the tooth surface. The sugars may be already present in foods or produced by starch breakdown in the mouth. These acids dissolve the minerals in the enamel (calcium and phosphorus) - a process which is known as demineralisation. Enamel demineralisation takes place below a pH of about 5.5 - this has been called the 'critical pH'.

- The acids produced by bacteria in the mouth are gradually neutralised by saliva. This causes the pH of the tooth surface to rise above the critical pH. The increase in pH causes a return of the dissolved calcium and phosphate back to the tooth enamel. This is called remineralisation. If foods or drinks containing carbohydrate are eaten frequently during the day there is little chance for remineralisation to occur. In contrast, if there is sufficient time between meals (approximately 2-3 hours) and the damage is not too great, the teeth can repair themselves.
Demineralisation - white spots

- Appear in the enamel, underneath the plaque
- Initial demineralisation usually takes several months, though under extreme conditions can occur in a matter of weeks
- The early changes are caused by a selective dissolution of the more solubable crystal components in the enamel surface.
- Deeper layers of enamel, which become exposed at later stages have a lower resistance and the demineralisation proceeds more rapidly here, undermining the enamel surface
white spots – initial lesion – advanced caries
Demineralisation - white spots

- Undermined enamel eventually breaks, forming a cavity, which is entered by bacteria in large numbers and the speed of process increases – the demineralisation of internal regions of enamel – forming a visible cavity

- wide individual response to caries

- Fluoride in drinking water increases resistance
Dental caries in enamel

1. Superficial layer 1-10% density loss
2. Body of lesion 25% density loss
3. Dark zone 6% density loss
4. Translucent zone 1% density loss
Dental caries in dentine

1. Zone of destruction – desintegration of organic stroma
2. Zone of penetration – MIO in dentinal tubuli
3. Zone of demineralisation – of dentine and sclerotic dentine
4. Translucent zone – deposition of calcium - sclerotisation
5. Zone of fat degeneration of Thomas filaments
Why to treat caries in deciduous dentition

- Destruction of HDT leads to the loss of function of the tooth
- Untreated caries can lead to local and systemic complications – inflammation in orofacial region
- Contact transmission of infection
- Higher risk of caries prevalence
- Orthodontic anomalies (inclinations and ectopic eruptions of permanent teeth)
- Esthetics
- Influence on GIT (disorder of food intake)
Why to treat caries in deciduous dentition

- Pronunciation disorders (frontal teeth loss)
- Psychological impact when frontal teeth are destructed
- Unsufficient oral hygiene
- Close relation to caries occurrence/prevalence in permanent dentition (kids with high caries experience in MD are in risk to have high incidence of caries in PD as well)
Classification of dental caries in milky dentition

1. Course: acute, chronic

2. Depth of penetration: superficial, deep, arrested caries

3. Type of DT: enamel, dentine

4. Primary, secondary, recidivans

5. Black classes (I, II, III, IV, V)
akútny kaz

chronický kaz

kariézna sklovina

vonkajší kariézny dentín
infikovaný
neminerализovateľný
devitálny
farbiteľný detektorom
/má byť odstránený/

vnútorný kariézny dentín
neinfikovaný
mineralizovateľný
vitálny
nefarbiteľný detektorom
/má byť zachovaný/

tmavé sfarbenie
maskuje zafarbenie detektorom
/má byť odstránený/
Caries in decidual dentition

Specifics of macromorphology:

- size and shape of teeth
- enamel ridges
- differences in morphology (thinner layer of enamel and dentine)
- size of pulp chamber
Caries in decidual dentition

Specifics of micromorphology:

- lesser mineralisation of enamel
- different outline of enamel prism
- wide dentine tubuli
- biological quality/value of pulp
Caries in decidual dentition

Clinical picture:

Subjective:

- painless (initial caries)
- pain on pressure (chewing) and chemical stimuli (deep caries)
- food retention
Caries in decidual dentition

Clinical picture:

Objective:

- discoloration of enamel
- rough surface,
- retention of probe
- plaque
- defect in dentine (retention of food)
Caries in decidual dentition

Susceptibility of individual deciduous teeth to dental caries:

- second molar
- first molar
- upper incisors
- canines
- lower incisors
Caries in decidual dentition

Susceptibility to caries concerning age of a child

- 4. – 8. years old - milky teeth
- 6. – 9. years old - permanent teeth
Caries in decidual dentition

**Diagnosis** of the lesion:

- direct vision
- dental probe
- dental floss
- radiographs
- diagnostodent

*Prior to inspection – removal of plaque covering the surfaces – gingival region, fissures*
Predilection sites of dental caries

For both primary and the secondary dentition

- Pits and fissures
- Proximal surfaces
- Gingival third of crown surface

In primary dentition a less pronounced fissure system is found – especially first primary molars – only few isolated pits
Predilection sites of dental caries - MT

- Young pre-school children: Spacing in the molar areas reduces the number of proximal lesions.
- With increasing age, proximal contacts are established which gives rise to an increase of proximal lesions in primary molars (4-5y.).
- The high prevalence of caries of the upper central primary incisors is partly due to the fact, that the incisal papilla at this age is situated close to the mesiolingual aspect of those teeth – which provokes plaque accumulation, which in turn may lead to inflammation of the papilla followed by even greater retention of plaque.
Caries therapy in deciduous dentition

- **white spot lesions**
  - lesions without cavitation are treated by impregnation with F varnish
  - in home care we advice the usage of fluoride tooth paste 2x per day
  - until 6th year of age pastes with higher content of F are not recommended
Caries therapy in deciduous dentition

- **Superficial caries:**
  - preparation depends on the type of filling material used
  - preparation limited only to caries (effort to preserve sound HDT)

- **Caries on occlusal surface:**
  - preparation is led by occlusal anatomy (do not remove enamel ridge)
  - preparation in enamel is done by diamond rounded bur or inverted cone diamond bur
Caries therapy in deciduous dentition

- cavity: box shape (amalgam)
- bowl shape, rounded walls (GIC)
- cavity depth 2-3 mm (thinner layers of HDT in milky tooth)

- Aproximal caries of distal teeth:
  - isthmus width - \( \frac{1}{2} \) intercuspal distance (amalgam)
  - gingival step right angle, but rounded
Caries therapy in deciduous dentition

- Possibility of preparation of proximal surface without occlusal cavity (if neighbouring tooth not present or spacing)
  - usage of matrices
  - note! pulp horns enter high into cusps

- tunel preparation
Caries therapy in deciduous dentition

Treatment of deep caries

- Carefull preparation of soft carious dentine, mainly with hand instruments
- When using handpiece – big round burs in slow rotation with minimal pressure
- When caries pulpae proxima – leave thin layer of soft dentine on the bottom of the cavity and place Ca(OH)2
Caries therapy in deciduous dentition

Aproximal caries of frontal teeth

- Entrance to carious lesion from vestibular surface (cooperation, better vision)
- Preparation limited to the extense of caries
Caries therapy in deciduous dentition

- **Preparation of decidual teeth**
  - turbine (only in enamel, only in cooperative children)
  - red micromotor (stability of rotation)
  - blue and green micromotor (speed reduction, rounded burs in dentine)
  - hand instruments (excavator in dentine)
Caries therapy in deciduous dentition

Filling materials

1. Glass ionomer cementums (Bl I, II, III, IV, V)
2. Amalgam (Bl I, II)
3. Compomer materials
4. Composite materials – endodontically treated teeth
5. Prefabricated crowns – such as stainless steel crowns,
Restoration – factors to consider

- Age of the child
- Degree of caries involvement
- Condition of tooth and supporting bone on x-ray
- Time of normal eruption and shedding
- Child’s health
- Space consideration in the arch
Few more notes (cariology in MD)

Anatomical differences in primary teeth

- Prompt treatment of initial lesions:
  - large pulp chambers
  - prominent pulpal horns
  - proximity to the external surfaces

Modifications of cavity preparation rules – (Black) for primary dentition
The initial pit and fissure lesion presents itself as a discoloration of the fissure system (dark or white).

The manifest pit and fissure lesion causing undermining of the enamel, as the lesion spreads towards E-D junction.

Not yet gross loss of substance – careful probing.

On inspection – undermined enamel – greyish discoloration extending laterally from the fissure.

In later stages, enamel breaks – lesion easily detected by visual inspection.
Few more notes (cariology in MD) Smooth surface lesions

- Acute lesions – loss of surface continuity
- Exposure of dentin
- Large lesions extending to less caries-prone areas
- White opaque enamel circumscribing the actual cavity is a sign of high activity
- Acute dentinal lesions – yellow, soft dentin
- Chronic lesions – darker, hard dentin
Few more notes (cariology in MD)

Proximal lesions

- Early stages cannot be clinically detected
- Later stages – removal of plague + gentle retraction of the interdental papila
- Especially in primary dentition proximal lesions may extend buccally and palatally as an initial decalcification located under contact area
Few more notes (cariology in MD)Proximal lesions

- More advanced stages – undermined enamel
- Dark or greyish shadow observed from occlusal aspect, extending centrally
- In the primary dentition – broad contact areas – detection in very advanced stages
Few more notes (cariology in MD)

Treatment of deep lesions

- Removing of undermined enamel, good view of the operation area
- Removal of carious dentine with spoon excavator, low speed micromotor, round bur
- At low progression of the lesion, the pulp of primary tooth can produce irritation tertiary dentin to a large extent
Few more notes (cariology in MD)

Treatment of deep lesions

- Cavity clinically caries-free water spray, dry
- Eventually also with a dry cotton pellet
- Apply a pure calcium-hydroxide-water mixture or CaOH cement over the deepest parts – ability to reduce pulpal irritation
- Phosphate cementum, GIC
ECC – early childhood caries

- Infectious disease influencing the quality of life of a child
- Develops as early as in the first year of life (infants and toddlers)
- Rapid type of caries
- Cervical type of cavities
- Prolonged bottle feeding
- Decreased salivary flow during sleep

Other terms commonly used:

Nursing Bottle Caries, Baby Bottle Tooth Decay (BBTD), Baby Bottle Caries, Nursing Bottle Syndrome, honey teeth
ECC definition (AAPD)

The presence of one or more decayed (non-cavitated or cavitated lesions), missing (due to caries) or filled tooth surfaces in any primary tooth in a preschool-age child between birth and 71 months of age.

In kids younger then 36 months is any sign of caries on smooth surface regarded as severe ECC. In the age of 3-5 is S-ECC (severe ECC) characterised by the presence at least 1 carious lesion on frontal milky teeth or score dmf $\geq 4$ in 3.years of age, $\geq 5$ in 4.years of age, $\geq 6$ in 5.years of age
ECC – early childhood caries

- Numerous definitions and synonyms
- Generally - caries that spreads quickly, destroying the crowns of many or all of the erupted teeth
- Affects surfaces that are normally less likely to decay (habitually clean)
- Can lead to early pulp damage
- There is an absence of decay of the lower incisors
- Teeth - brown or black
- Parents often say – teeth have erupted decayed

Other terms commonly used:

Nursing Bottle Caries, Baby BottleTooth Decay (BBTD), Baby Bottle Caries, Nursing Bottle Syndrome
**ECC – early childhood caries**

- **Etiology**

  1. *S. mutans* is considered to be the main causative agent of ECC. *S. mutans* not only produces acids, but an acidic environment is suitable for its existence.

In kids with ECC the level of SM exceeds 30% of the cultivated flora from dental plaque. Kids without active caries have level of SM less than 1%.
ECC – early childhood caries

Transmission of SM

- From mother to child (identical bacterial DNA profiles), horizontal transmission
- From family members and caretakers to child, vertical transmission
- Transmission of SM from mother’s saliva to child: mouth kissing, licking pacifier, spoon or fingers of child
2. Second not less important factor in etiology of ECC are dietary habits

- main cause: prolonged or frequent use of sugary drinks in a nursing bottle
- If a child has free access to sweet drinks for the whole day, or sweetened baby food
- when the child is left with a bottle or during the night or prior to sleeping.
- inappropriate nursing habits - breast milk if feeding is carried out on demand, nocturnally or for prolonged periods
ECC – early childhood caries

- Addition of sugar to baby food
- Dipping of pacifier into honey, sugar, preserves, sirups
- During the night saliva flow decreases thus its neutralising effect is reduced. Moreover in repeated saturation with cariogenic food (drink) acid pH is persistent repeatedly and for a long time in saliva and plaque leading to demineralisation of HDT. Saliva at night has only limited chance to neutralisation and remineralisation.
3. Susceptible dental tissue (tooth surface)
4. Insufficient oral hygiene
5. Low socio-economic status of the family

- Frequent intake of medicaments containing sugar
- Amount and quality of saliva
ECC – early childhood caries

Clinical picture:

- ECC starts by demineralisation of enamel on labial and palatal surface of upper frontal teeth in gingival third
  - White spots change their colour to light and dark brown and later cavitation occurs
- ECC has circular character
- Fast destruction of crown, infection reaches pulp, eventually crown fracture
ECC – early childhood caries

- ECC afflicts primarily upper incisors, then upper molars, lower molars and canines upper and lower
- Lower incisors are spared from caries
- In severe ECC even lower incisors are decayed
ECC – early childhood caries

**Therapy**

- Conservative treatment
- Endodontic treatment
- Surgical treatment
  (treatment in general anesthesia)
Conservative treatment:

Non cavitating lesions – local application of F varnishes every month, change in dietary habits, usage of F tooth pastes

Cavitating lesions:
Preparation of HDT
1. Fillings GIC
ECC – early childhood caries

2. chemical-mechanical preparation
Infected dentine dissolves by chemical agents and carious debris is then removed mechanically

*Carisolv* – (NaOH, NaCl, lysin, leucin, carboxymethylcelulose, erythrosin), two components mixed together to gel, which selectively bides to soft dentine and does not influence healthy dentine
3. ART
Atraumatic restorative technique
- Removal of carious dental tissues by hand instruments. Less HDT loss then when rotatory instruments are used
- This technique is suitable for central cavities on occlusal surfaces of molars
- Instruments used: enamel chisel, excavating spoons, carvers for removal of excessive filling material
- Most suitable filling material is GIC
ECC – early childhood caries

- **Surgical therapy**
  Extractions, in sever form of ECC when many extractions needed – these are performed under general anesthesia

- **Prosthetic treatment**
  Prefabricated crowns - stainless steel crowns for molars, resin crowns for incisors
Consequences of untreated ECC

- Spread of caries into deeper structures of the tooth – deep caries
- Spread of infection into pulp
- Crown fractures
- Turner’s tooth
- Focal infection, inflammatory complications
- Retardation of development of child – food intake
- Psychological aspect
Prevention of rampant caries

- Milk or water should constitute the majority of drinks given to young children
- If another drink is chosen the following advice should be followed:
  - Always follow manufacturers' instructions on usage and dilution of drinks
  - Serve only at meal time and keep drinking times short
  - Never leave infants alone with any drink
  - Never use drinks on a dummy or as a comforter
  - Ideally serve from a spoon or cup, but not from a bottle
  - Do not give at bedtime or during the night
Prevention od rampant caries

- Prevention of transmission of SM
- Oral hygiene (instructions of techniques and usage of tooth pastes given to parents)
- Dietary recommendations
- Regular check-ups in dental office
Prevention of rampant caries

- alter the way in which sugary drinks are consumed
- parents should also remember to brush their children’s teeth – wet gauze- as soon as the teeth erupt
- children at high risk and living in areas with water less than 0.3ppm fluoride ion, supplements may be used
- fluoride supplements only be given following guidance from a dentist

Fluoride supplements should not normally be given to children living in areas with water containing fluoride at a level of 0.7ppm or more.