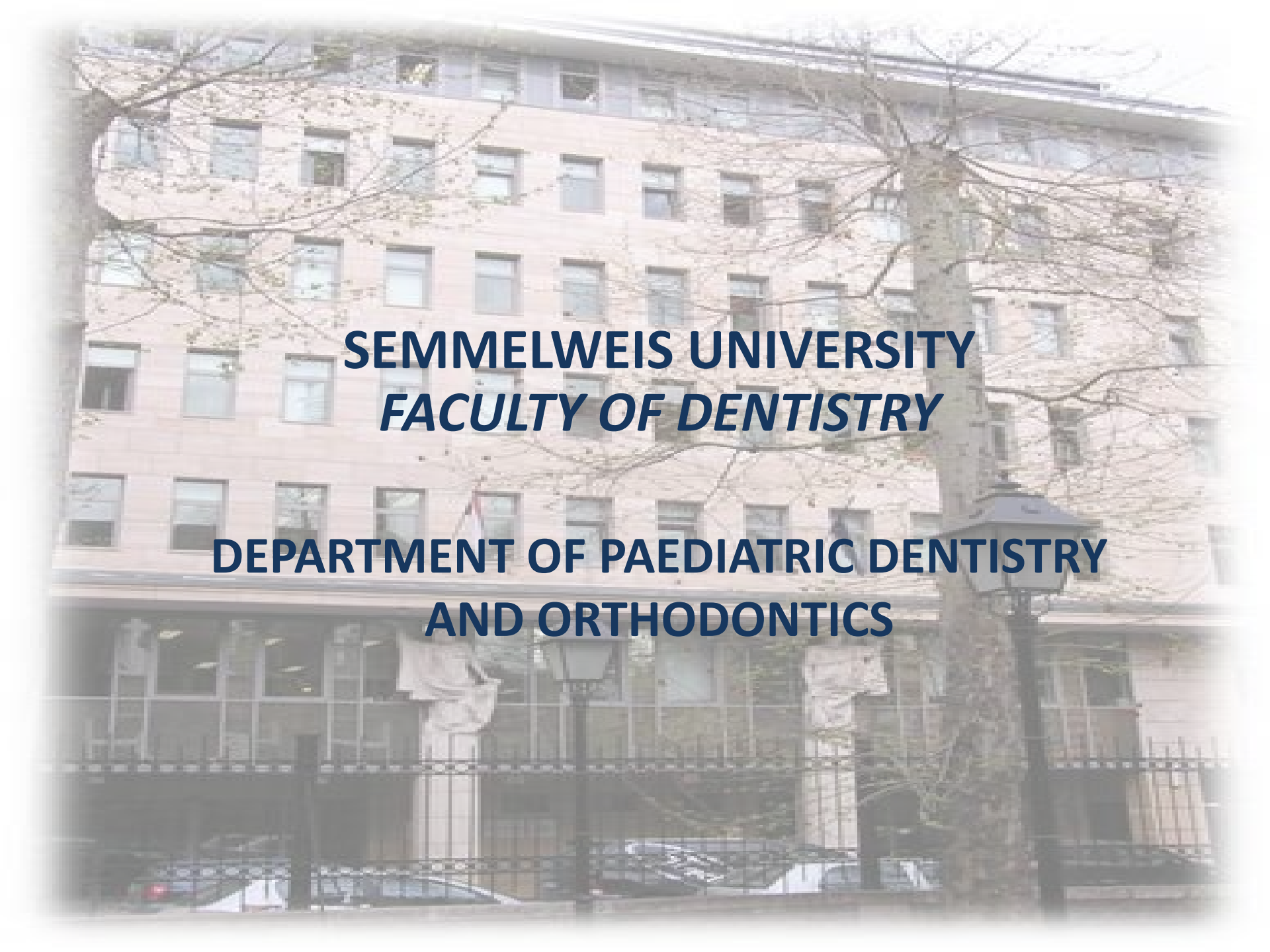


SEMMELWEIS UNIVERSITY
FACULTY OF DENTISTRY

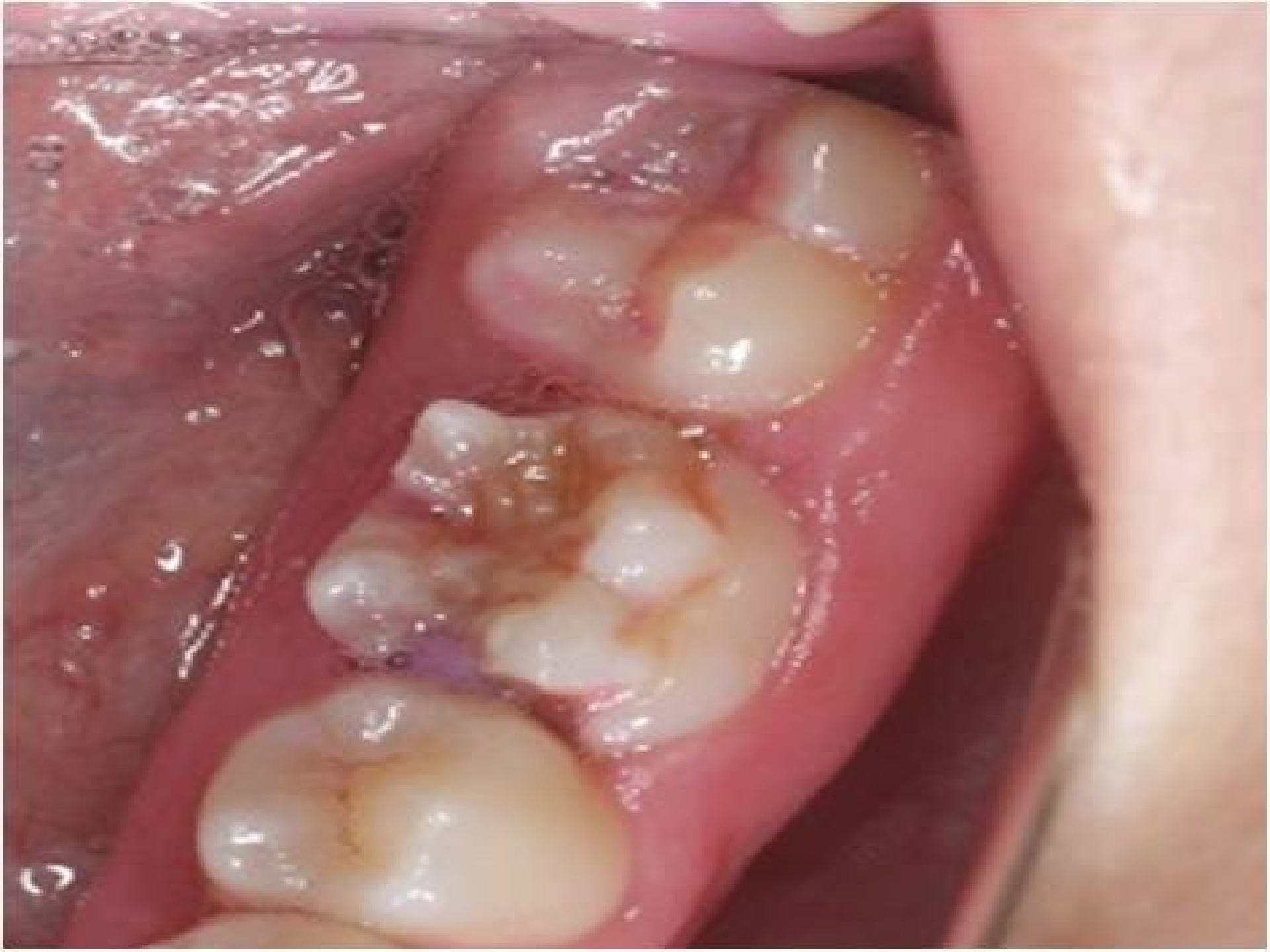
DEPARTMENT OF PAEDIATRIC DENTISTRY AND ORTHODONTICS

The Molar Incisor Hypomineralisation Syndrome MIH

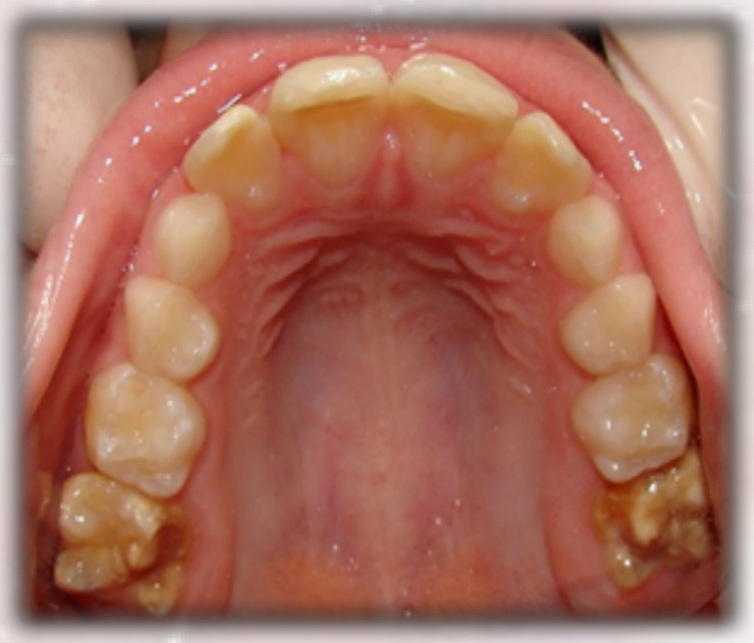
Etiology, Manifestation, Treatment Possibilities

Assoc . Prof. med habil Noémi Katinka RÓZSA

DMD, MSc, PhD



MIH - SYNDROME



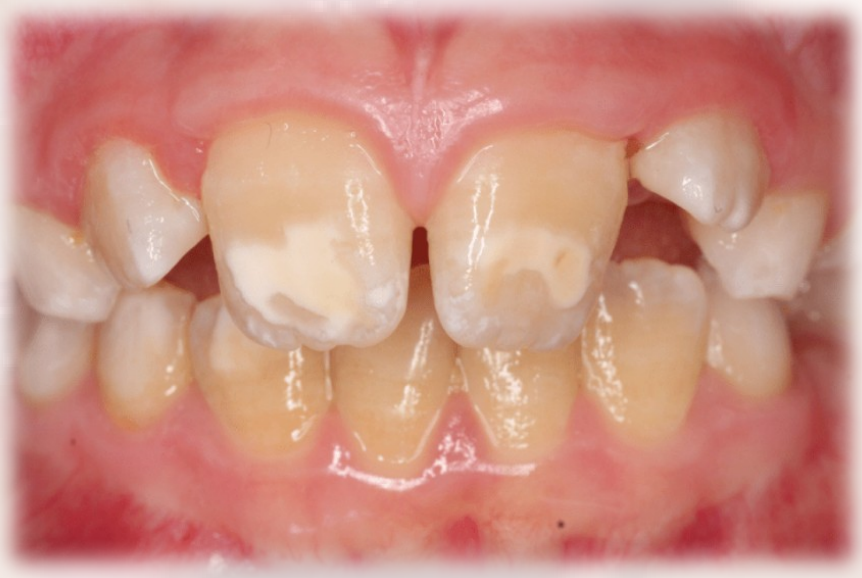
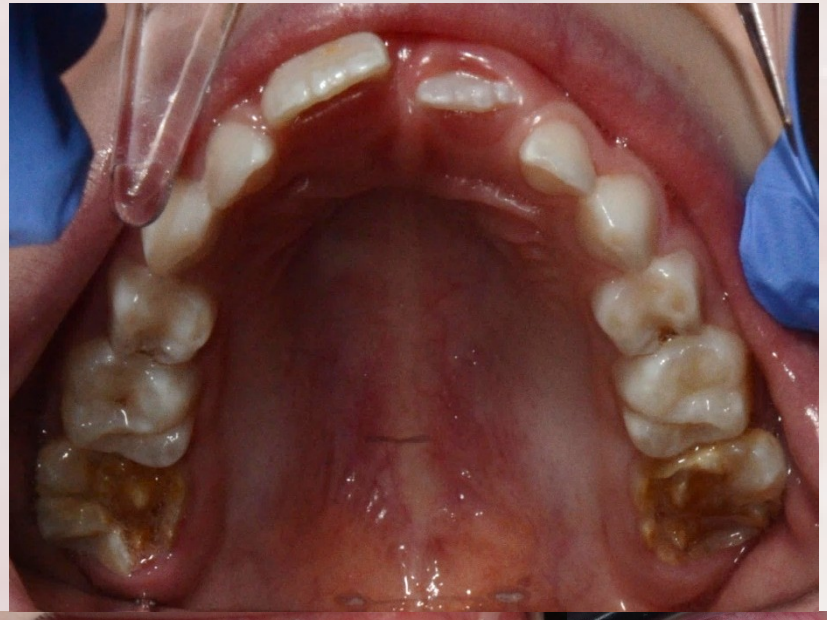
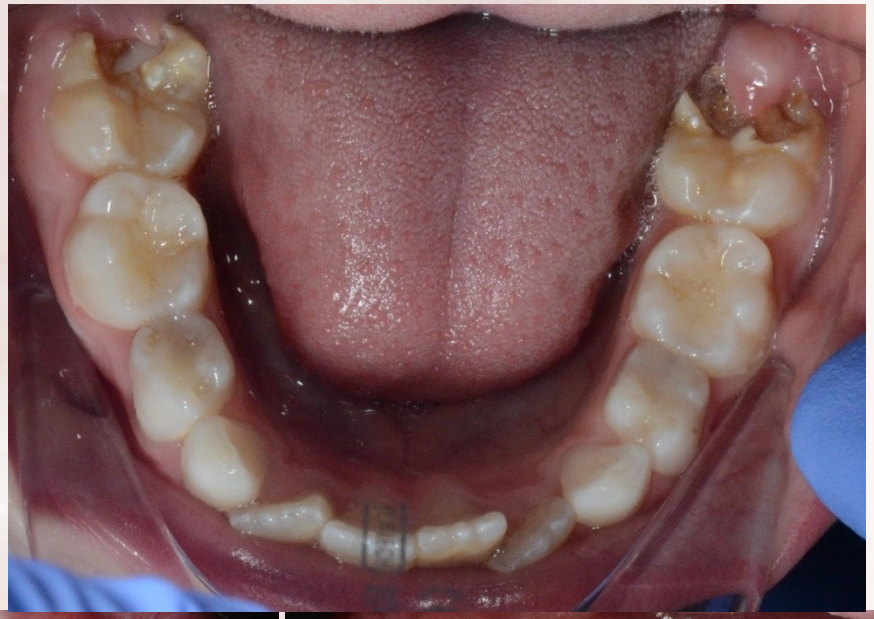
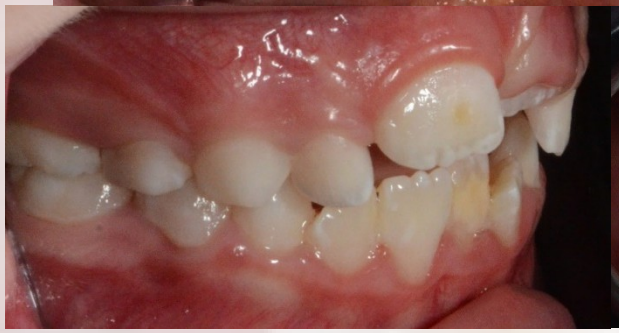


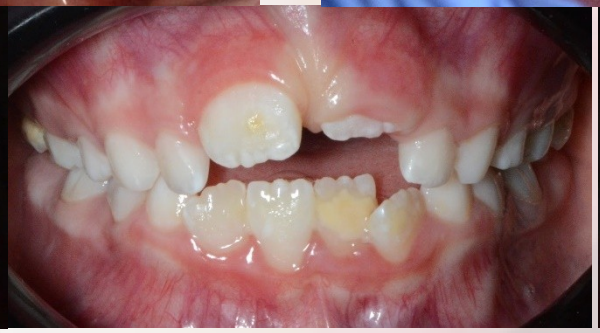
Photo source: SE FOK Dep. for Pedodontics and Orthodontics, Rózsa N.

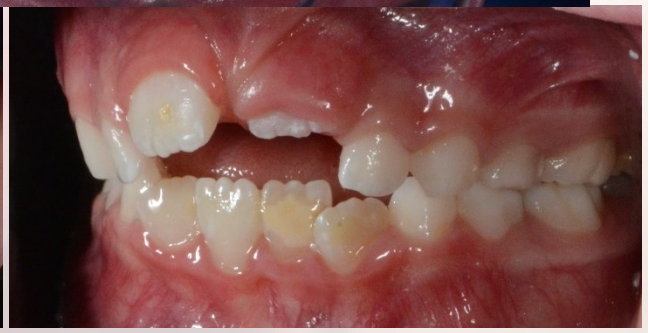
MIH - SYNDROME





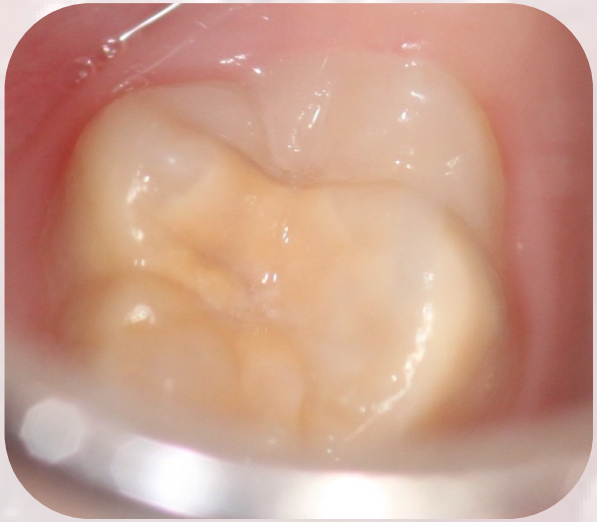


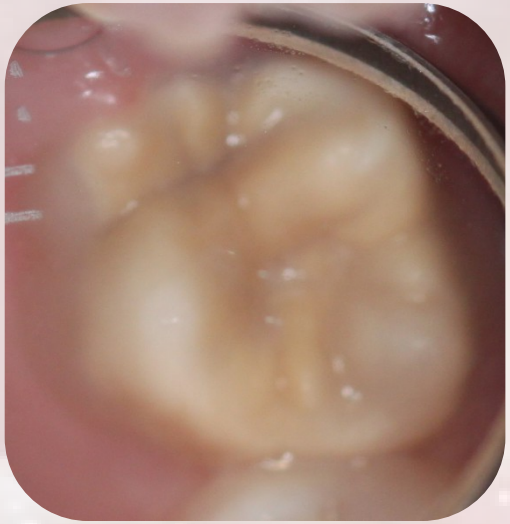


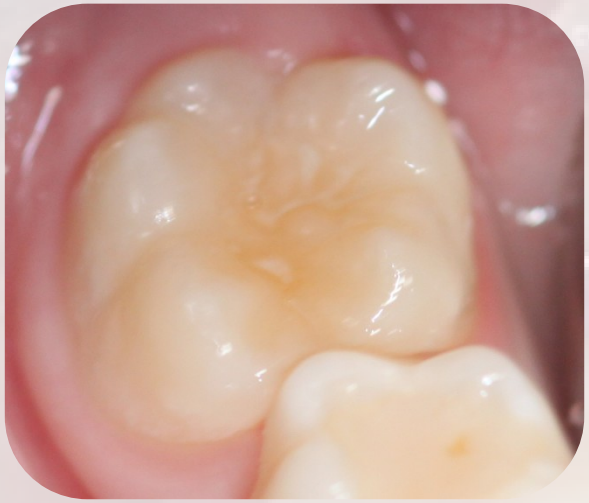


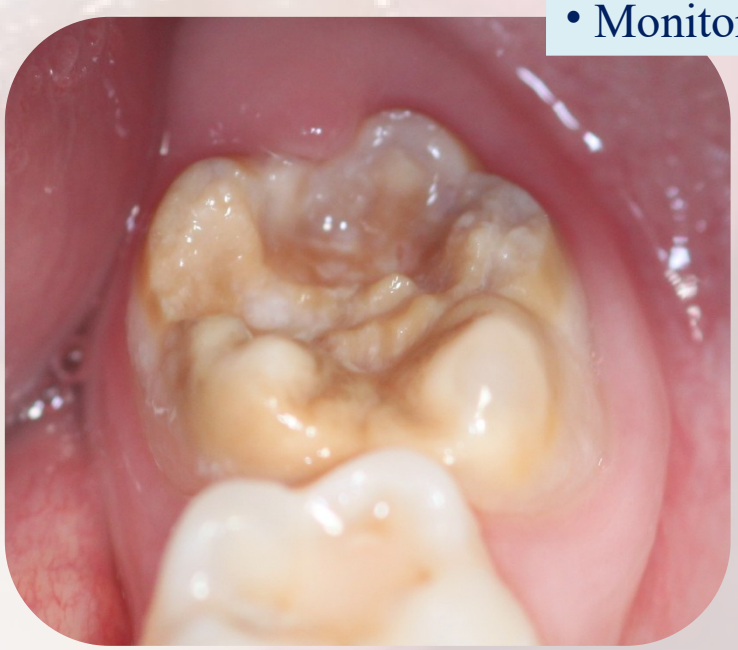
SE FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga, Mlinkó E.,Rózsa N.

MIH – „Molar-incisor hypomineralisation”









- Hypomineralisatio, hypoplasia
- **localisation: permanent first molars, permanent incisors!;**
- Aetiology– multifactorial – genetic?;
- Retrospective studies;
- Complex therapy;
- Monitoring!

Weerheijm et al. Molar-Incisor Hypomineralisation. *Caries Research 2001; 35:390-391.*

MIH – „Molar-incisor hypomineralisation”

Introduction, Definition, Classification

(Jackson, 1961): Nonendemic enamel spots

(Koch et al., 1987): 1987: first described, in literature, as a clincial entity, as an ’idiopathic hypomineralisation’ of the enamel of incisors and first permanent molars.

(Leppäniemi et al., 2001): „cheese molars”

(van Ameringen és Kreulen, 1995): non-fluoride induced hypomineralisation of first permanent molars

2001: at the European Academy of Paediatric Dentistry (EAPD) congress Weerhejm et al. suggest the nomenclature ’Molar Incisor Hypomineralisation’ (MIH), defining the syndrome as a ’hypomineralisation of systemic origin of one to four permanent first molars frequently associated with affected incisors’.

2003: at the EAPD congress the Weerheijm et al. MIH judgement criteria (classification) was adapted, allowing reproducibility of future study parameters:

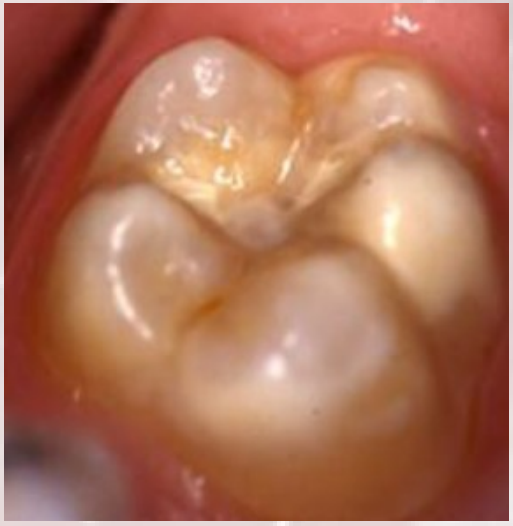
- ***Presence of well demarcated opacities***
- ***Posteruptive enamel breakdown***
- ***Atypical restorations***
- ***Extraction of molars due to MIH***
- ***Failure of eruption of a molar or incisors***

Criteria and Clinical Appearance [4]

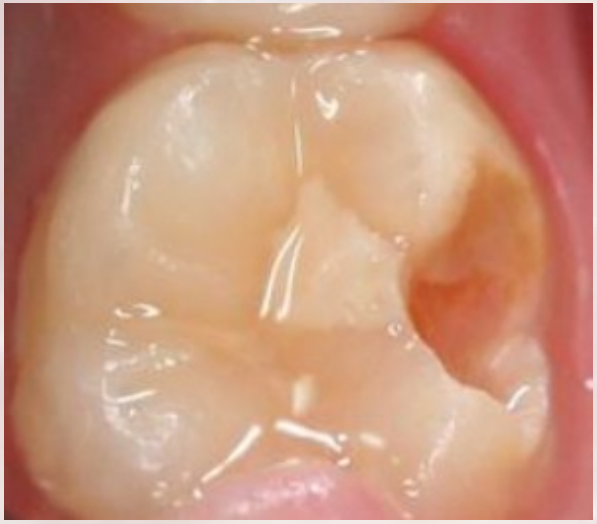
- White-chalky/creamy or yellow-brown demarcated opacities, appearing with a smooth, hard surface on enamel of normal thickness.
- Sub-surface underneath is porous and soft, highly prone to destruction under the strain of the masticatory forces, posteruption.
- Hypomineralisation acts as a plaque reservoir: caries predilection.
- Tooth hypersensitivity often presents from the onset, children will avoid brushing the affected teeth: caries predilection.
- The fragile nature of the enamel, it's propensity as a caries predilection area, together with the hypersensitivity induced compromised oral hygiene, greatly increases the risk of decay leading to rapidly irreversible damage to the coronal structures of the affected tooth/teeth.

Criteria and Clinical Appearance [5]

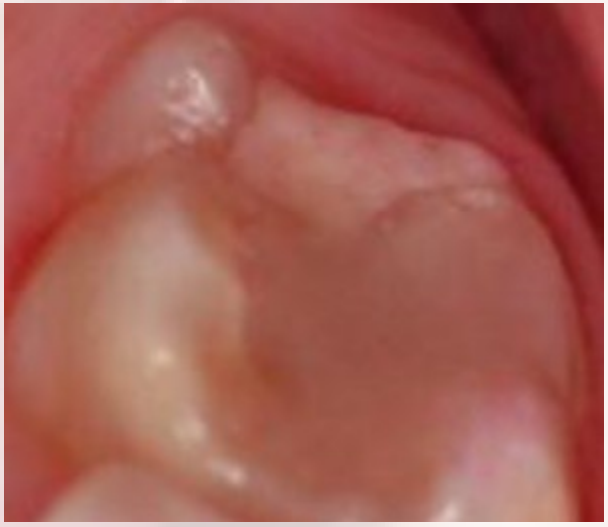
Demarcated opacity



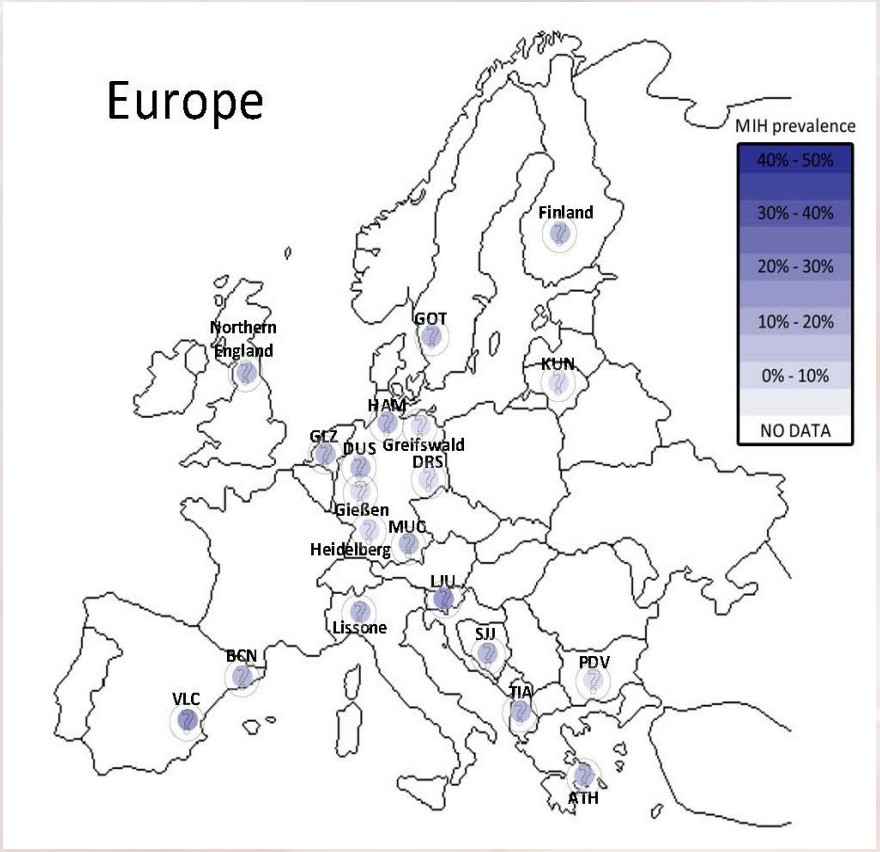
Posteruptive enamel breakdown

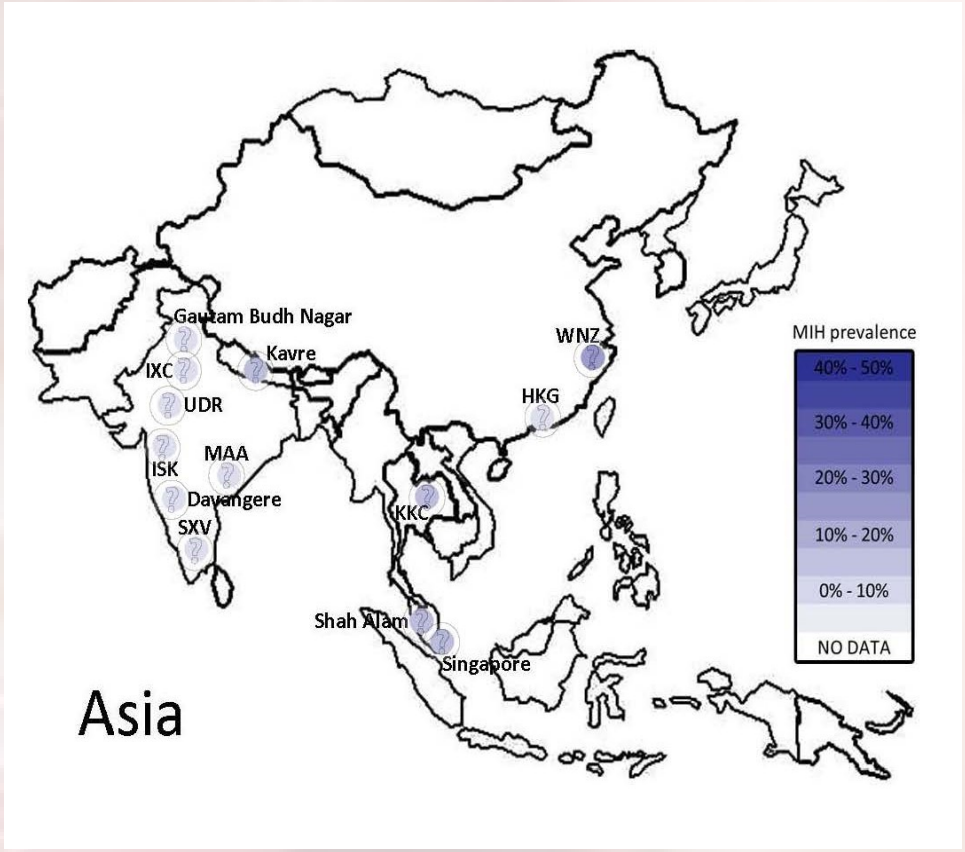


Atypical restoration



Prevalence: 3,6 - 25%;
Europe– EAPD - Weerheijm and Mejàre (2003).

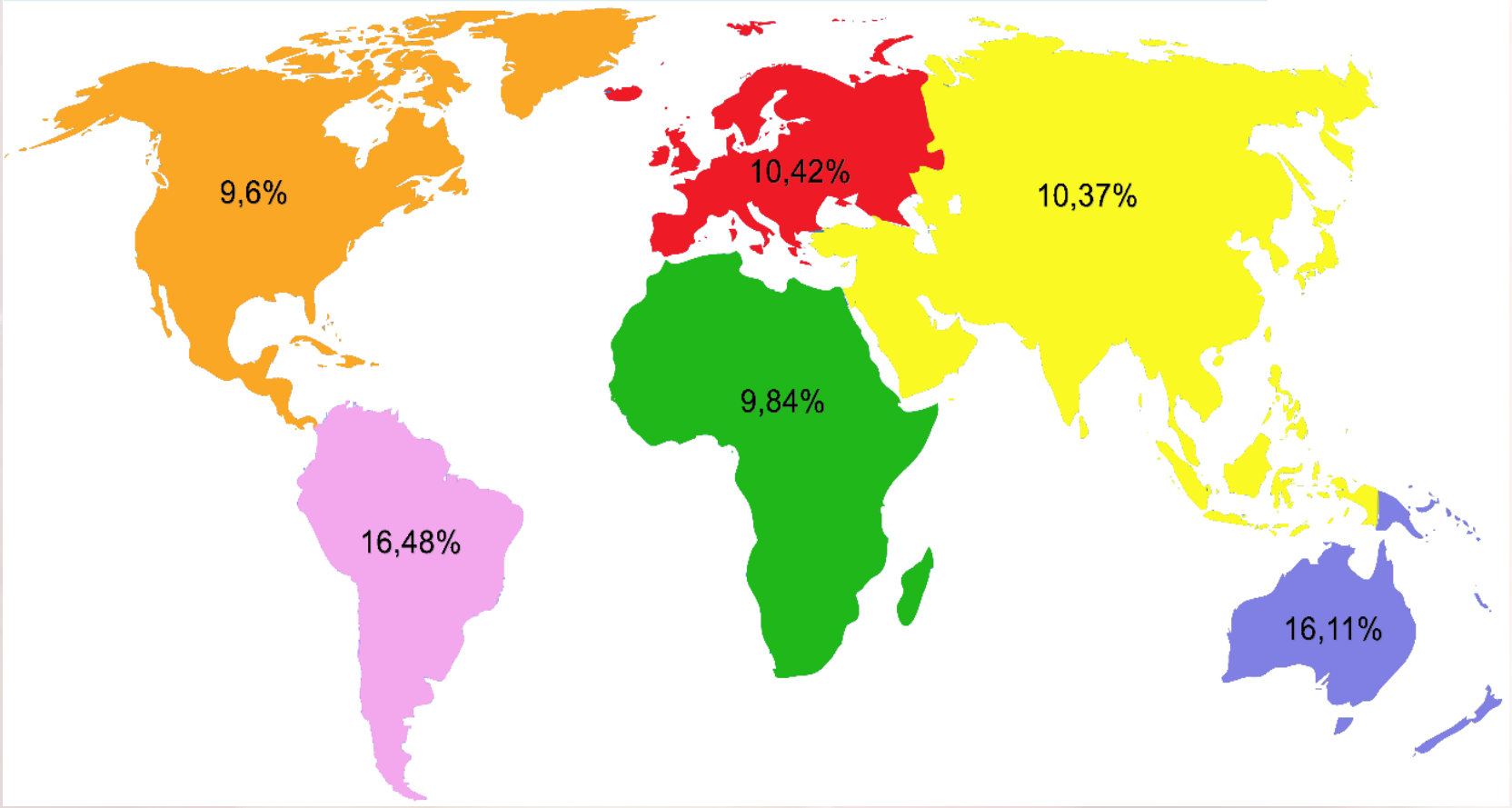




Source:

- *Moldován A, Reichel L. The prevalence of Molar Incisor Hypomineralisation in the Hungarian population and the main etiological factors. SE Int St Conference 2017;*
- *Weerheijm KL, Mejàre I. Molar incisor hypomineralisation (MIH): A questionaire inventory of its occurence in member countries of the European Academy of Paediatric Dentistry (EAPD). Int J Paediatr Dent 2003; 4 :114-120.*

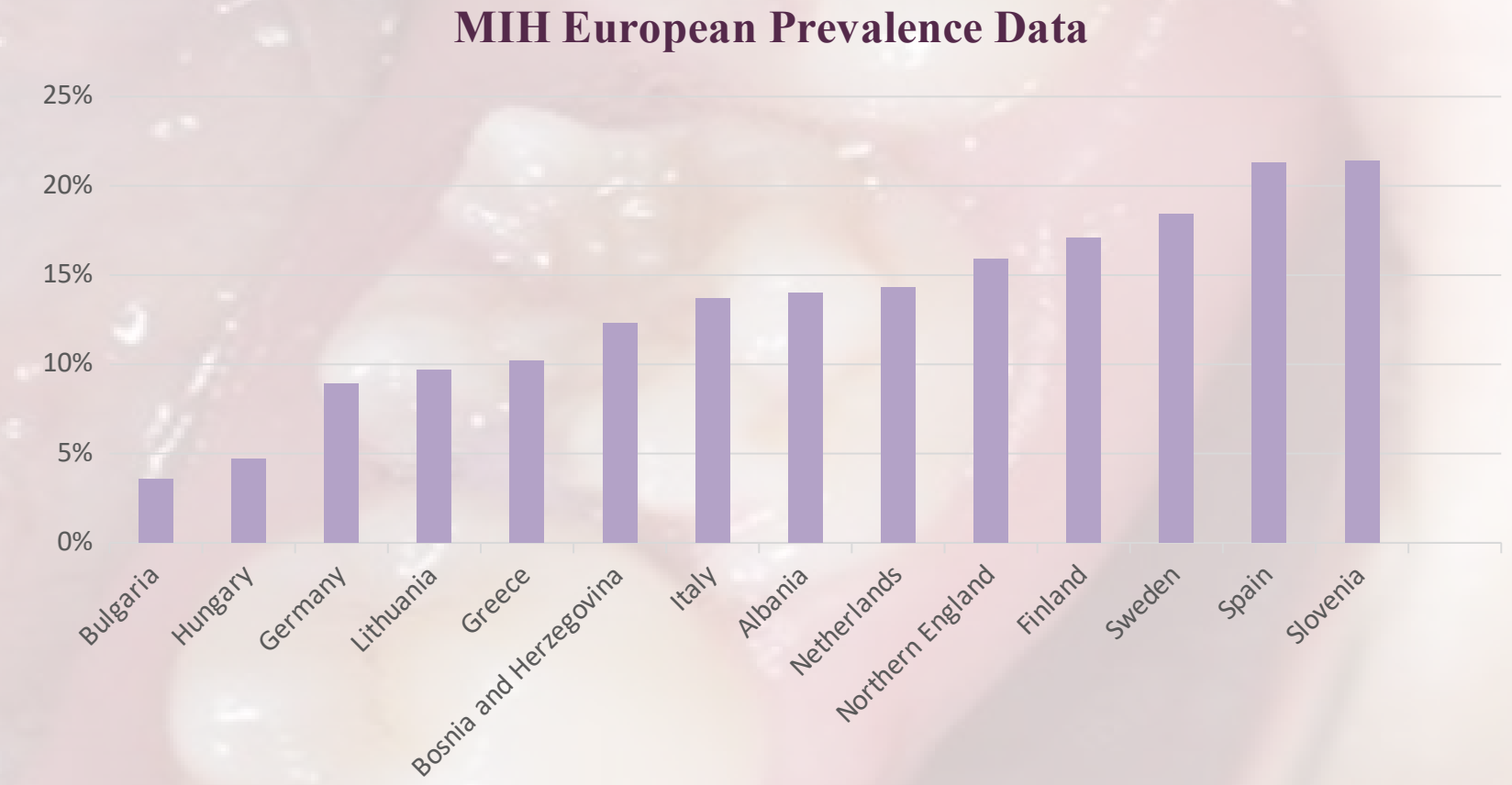
Prevalence worldwide: average → 10,97%;
2,8-40,2%;
1/6 children (Hubbard, 2018).



MIH - SYNDROME

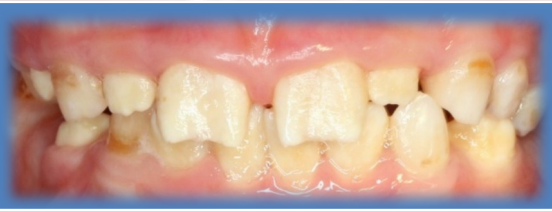
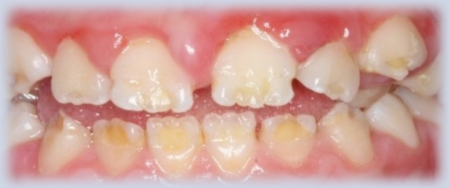
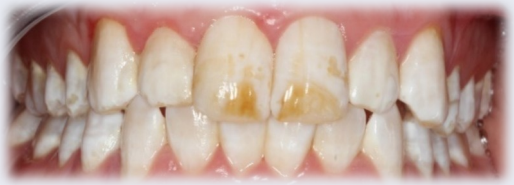
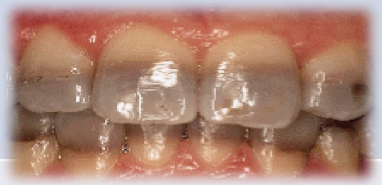
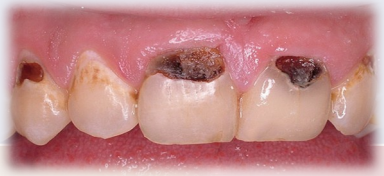
Prevalence: 3,6 - 25%.

MIH



Source: Moldován A, Reichel L. The prevalence of Molar Incisor Hypomineralisation in the Hungarian population and the main etiological factors. SE Int St Conference 2017.

Differential Diagnosis

Amelogenesis Imperfecta		• All teeth are affected symmetrically
Enamel Hypoplasia		• Roughened surface with discrete pitting or circumferential irregularities which acquire a yellow brown stain posteruptively
Fluorosis		• Diffuse, symmetrical disorder • Decay resistant
Tetracycline Xantodontia		• Grey to yellowish colour • Symmetrical • Per administration during pregnancy and to children less than 6 years of age
Caries		• Usually occur in the most vulnerable (predilection) areas

Amelogenesis

- Imbalance occurring during the secretory stage results in enamel hypoplasia, whereas if it occurs during the maturation phase it results in hypomineralisation: a tissue (enamel) translucency anomaly with no alteration in enamel thickness.[6]

- Maturation phase: where the bulk of mineralisation occurs and likely the point where pathological disturbance(s) to the ameloblasts cause(s) MIH

    1. Ruffle-ended phase: ameloblasts in this phase release the mineral building blocks necessary for hydroxylapatite ($Ca_{10}(PO_4)_6(OH)_2$) crystal formation. In addition to releasing said calcium and phosphate ions, bicarbonate is also released to neutralise protons released during the crystallisation process, which would otherwise inhibit the calcium and phosphate precipitation during mineralisation.

    2. Smooth-ended phase: kallikrein-4 is released to eliminate proteins not degraded by enamelysin in the secretory phase. Degraded matrix proteins and water are removed, supported by the stratum intermedium which controls the fluid diffusion of the ameloblasts.

    3. Cyclical interconversion of these phases, until mineralisation is complete.

Tooth Calcification: Timeline

As no singular influencer has yet been found the etiology of MIH has been attributed to multifactorial causes[8] which, per the normal enamel thickness of MIH, suggesting the secretory phase of MIH is unaffected, the ameloblasts would need to be affected during the overlapping maturation phases of the affected teeth:

Average Chronology of Permanent Tooth Development [9]						
TOOTH	Calcification Begins		Crown Completion		Root Completion	
	Maxilla	Mandible	Maxilla	Mandible	Maxilla	Mandible
Central incisor	3rd month	3rd month	4.5 year	3.5 year	10.5 yr.	9.5 yr.
Lateral incisor	11th month	3rd month	5.5 year	4th year	11th yr.	10th yr.
Canine	4th month	4th month	6th year	5.75 year	13.5 yr.	12.75 yr.
1st premolar	20th month	22nd month	7th year	6.75 year	13.5 yr.	13.5 yr.
2nd premolar	27th month	28th month	7.75 year	7.5 year	14.5 yr.	15th yr.
1st molar	32nd w (IUL)	32nd w (IUL)	4.25 year	3.75 year	10.5 yr.	10.75 yr.
2nd molar	27th month	27th month	7.75 year	7.5 year	15.75 yr.	16th yr.
3rd molar	8th year	8th year	14th year	14th year	22nd yr.	22nd yr.

Tooth Calcification: Timeline

As seen in the previous table, the maturation (calcification) phase of amelogenesis commences with the first primary molars (FPMs) at approximately the 8th month in utero, followed by the central incisors of the upper and lower arches and the lateral incisors of the lower arch at approximately the 3rd month postpartum.

As calcification of the canines commences in both the upper and lower arches at approximately the 4th month postpartum, it may be reasonable to expect that the canines may be affected too. One recent study in Norway showed that one participant in four (25%) with MIH, had at least one affected canine.

Similarly given that the calcification for the lateral incisors of the upper arch commences considerably later, at approximately the 11th month postpartum, one may expect these to be less affected by MIH than their mandibular counterparts. A Greek study indicating that the mandibular lateral incisors were 1.6x more likely to be affected by MIH that their maxillary counterparts, whereas an Italian study put this figure at 2.9x more.

Second primary molars – indicators? **HSPM** – ***Hypomineralised second primary molars***

Physiology

The ameloblasts are considered most vulnerable during the transition from the secretory to the maturation stage, a post-secretory/pre-maturation stage during which time the cells of the enamel organ undergo morphological and functional changes.[14]

The full thickness of the enamel tissue has been realised, the stratum intermedium (which control the fluid diffusion - in and out - of the ameloblasts) is reduced to a single cell layer, fenestrated capillaries invade the enamel organ coming into close proximity of the reduced stratum intermedium layer thereby facilitating (indirect) access of the ameloblasts to the circulation.

During the transition stage the (amelogenin degraded) peptides are (further) degraded by kallikrein-4, are removed (by mechanism as yet unknown) and replaced by fluid, producing a highly porous and hydrated enamel tissue. The distal tight junctions of the (ruffle-ended) ameloblasts are still forming, leaving relatively free diffusible access to this highly porous and hydrated enamel tissue surface.

Physiology

Access of the ameloblasts to the circulation, together with high porosity and high levels of hydration, with 'free' diffusible access to said enamel surface morphology, means that not only calcium and phosphate ions may be taken up by the enamel tissue during the secretory -> maturation transition stage, but foreign materials too.

Albumin, for example, has the ability to inhibit crystal growth, therefore any conditions leading to hyperaemia (i.e. trauma, early childhood diseases) may lead to leakage of albumin on the growing crystals causing delayed maturation and mineralisation.

Alkaline phosphatase, found in high concentration in the cells of the stratum intermedium whilst lacking in ameloblasts, role in biomineralisation is likely:

- indirect mineralisation by transport of phosphate from the fenestrated capillaries
- indirect mineralisation by elimination of mineralisation inhibitors such as inorganic pyrophosphate
- indirect role in the stages of cell differentiation per phosphorylation of organic macromolecules required for activities related to cell differentiation
- uptake of nutrients from the circulation (fenestrated capillaries) into the enamel organ

Therefore disruptions to the stratum intermedium may cause ameloblast MIH disturbances as well

Genetics, Proteins, Transporters

Amelogenesis relies on highly specific proteins for enamel biogenesis, the three major structural enamel proteins being: amelogenin, ameloblastin and enamelin.

Tooth development occurs in a predictable and inheritable manner: genetic defects affecting the enamel proteins were found not to cause any other somatic defects.

In two cohort studies carried out with Brazilian and Turkish children, it was found that individuals who carried the G allele for enamelin were up to 17 times more likely to be affected by MIH than their A allele carrying (control group) counterparts.

Genetically disrupted enamel proteins in knock-out mice studies, showing hypomineraisation:

- SLC4A4 gene encoding the (basally located HCO_3^-/Na^+) co-transporter NBCe1
  - interupting buffering function of H^+ released per hydroxyapatite crystal formation
- STIM1 and STIM 1/2 genes encoding components of the Ca^{2+} release activated Ca^{2+} (CRAC) channels, which mediate the store-operated Ca^{2+} entry (SOCE) pathway[23]
  - disrupting the major Ca^{2+} entry pathway during in the maturation stage

However according to the OMIM (Online Mendelian Inheritance in Man) the pathological phenotypes of these faulty genes in humans manifest as enamel hypoplasia (not hypomineralisation).

Etiology (Exogenous Noxia(e))

As tooth development is under a strict genetic control, genetic predisposition coupled with environmental factors, so-called 'exogenous noxia(e)' (ENE), are suspected in MIH aetiology.[25]

As calcification of the first permanent molars begins just prior to birth, with crown completion around the fourth year after age, studies (retrospective and prospective) focus on the prenatal, perinatal and postnatal periods of child development.

Suspected MIH causing ENEs

Prenatal Factors
• Hypocalcemia
• A and D hypovitaminosis
• Diabetes mellitus (type I)
• Rubella
• Urinary tract infections

Perinatal Factors
• Caesarean section
• Prolonged delivery
• Twin delivery
• Infant hypoxia
• Very low birth weight
• Neonatal hypocalcemia
• Cyanosis

Postnatal Factors
• Children with a general disease
• Prolonged breast feeding (dioxin exposure)
• Hypocalcemia
• Nutritional problems
• Chicken pox, measles, rubella and other viral infection with high fever
• Respiratory diseases (asthma, lung problems)
• Antibiotics administration
• Anti-asthmatic medication
• Otitis media
• Thyroid and parathyroid disturbances

Etiology (Exogenous Noxia(e))

Study reviews and meta-analysis studies on MIH largely draw the same conclusions: exact etiological understandings concerning the development of MIH are lacking.

An MIH prevalence study, conducted by the Department of Paedodontics and Orthodontics at Semmelweis University to find the most likely etiological factors of the disease, revealed the same. Data (statistical) analysis was done using the Fisher's exact test. No statistical significances were found.

2-Way Summary Table: Observed Frequencies – Birth Complication				
	Birth Complication - 0	Birth Complication - 1	Row – Totals	
Control	15	3	18	
MIH	15	3	18	
Totals	30	6	36	p=1.0000

2-Way Summary Table: Observed Frequencies – Extended Nursing				
	Extended Nursing - 0	Extended Nursing - 1	Row – Totals	
Control	11	7	18	
MIH	16	2	18	
Totals	27	9	36	p=0.1212

2-Way Summary Table: Observed Frequencies – Infectious Disease				
	Infectious Disease - 0	Infectious Disease - 1	Row – Totals	
Control	12	6	18	
MIH	14	4	18	
Totals	26	10	36	p=0.7112

Source: Moldován A, Reichel L. The prevalence of Molar Incisor Hypomineralisation in the Hungarian population and the main etiological factors. SE Int St Conference 2017.

Etiology (Exogenous Noxia(e))

2-Way Summary Table: Observed Frequencies – Metabolism				
	Metabolism - 0	Metabolism - 1	Row – Totals	
Control	15	3	18	
MIH	18	0	18	
Totals	33	3	36	p=0.2280

2-Way Summary Table: Observed Frequencies – Allergy				
	Allergy - 0	Allergy – 1	Row – Totals	
Control	14	4	18	
MIH	17	1	18	
Totals	31	5	36	p=0.3377

2-Way Summary Table: Observed Frequencies – Respiratory				
	Respiratory - 0	Respiratory - 1	Row – Totals	
Control	11	7	18	
MIH	14	4	18	
Totals	25	11	36	p=0.4705

2-Way Summary Table: Observed Frequencies – Antibiotics				
	Antibiotics - 0	Antibiotics - 1	Row – Totals	
Control	13	5	18	
MIH	14	4	18	
Totals	27	9	36	p=1.0000

2-Way Summary Table: Observed Frequencies – Inherited				
	Inherited - 0	Inherited - 1	Row – Totals	
Control	15	3	18	
MIH	18	0	18	
Totals	33	3	36	p=0.2286

Source: Moldován A, Reichel L. The prevalence of Molar Incisor Hypomineralisation in the Hungarian population and the main etiological factors. SE Int St Conference 2017.

Treatment

Genetically encoded faulty enamel proteins may also affect the deciduous dentition. Second primary molars presenting with hypomineralisation (deciduous molar hypomineralisation (DMH); otherwise known as hypomineralised second primary molar - HSPM) were found to be a risk factor for the development of MIH. The odds ratio for MIH, based on HSPM, fell within the 95% confidence interval suggesting that, clinically, **HSPM could be used as an MIH predictor/indicator.**

Early diagnosis of MIH could be useful in treatment planning.

- The dietary and oral hygiene habits of the child could be assessed and early prophylactic interventions introduced.
- Implementation of corrective recommendation(s) could be monitored, minimising cariogenicity and erosivity whilst introducing/reinforcing positive oral hygiene habits prior to eruption of the first permanent molars.
- In this way posteruptive breakdown of the permanent first molars (PFM) could be managed, allowing for the earliest intervention possible, prior to enamel breakdown per masticatory forces exerted on the soft and porous enamel subsurface tissue.

In addition to early diagnosis a six-step approach to management of MIH described by William et al also includes risk identification, prior to eruption of the primary first molars (PFMs).

Treatment

Steps	Recommended Procedures
Risk Identification	Assess medical history for putative etiological factors
Early Diagnosis	• Examine at-risk molars on radiographs if available • Monitor these teeth during eruption
Remineralisation and desensitisation	Apply localised topical fluoride
Prevention of dental caries and posteruptive breakdown (PEB)	• Institute thorough oral hygiene home care program • Reduce cariogenicity and erosivity of diet • Place pit and fissure sealant
Restorations or extractions	• Place intracoronal (resin composite) restoration bonded with a self-etching primer adhesive or extracoronal restorations (stainless steel crowns) • Consider orthodontic outcomes post-extraction
Maintenance	• Monitor margins of restoration for PEB • Consider full coronal coverage restoration in the long term

The 'early diagnosis' criteria proposed by William et al. does not include HSPM clinical features as an MIH predictor and the putative factors of MIH are still conjecture at this point, however ***the benefits of preeruptive identification of MIH*** are clear: the clinician is able to formulate a treatment plan at the earliest possible stages of MIH, including interventive prophylactic measures, whilst educating the parents and patient alike on their continued roles at home (i.e. oral hygiene and dietary maintenance) in order to ensure the success of the treatment.

Treatment Summary of treatment modalities for PFMs

Steps	Recommended Procedures
Preventive	• Topical fluoride application • Desensitising toothpaste • Apply a CPP-ACP topical crème daily using a cotton bud • Glass ionomer cement (GIC) sealants can provide caries protection and reduce surface permeability
Direct restoration	• Cavity margin placement – all defective enamel is removed – only the very porous enamel is removed, until good resistance of the bur to enamel is felt • GIC restorations – conventional GIC, resin modified GICs (RMGIC) – adhesive capability to both enamel and dentin – long term fluoride release – Poorer mechanical properties · not recommended to be used in stress bearing areas · be used as an intermediate restoration • Composite resin restorations – longer-term stability compared with other restorative materials – the polyacid modified resin composites (PMRC→compomers) · have good handling characteristics · release and take up fluoride · have tensile and flexural strength properties superior to GIC and RMGIC, but inferior to that of resin composite · use of PMRCs in permanent teeth is restricted to no stress-bearing areas

Treatment

Summary of treatment modalities for PFMs

Steps	Recommended Procedures
Full coverage restoration	• When PFMs have moderate to severe PEB, preformed stainless steel crowns (SSCs) are the treatment of choice – prevent further tooth deterioration – control tooth sensitivity – establish correct interproximal contacts and proper occlusal relationships – are not as technique sensitive or costly as cast restorations – require little time to prepare and insert – if not adapted properly may produce an open bite, gingivitis or both – properly placed, SSCs can preserve PFMs with MIH until cast restorations are feasible • Partial and full coverage indirect adhesive or cast crown and onlays – compared to SSCs, cast restorations · require minimal tooth reduction · minimise pulpal trauma · protect tooth structure · provide high strength for cuspal overlays · control sensitivity · maintain periodontal health due to their supragingival margins
Extraction and orthodontic consideration	• Timely extraction is a feasible treatment option in cases of – severe hypomineralisation – severe sensitivity or pain – large multi-surface lesions – difficulty of restoration – inability to achieve local anaesthesia – behaviour management problems preventing restorative treatment – apical pathology – orthodontic space requirements, where PFMs are heavily restored in the presence of healthy premolars – crowding distally in the arch and third permanent molars reasonably positioned – financial considerations precluding other forms of treatment • If the orthodontic condition were favourable, the ideal dental age for extracting the defective PFM would be 8.5 to 9 years of age

MOLARIS INCISIVUS HYPOMINERALISATIO
MIH - Sindrome

Molars:

- Fissure sealing
- Atraumatic restoration
- Composite restoration
- Preformed metal crown
- Indirect restoration
- Extraction of molar tooth

MOLARIS INCISIVUS HYPOMINERALISATIO
MIH - SZINDRÓMA

Front teeth:

Microabrasio:

- Enamel max, 100micron removal
- 18% hydrogen chloride or 37.5% orthophosphoric acid
- Abrasive polish
- After treatment: remineralisation

Bleach-etch-seal technique:

- Wright: tooth bleaching with 5% sodium hypochlorite 20mp, followed by 37% orthophosphoric acid, followed by liquid resin

Resin infiltration-ICON

- Low viscosity resin infiltrates demineralised enamel

External tooth whitening:

- From adolescence
- Side effects: gingival irritation, tooth hypersensitivity

Composite restorations/veneers

Treatment

Treatment success is most likely affected by the presence of treatment complications, therefore management thereof is likely an important part of any MIH related treatment plan. Some of these will require education of and cooperation of the patient:

- Regular home usage of desensitising toothpaste reducing tooth hypersensitivity, thereby reducing poor oral hygiene associated rapidly developing tooth decay.
- Application of Tooth Mousse: topical cream providing calcium and phosphate ions to the enamel prisms, allowing formation of apatite crystalline structures.
- Application of Elmex gel: intensive anti-decay prophylaxis against demineralisation.
- Disciplined diet to reduce cariogenicity and erosivity.

Difficulty in achieving anaesthesia may be managed using the QuickSleeper or SleeperOne (electronic dental *anaesthesia)* systems, or the Wand Plus (computer-controlled local *anaesthetic* delivery) system.

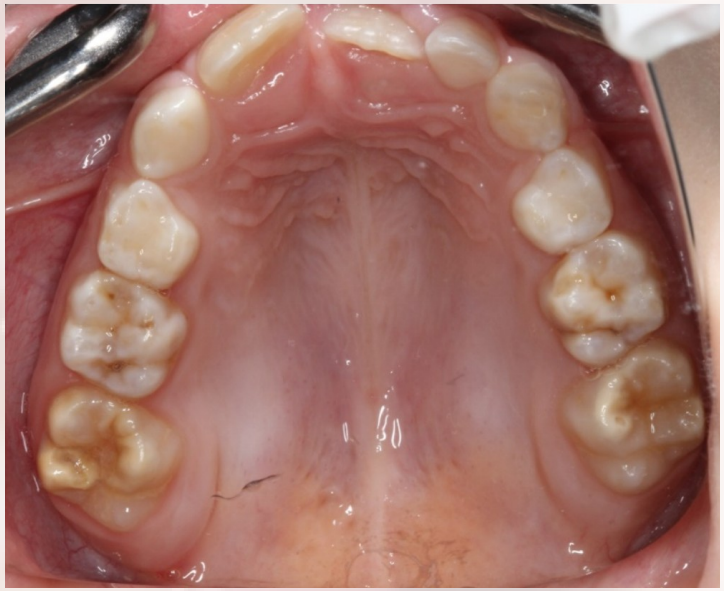
The extent of the remaining treatment complications:

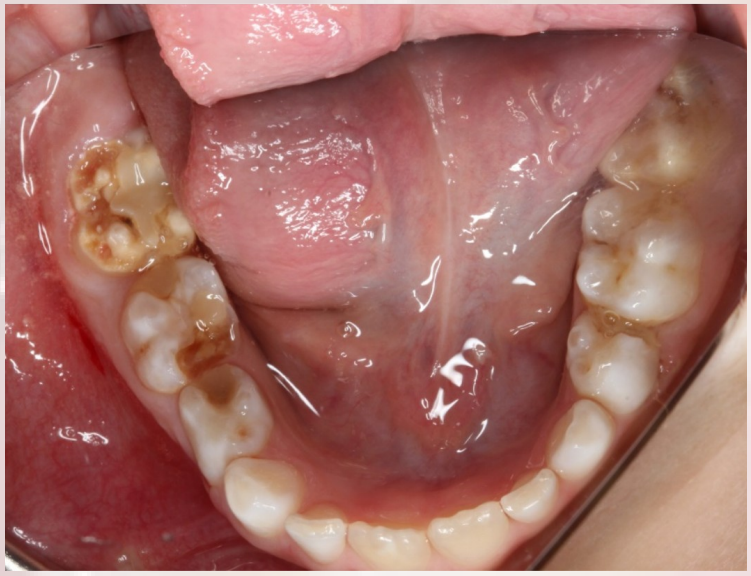
- Difficulty in determining how much affected enamel to remove.
- Selection of a suitable restorative material.
- Repeated marginal breakdown of restorations.

will depend on the success of the clinical handling, treatment and progress of treatment of MIH which will be determined by an early MIH diagnosis, prevention of decay (fissure sealing) and prevention of loss of enamel tissue structure due to masticatory forces, whereas other factors will be up to the creativity of, or possibly even beyond the control of, the clinician to manage (i.e. limited cooperation of a young child).

MIH - SYNDROME

- Enamel developmental defect
- *ENDOGRNOUS - SYSTHEMIC*
- *localised, asymmetric!*





Source: Kellerhof NM, Lussi A. Molar-Incisor-Hypomineralization. Schweiz Monatsschr Zahnmed. Vol 114:3/2004

MOLARIS INCISIVUS HYPOMINERALISATIO
MIH - SZINDRÓMA

DDE – Index (FDI, 1992)
Developmental Defects of Enamel – Index

- **2003: at the EAPD Congress the Weerheijm et al. MIH judgement criteria (classification) was adapted, allowing reproducibility of future study parameters:**

  - ***Absence or presence of demarcated opacities***
  - ***Posteruptive enamel breakdown***
  - ***Atypical restorations***
  - ***Extraction of molars due to MIH***
  - ***Failure of eruption of a molar or incisors***

Forrás: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

MIH – „Molar-incisor hypomineralisation”

The Würzburg MIH concept:
The MIH treatment need index (MIH TNI)
R. Steffen, N. Krämer, K. Bekes; 2017

Index	Definition
0	No MIH, clinically free of MIH
1	MIH without hypersensitivity, without defect
2	MIH without hypersensitivity, with defect
2a	<1/3 defect extension
2b	>1/3 <2/3 defect extension
2c	>2/3 defect extension or/and defect close to the pulp or extraction or atypical restoration
3	MIH with hypersensitivity, without defect
4	MIH with hypersensitivity, with defect
4a	<1/3 defect extension
4b	>1/3 <2/3 defect extension
4c	>2/3 defect extension or/and defect close to the pulp or extraction or atypical restoration

Forrás: Steffen, R., Krämer, N. & Bekes, K. The Würzburg MIH concept: the MIH treatment need index (MIH TNI). *Eur Arch Paediatr Dent* **18,** 355–361 (2017).

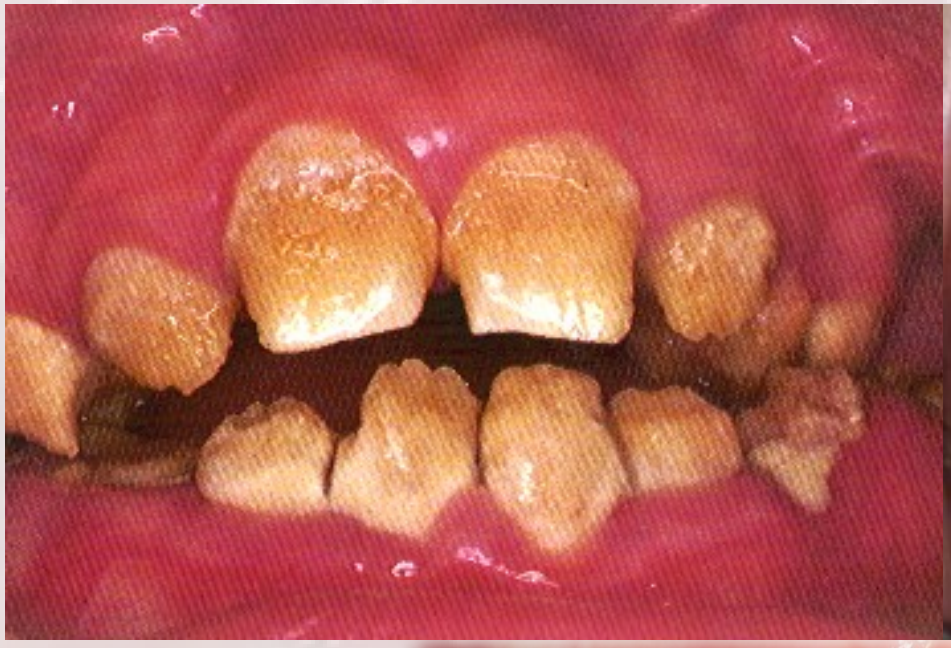
MIH – SYNDROME
Diagnostic criteria

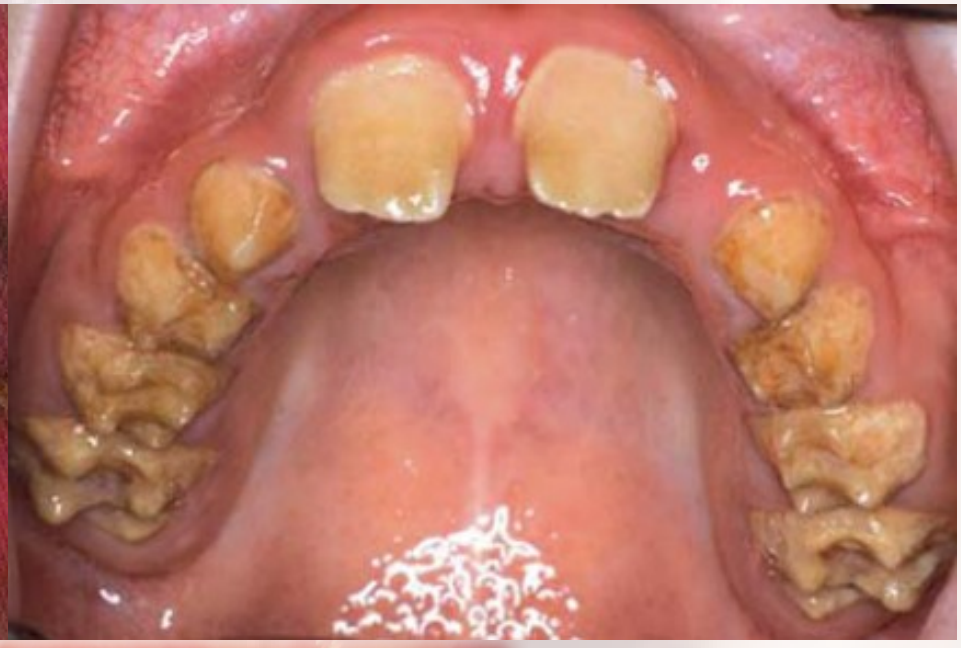
Code	Criteria
0	Enamel defect free
1	White/creamy demarcated opacities, no PEB
1a	White/creamy demarcated opacities, with PEB
2	Yellow/brown demarcated opacities, no PEB
2a	Yellow/brown demarcated opacities, with PEB
3	Atypical restoration
4	Missing because of MIH
5	Partially erupted (i.e., less than one-third of the crown high) with evidence of MIH
6	Unerupted/partially erupted with no evidence of MIH
7	Diffuse opacities (not MIH)
8	Hypoplasia (not MIH)
9	Combined lesion (diffuse opacities/hypoplasia with MIH)
10	Demarcated opacities in incisors only

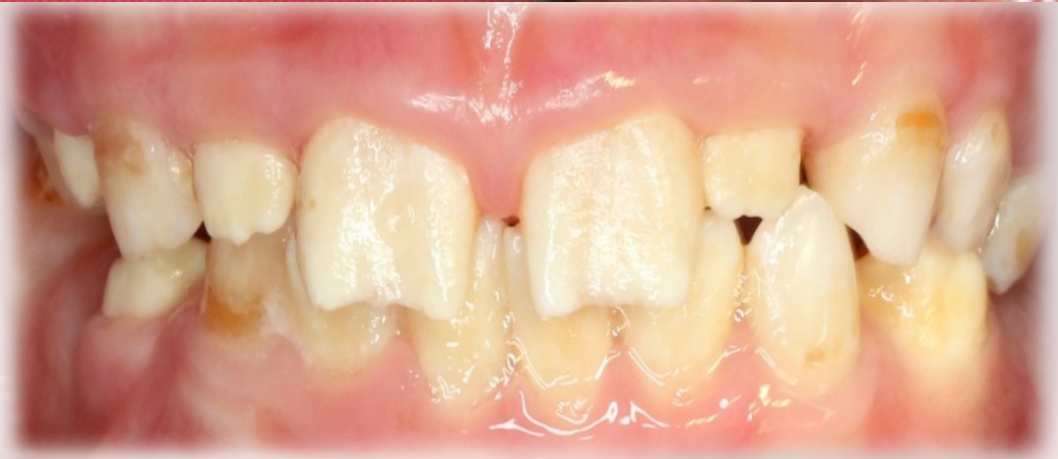
MIH: Molar incisor hypomineralisation, PEB: Posteruptive enamel breakdown, EAPD: European academy of pediatric dentistry

DIFFERENCIAL DIAGNOSIS:

- Amelogenesis imperfecta hereditaria



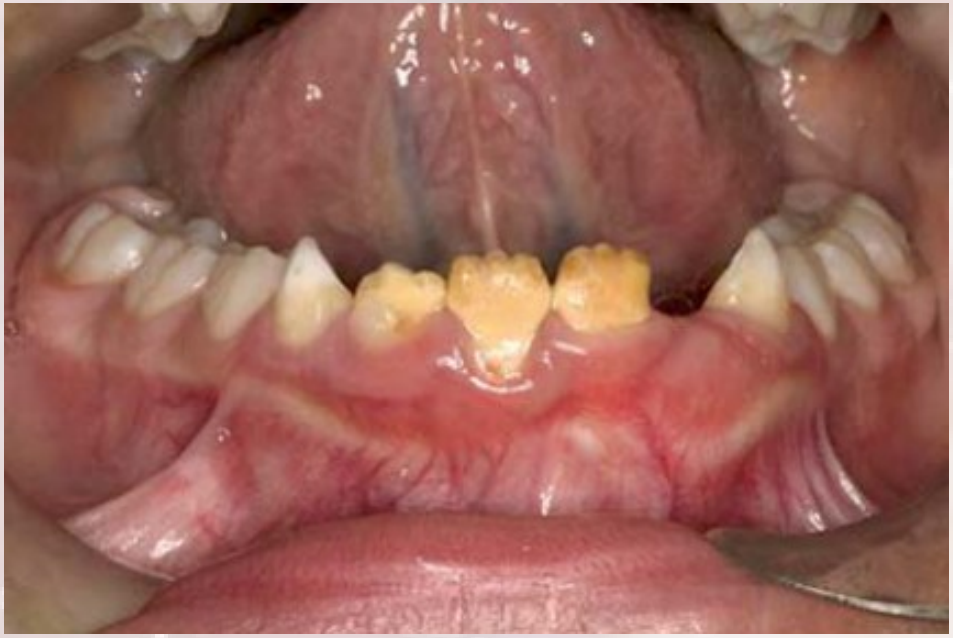


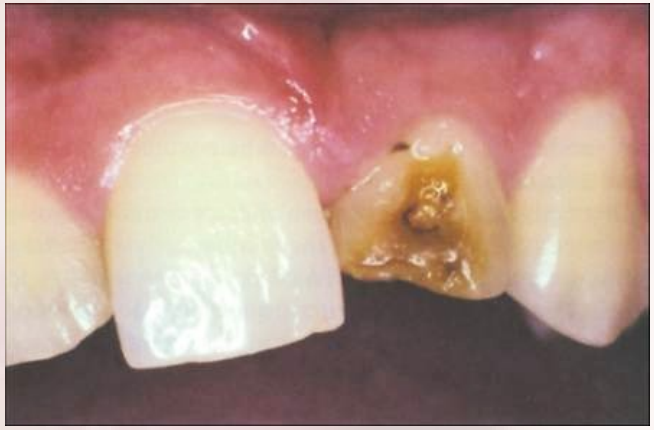


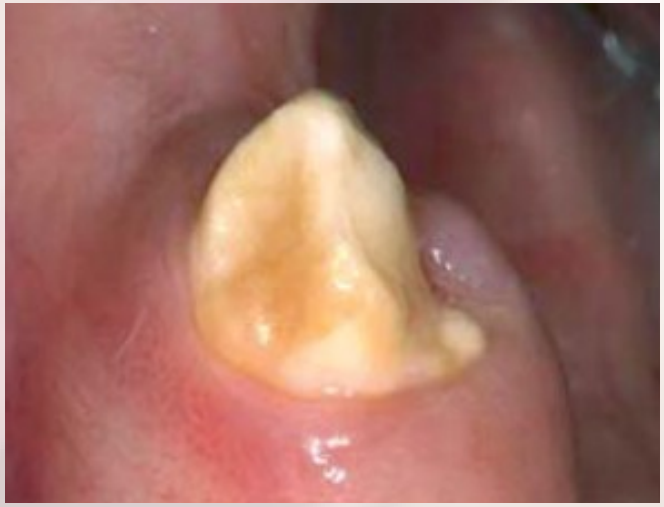
Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.
SE-FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga.

DIFFERENCIAL DIAGNOSIS:

- Enamelhypoplasia – intrusio, inflammatio







- ! Localized

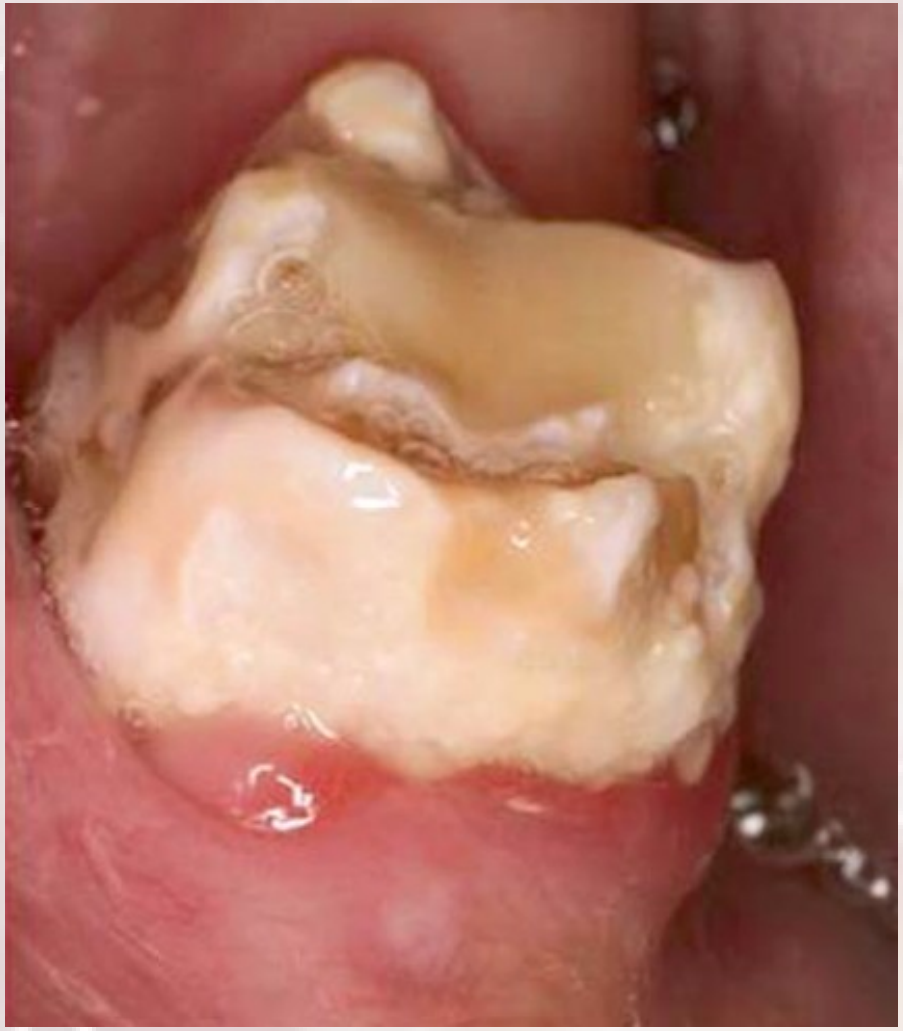
Turner - tooth

Source:

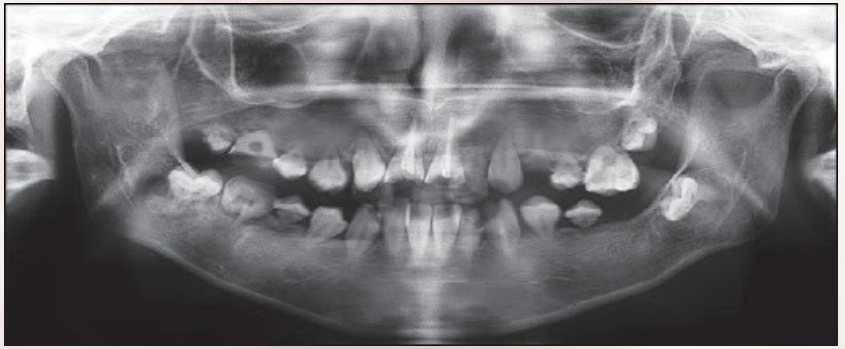
- *Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.*
- *SE FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga.*

DIFFERENCIAL DIAGNOSIS:

- Enamelhypoplasia – tumor therapy, iradiation



- Aplasia
- Microdontia
- Enamel hypoplasia
- Root developmental defects

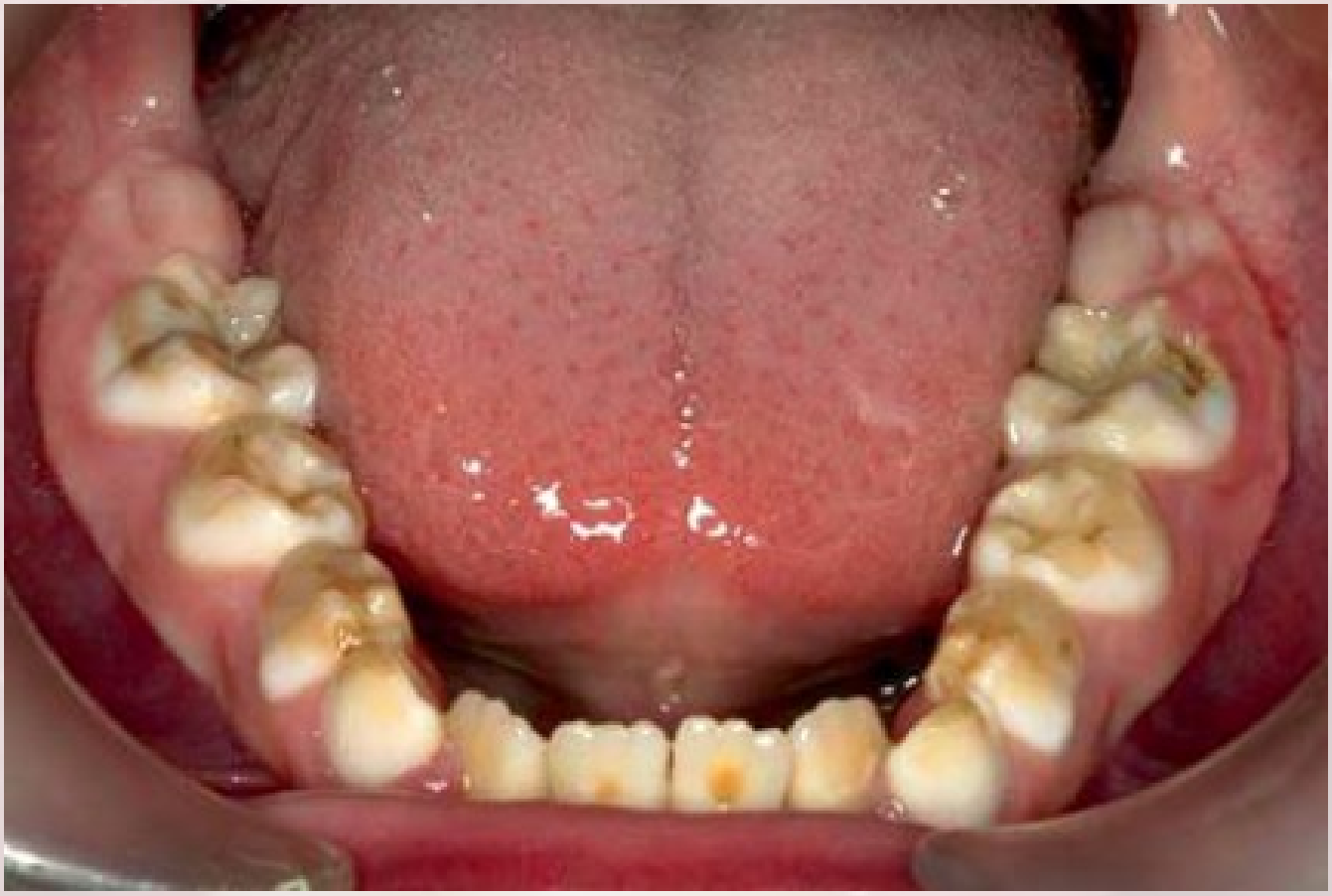


Source:

- *Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.*
- *SE FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga.*

DIFFERENCIAL DIAGNOSIS:

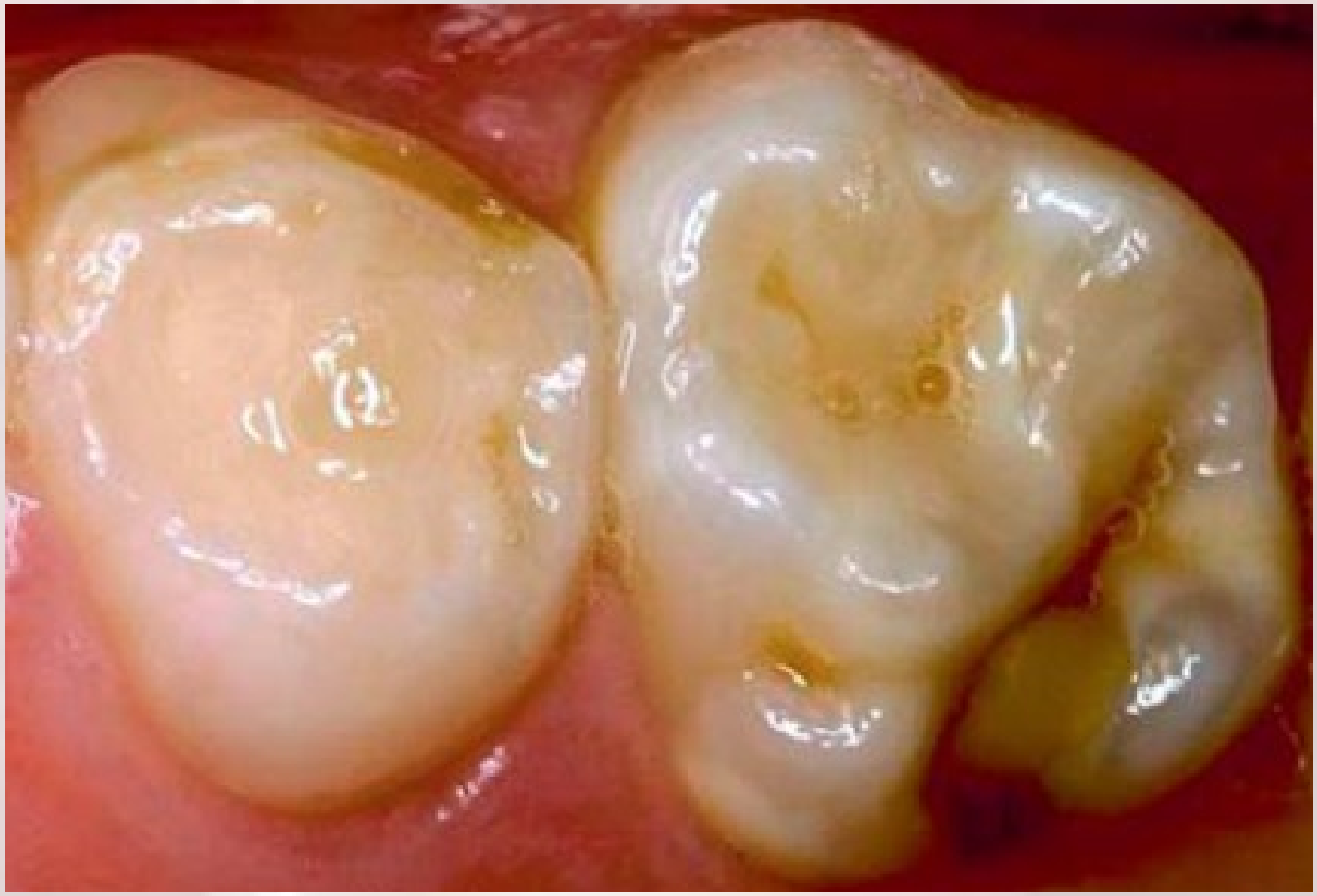
- Enamel hypoplasia – Rhesusfaktor-incompatibility



Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

DIFFERENCIAL DIAGNOSIS:

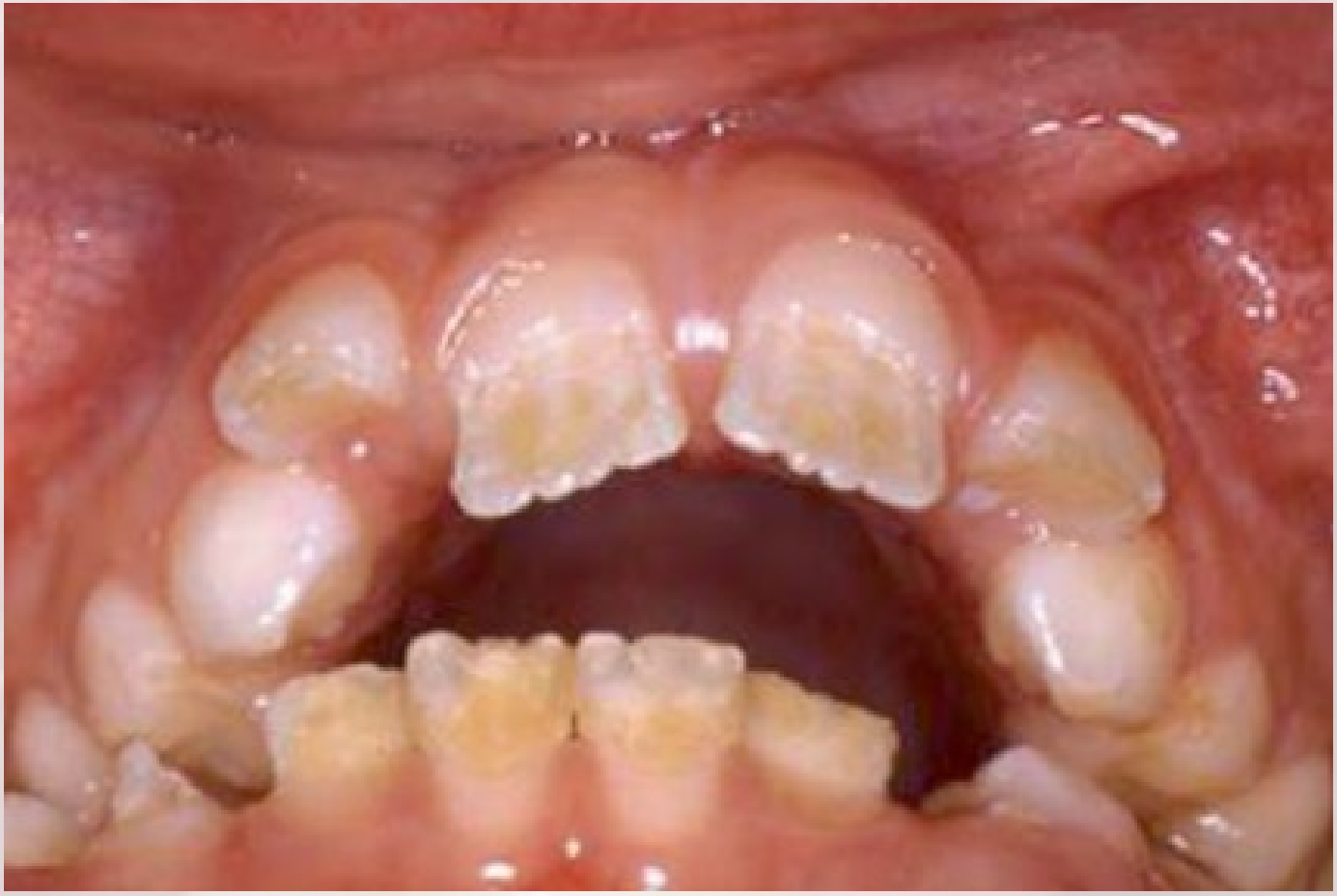
- Enamel hypoplasia – newborn icterus



Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

DIFFERENCIAL DIAGNOSIS:

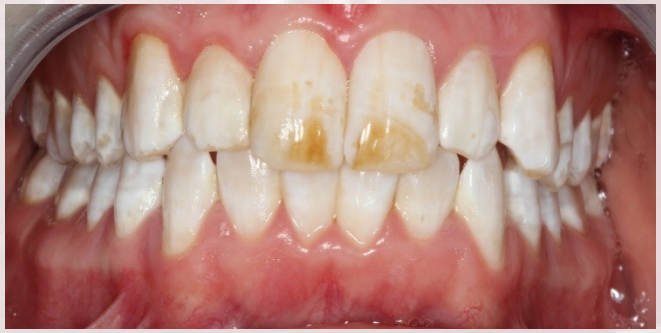
- Rachitis

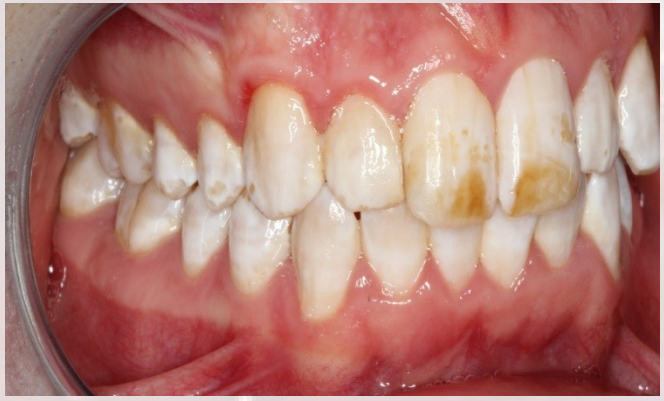


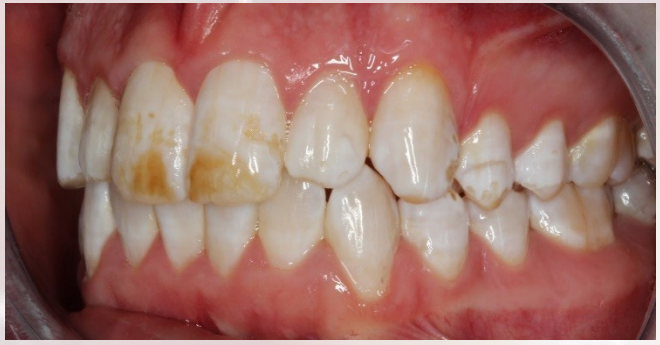
Forrás: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

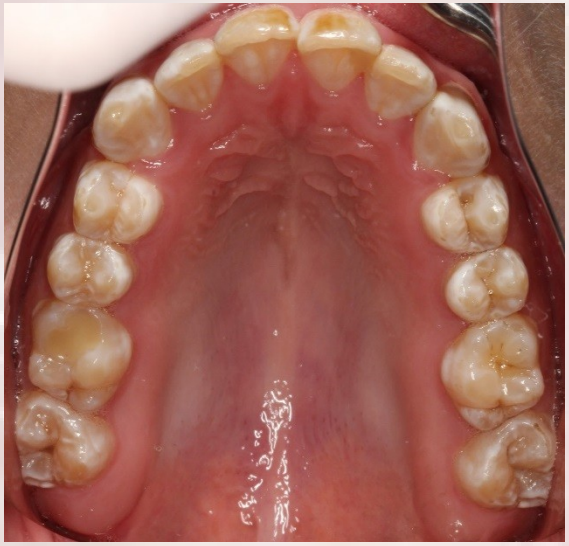
DIFFERENCIAL DIAGNOSIS:

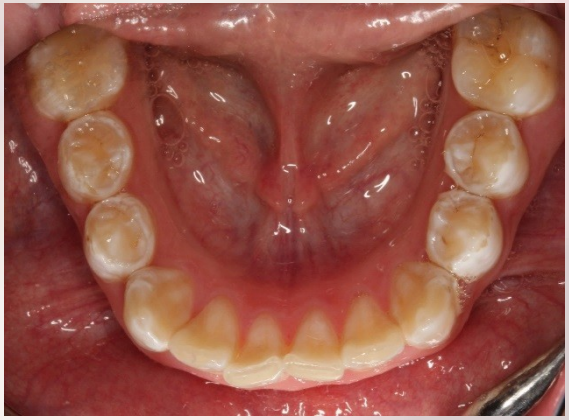
- *Fluorosis endemica dentium*

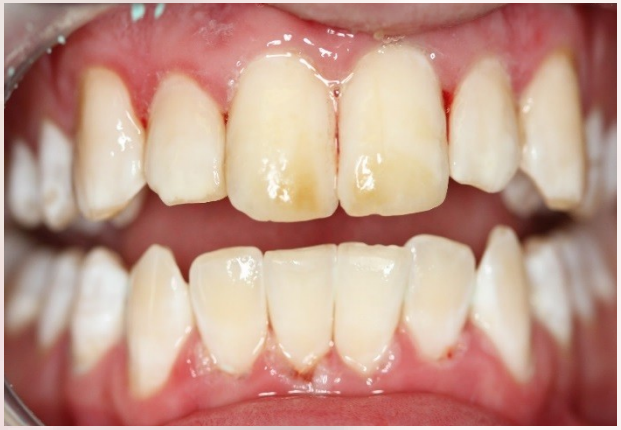












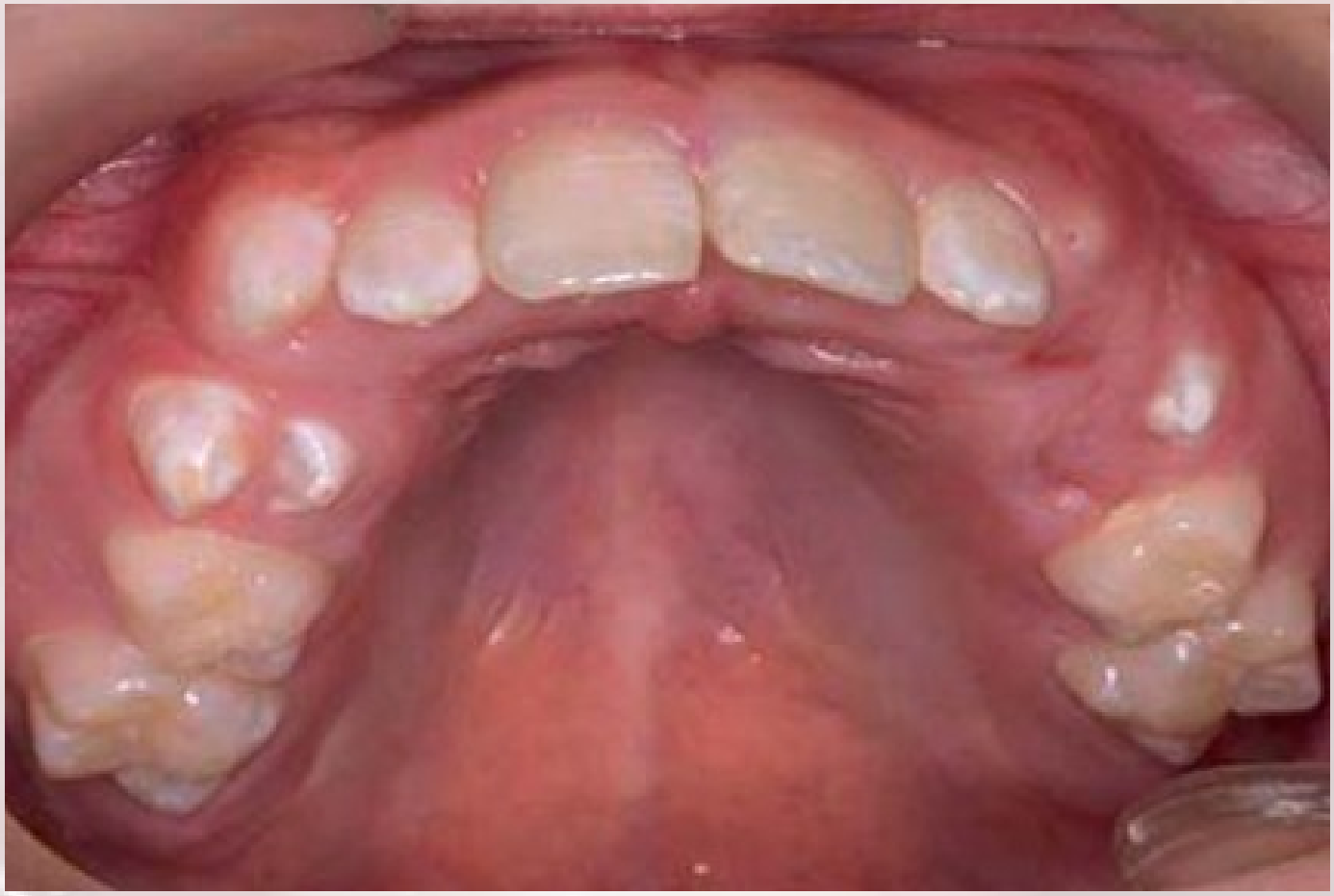
4x ICON® (DMG)
Kariesinfiltratio

♂, Dentocar tabl.

Source: SE FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga.

DIFFERENCIAL DIAGNOSIS:

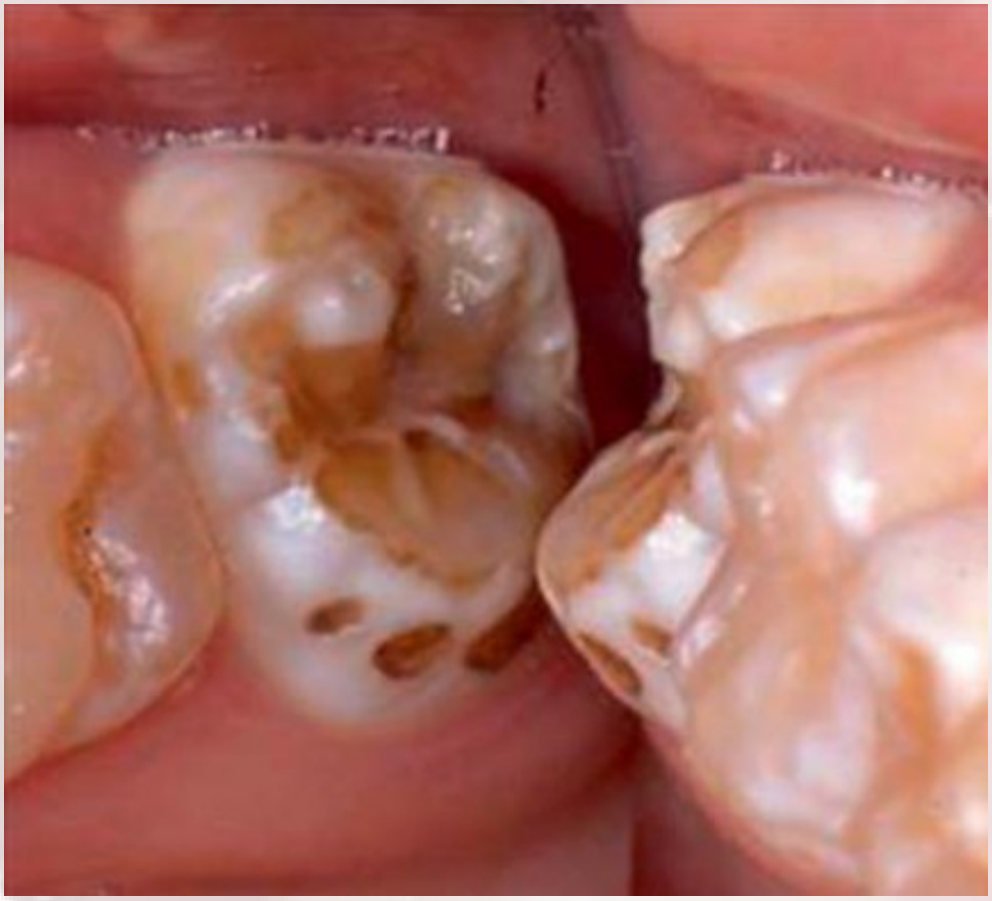
- Fluorosis endemica dentium
  - ➢Thylstrup and Fejerskov scale: 2-3



Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

DIFFERENCIAL DIAGNOSIS:

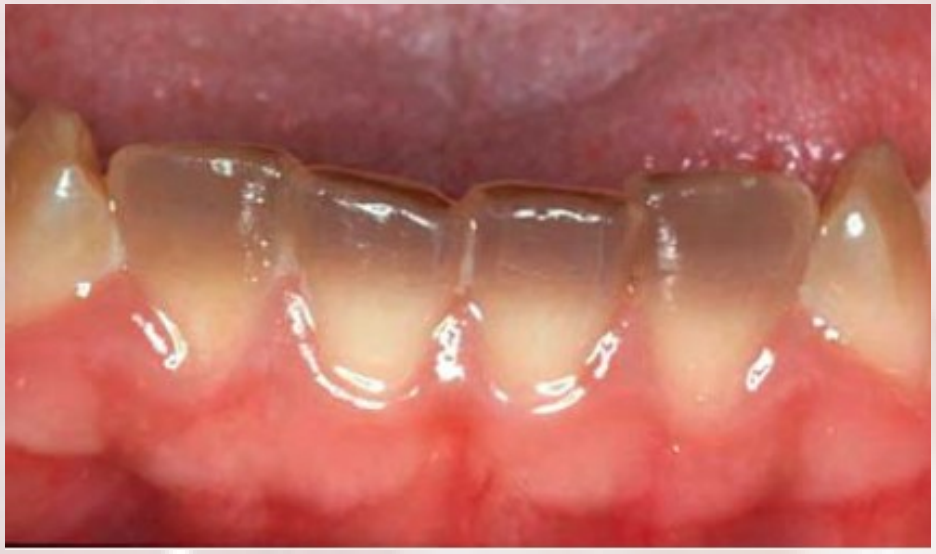
- Fluorosis endemica dentium
  - Thylstrup and Fejerskov: 7

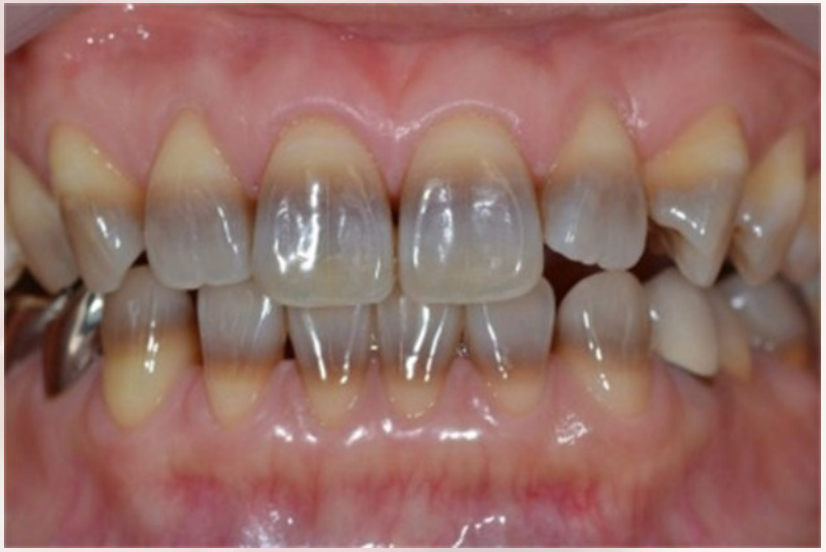


Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

DIFFERENCIAL DIAGNOSIS:

- Tetraciklin - Xanthodontia



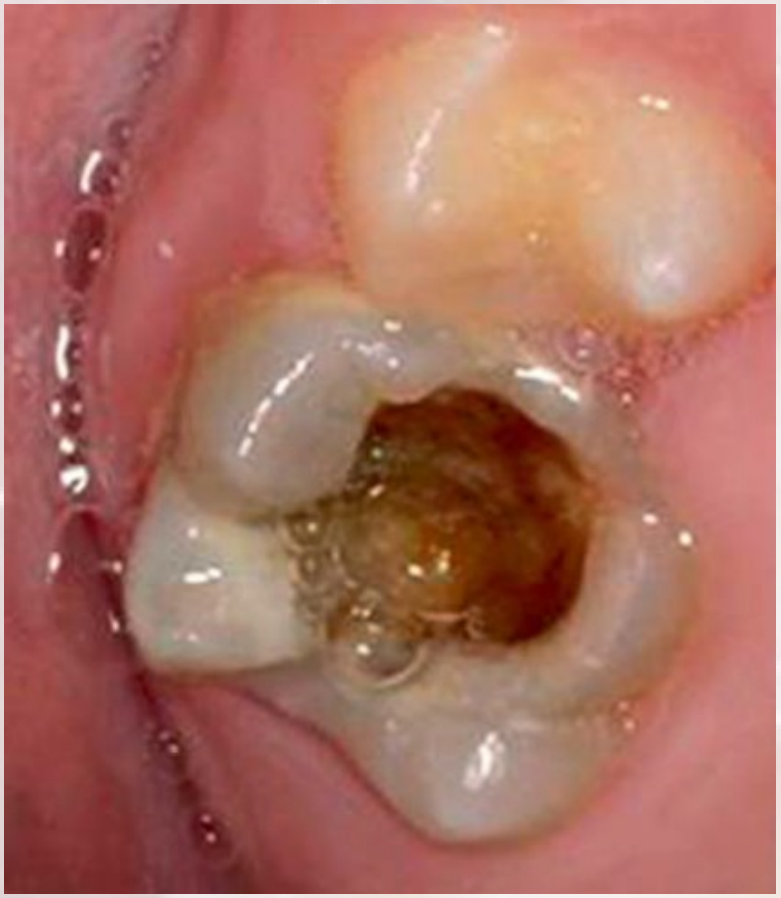


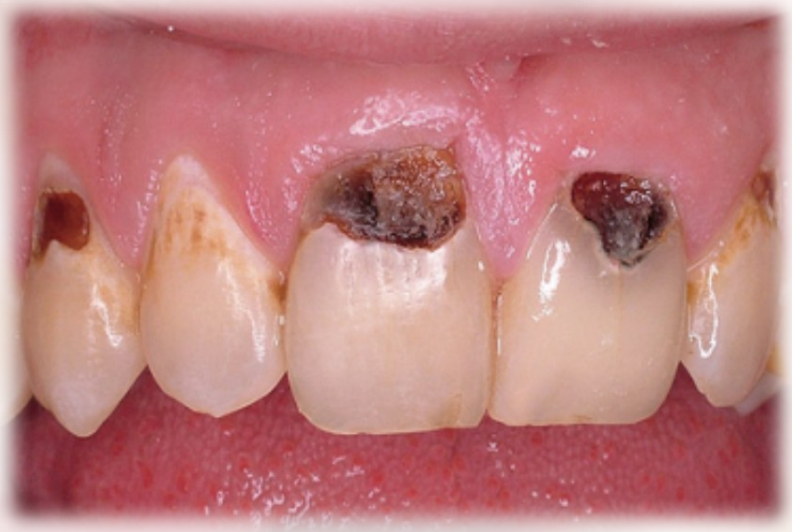
Source:

- *Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.*
- *SE FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga.*

DIFFERENCIAL DIAGNOSIS:

- Caries





Source:
Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.
SE-FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga.

CLASSIFICATION:

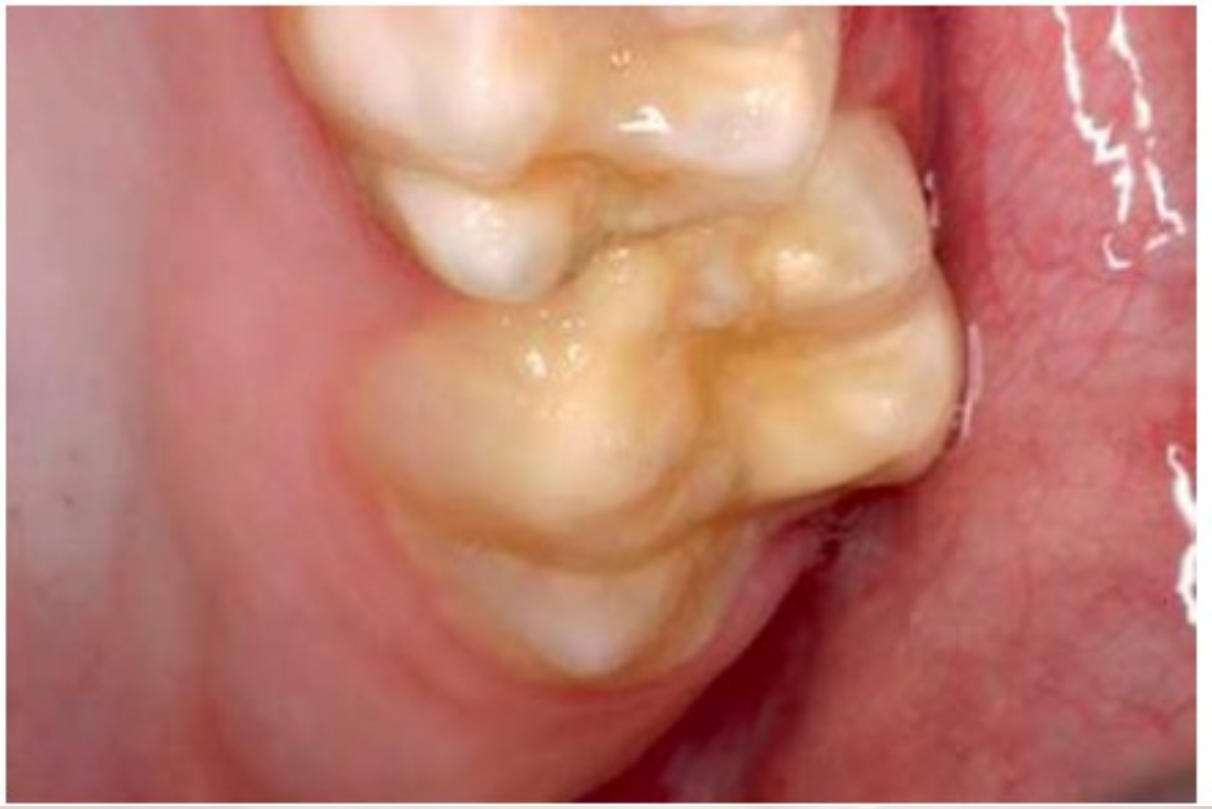
Wetzel and Reckel

Alaluusua et al.

Mathu-Muju and Wright (2006)

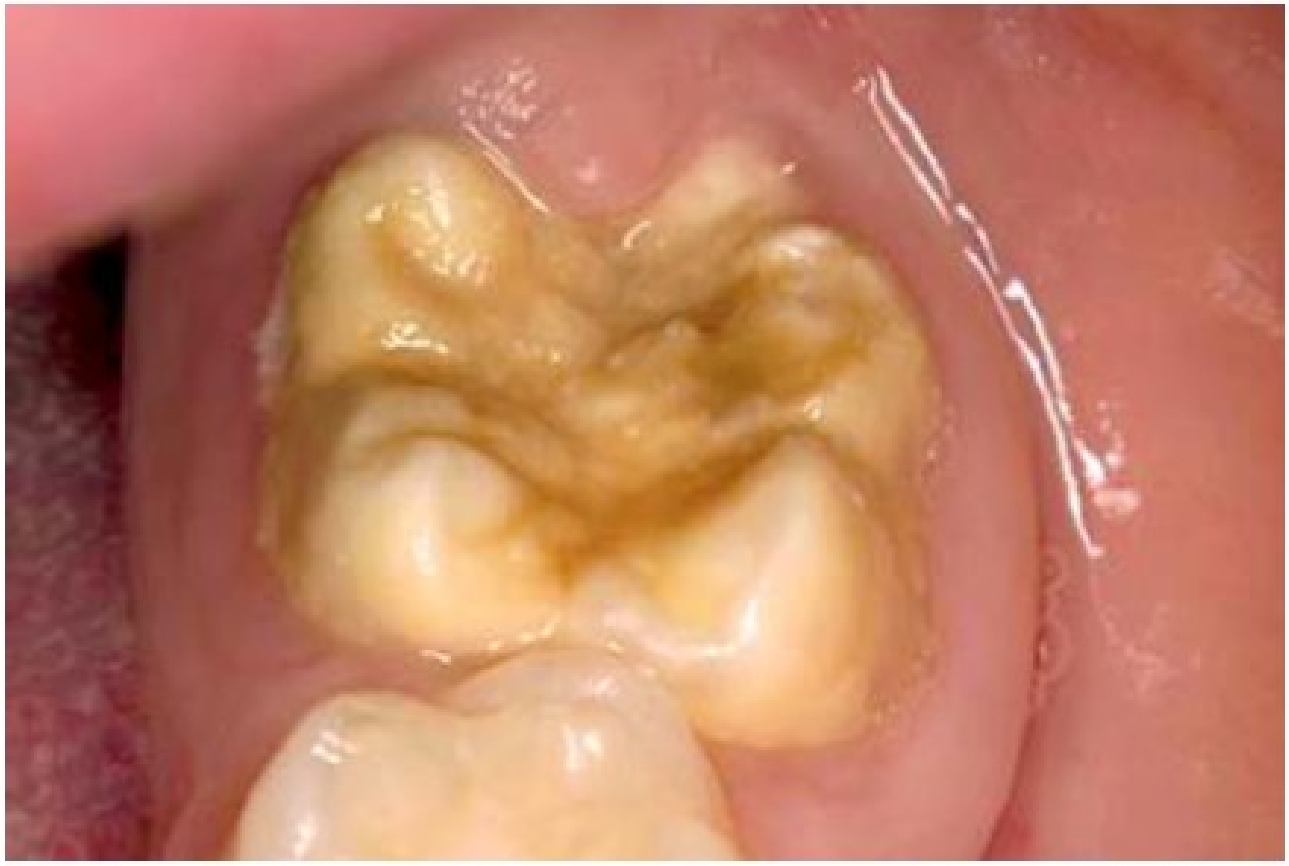
- *Mild*
- *Medium*
- *Severe*

Wetzel and Reckel: 1, MILD



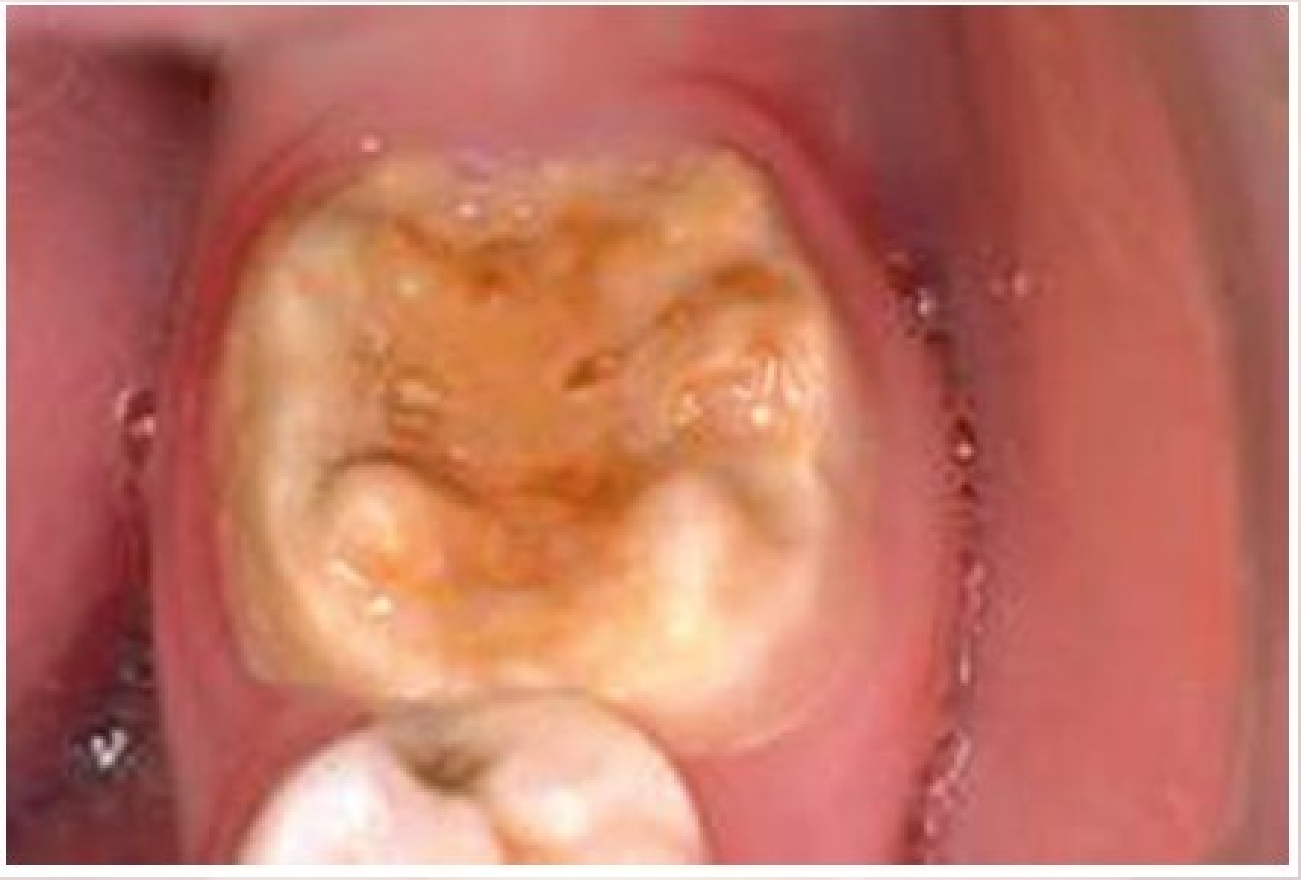
Forrás: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

Wetzel and Reckel: 2 medium



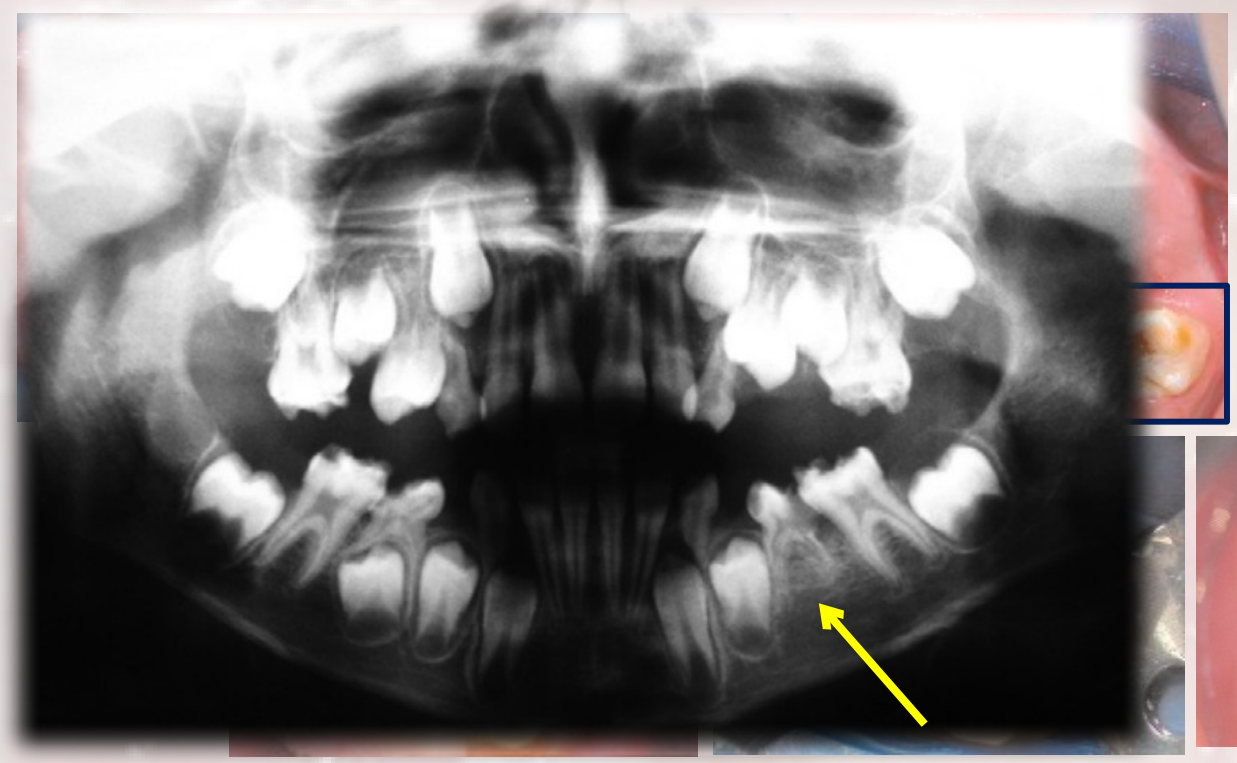
Forrás: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

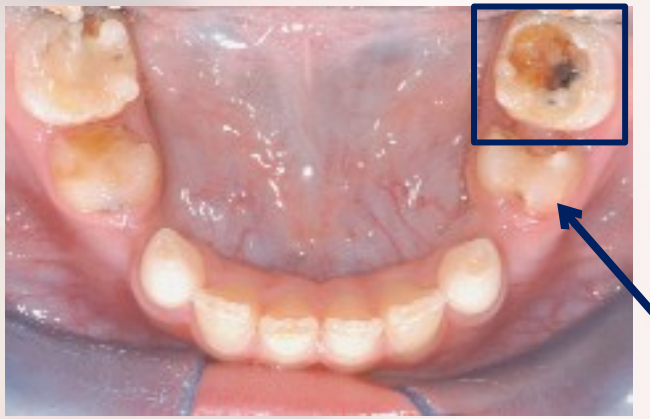
Wetzel and Reckel: 3 severe

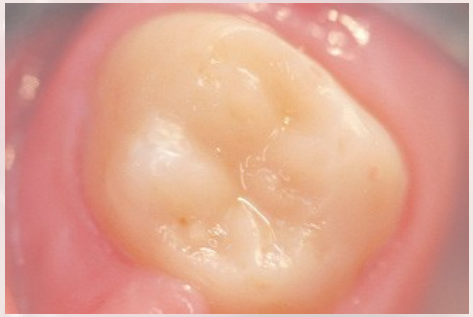


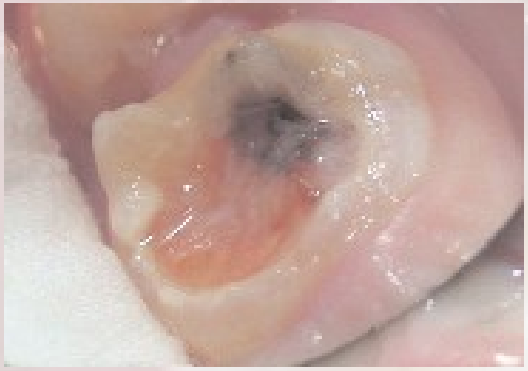
Forrás: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

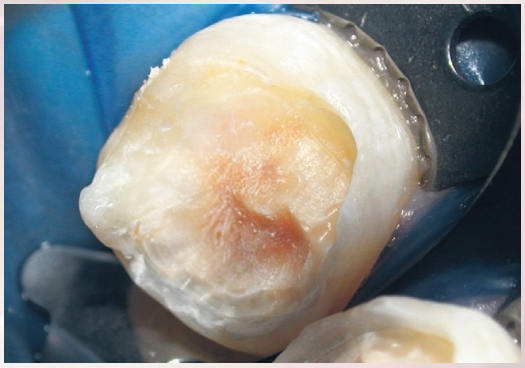
MIH - SYNDROME

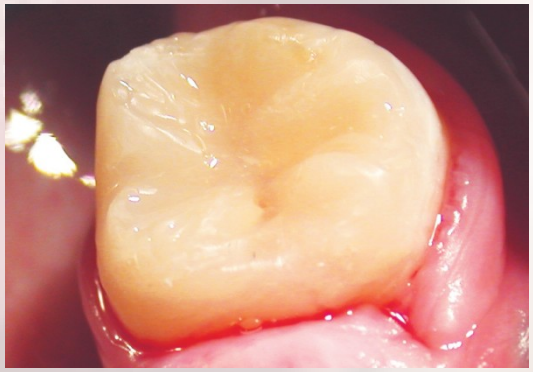








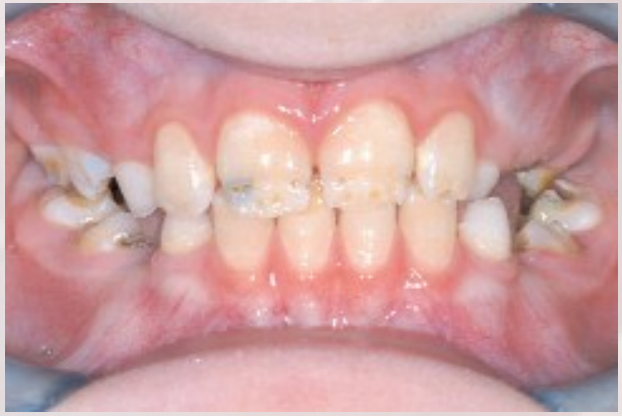
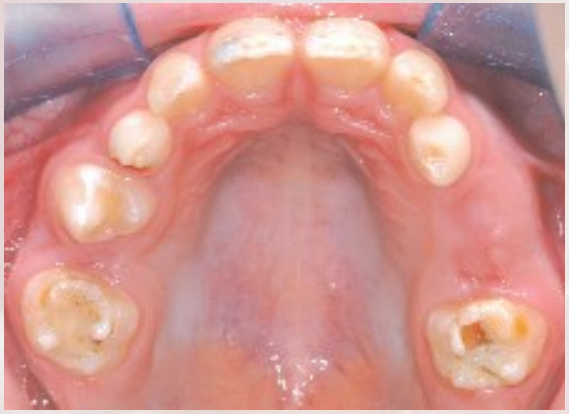
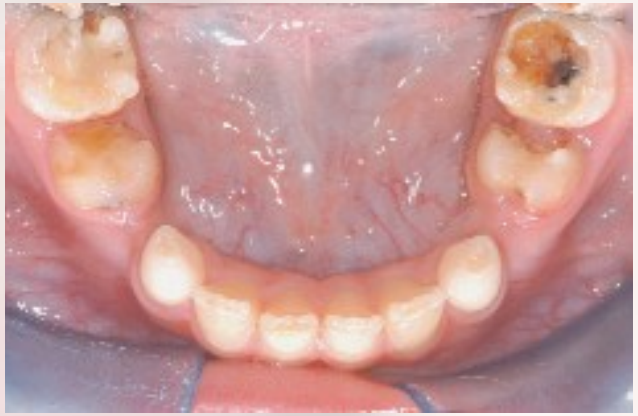
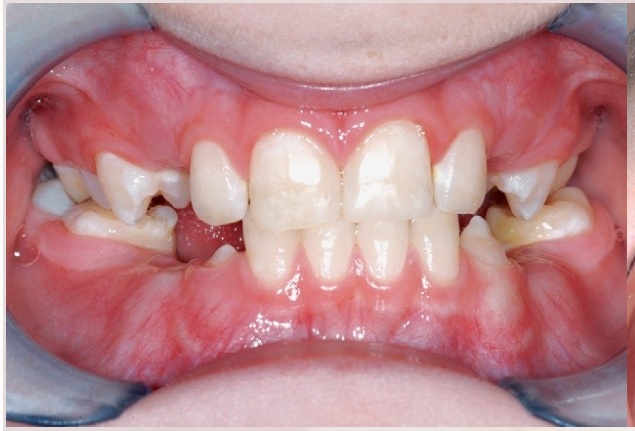
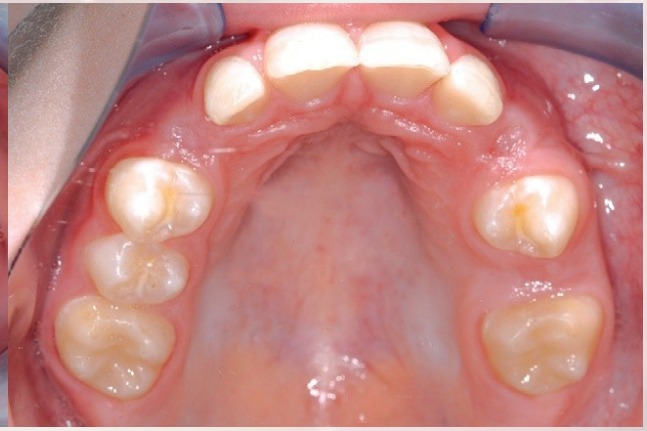
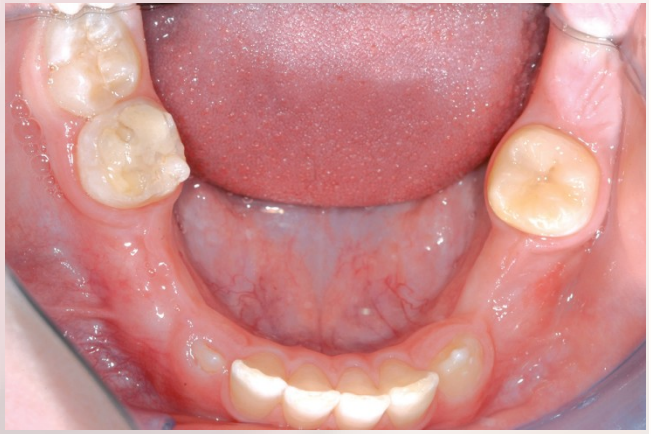




Source: Giraki M: Konservierende Restauration von Zähnen mit Schmelzhypoplasien. Oralprophylaxe und Kinderzahnheilkunde 2006; 28(4):164-168.

MIH - SYNDROME

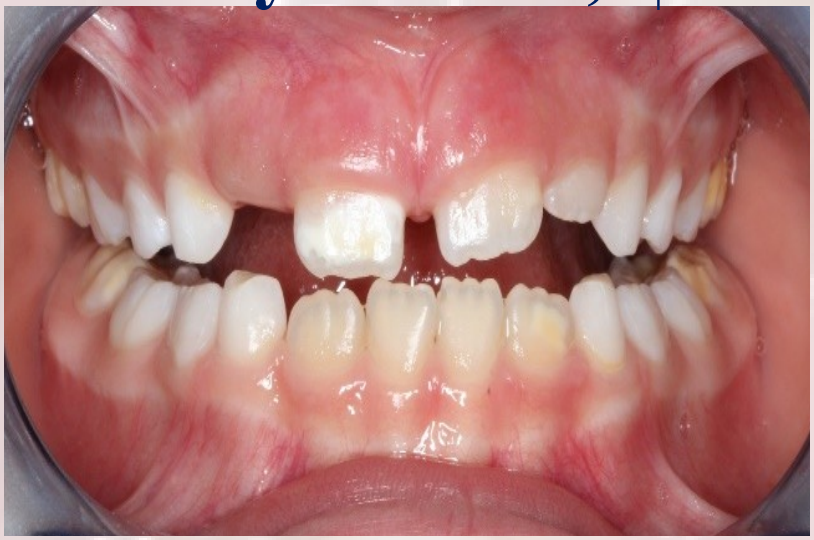
9 years old

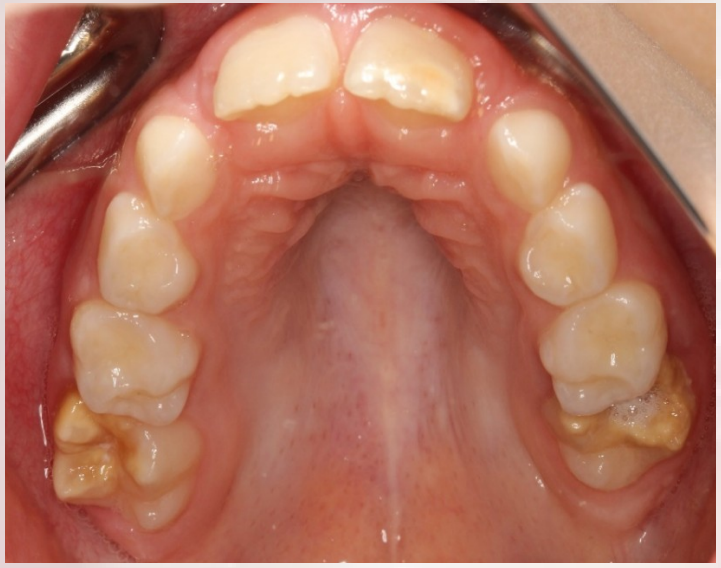







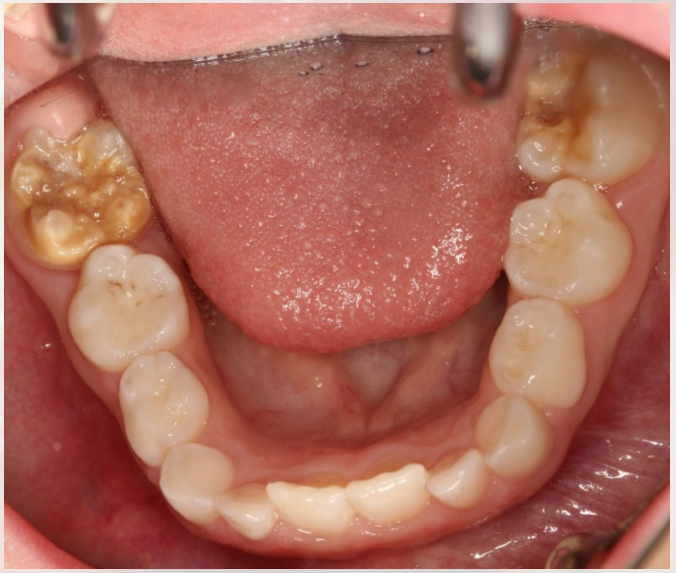
Source: Giraki M: Konservierende Restauration von Zähnen mit Schmelzhypoplasien. Oralprophylaxe und Kinderzahnheilkunde 2006; 28(4):164-168.

MIH - SYNDROME

7 years old, ♀



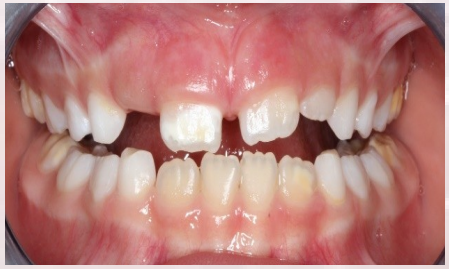


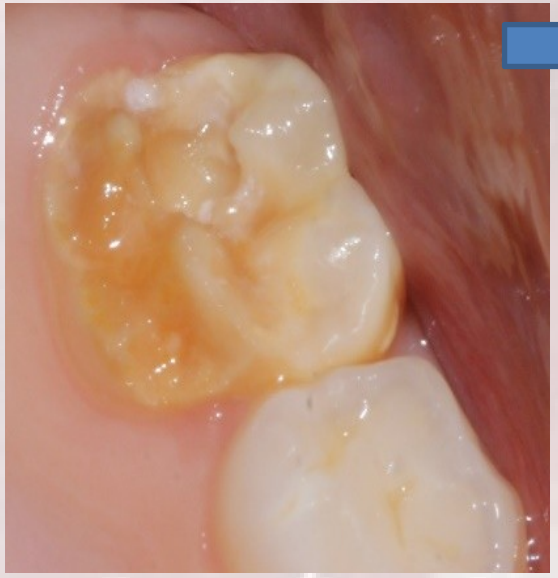


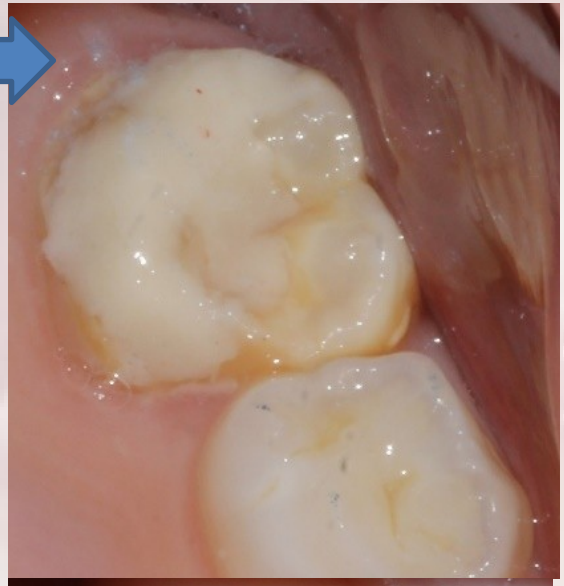
Source: Esetbemutatás – SE-FOK Gyermekfogászati és Fogszabályozási Klinika. E. Mlinkó

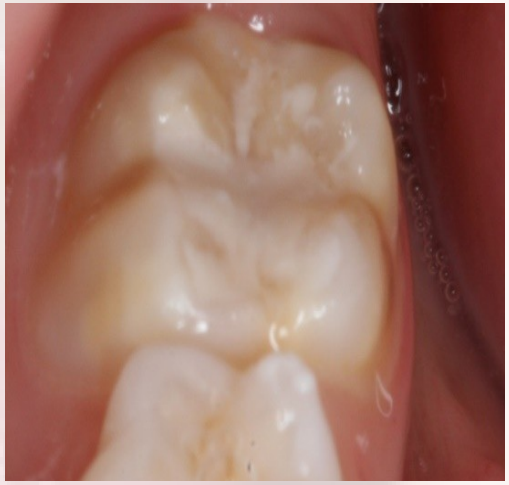
MIH - SYNDROME

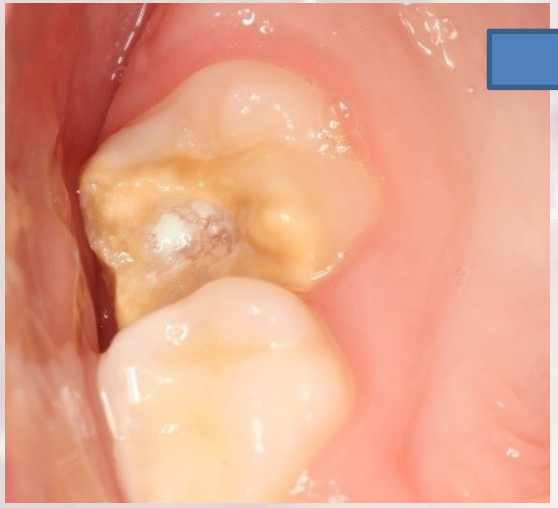
7 years, ♀

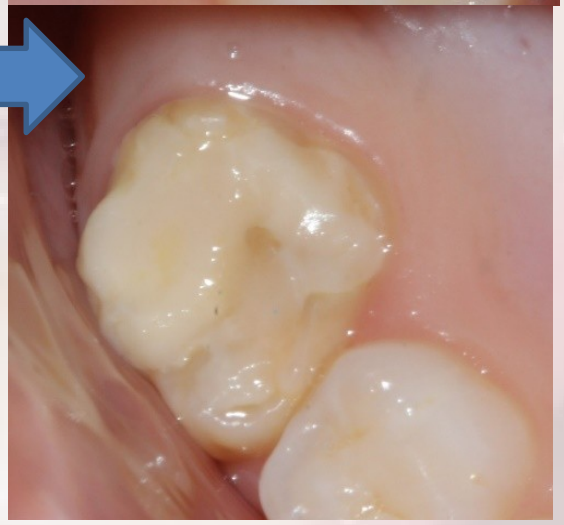


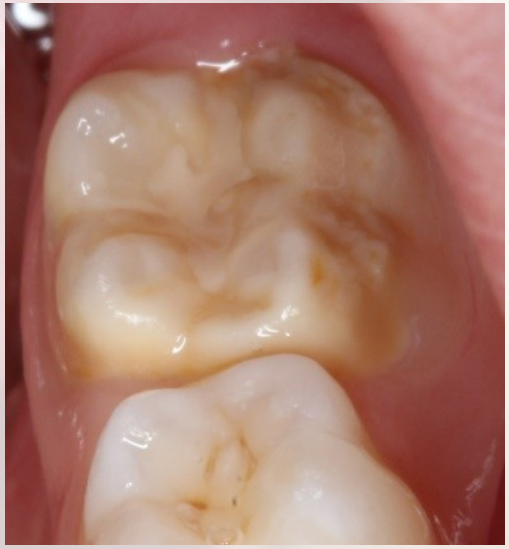






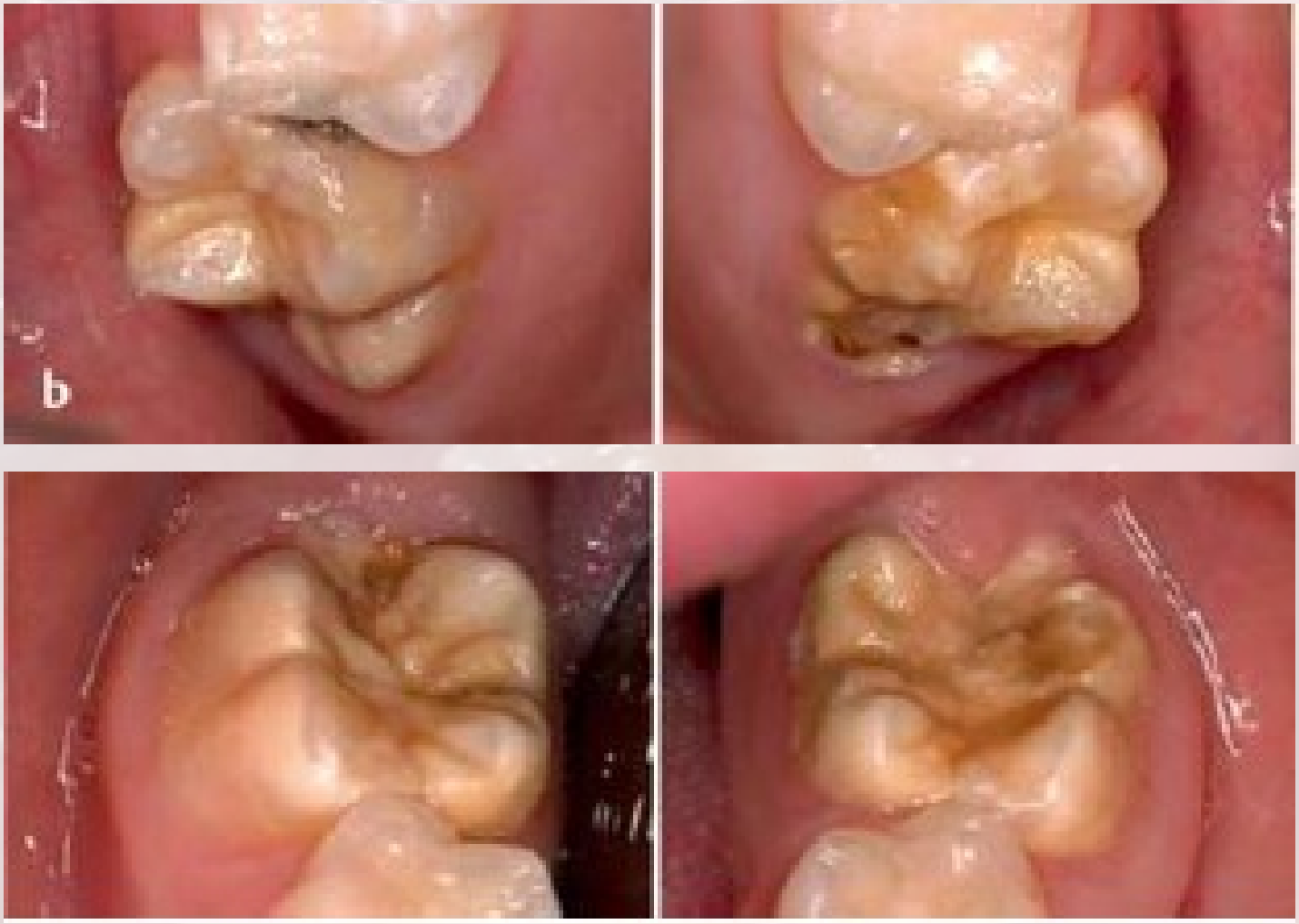






Source: Esetbemutatás – SE-FOK Gyermekfogászati és Fogszabályozási Klinika. E. Mlinkó

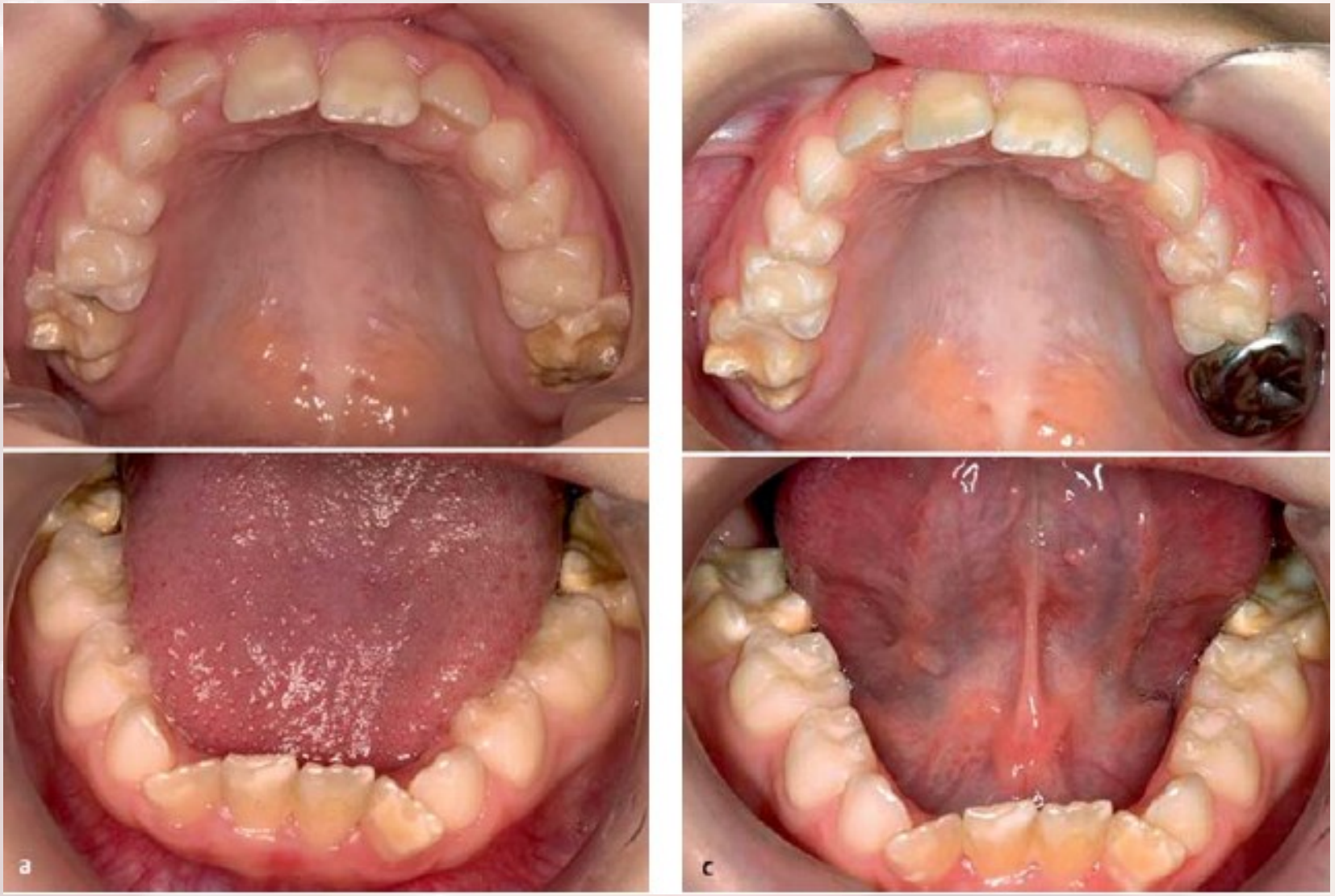
Case 1.



16; 36 és 46: 2- medium -Wetzel and Reckel,
26: 3-severe, before treatment.

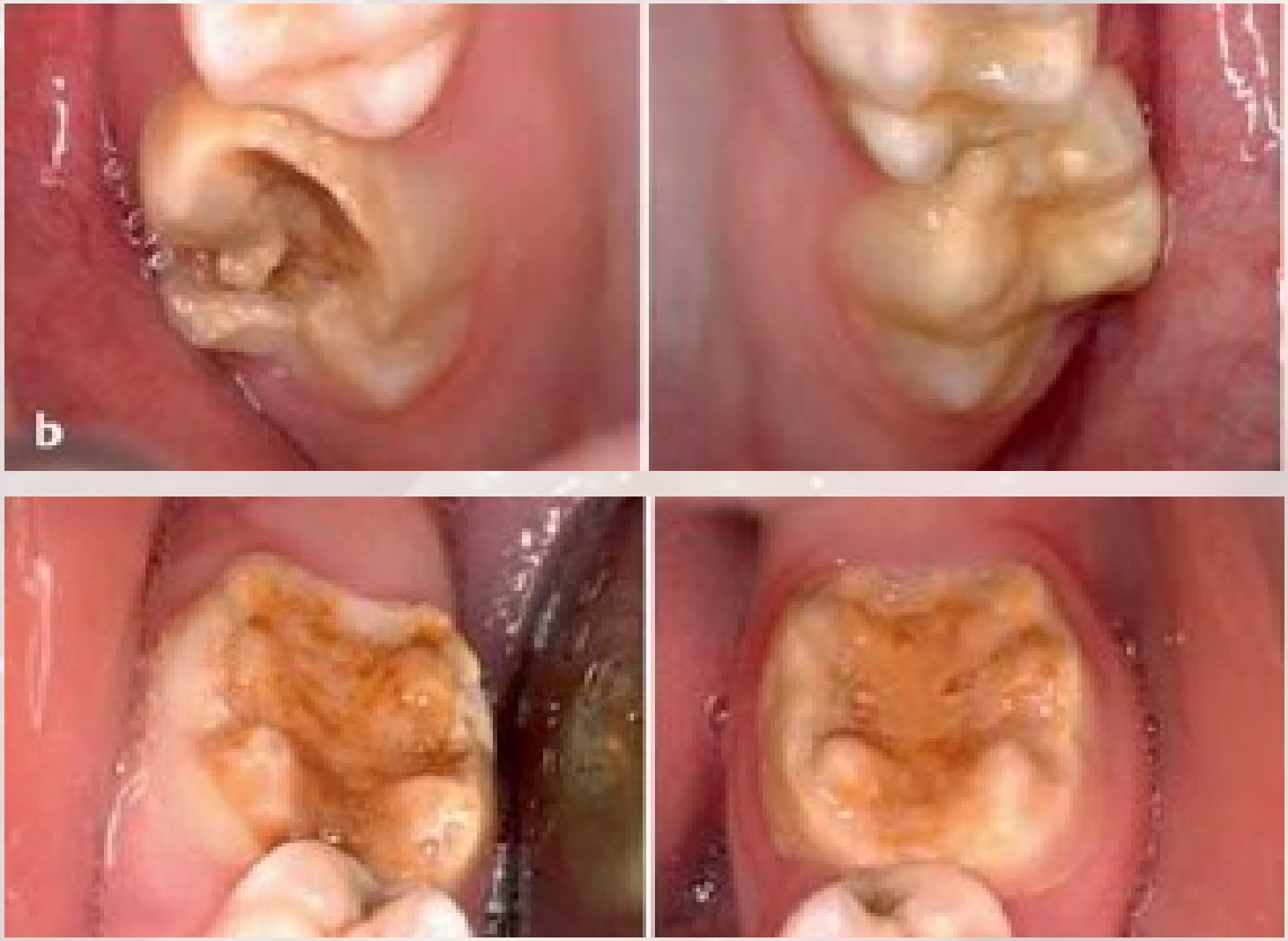
Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

Case1.



Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

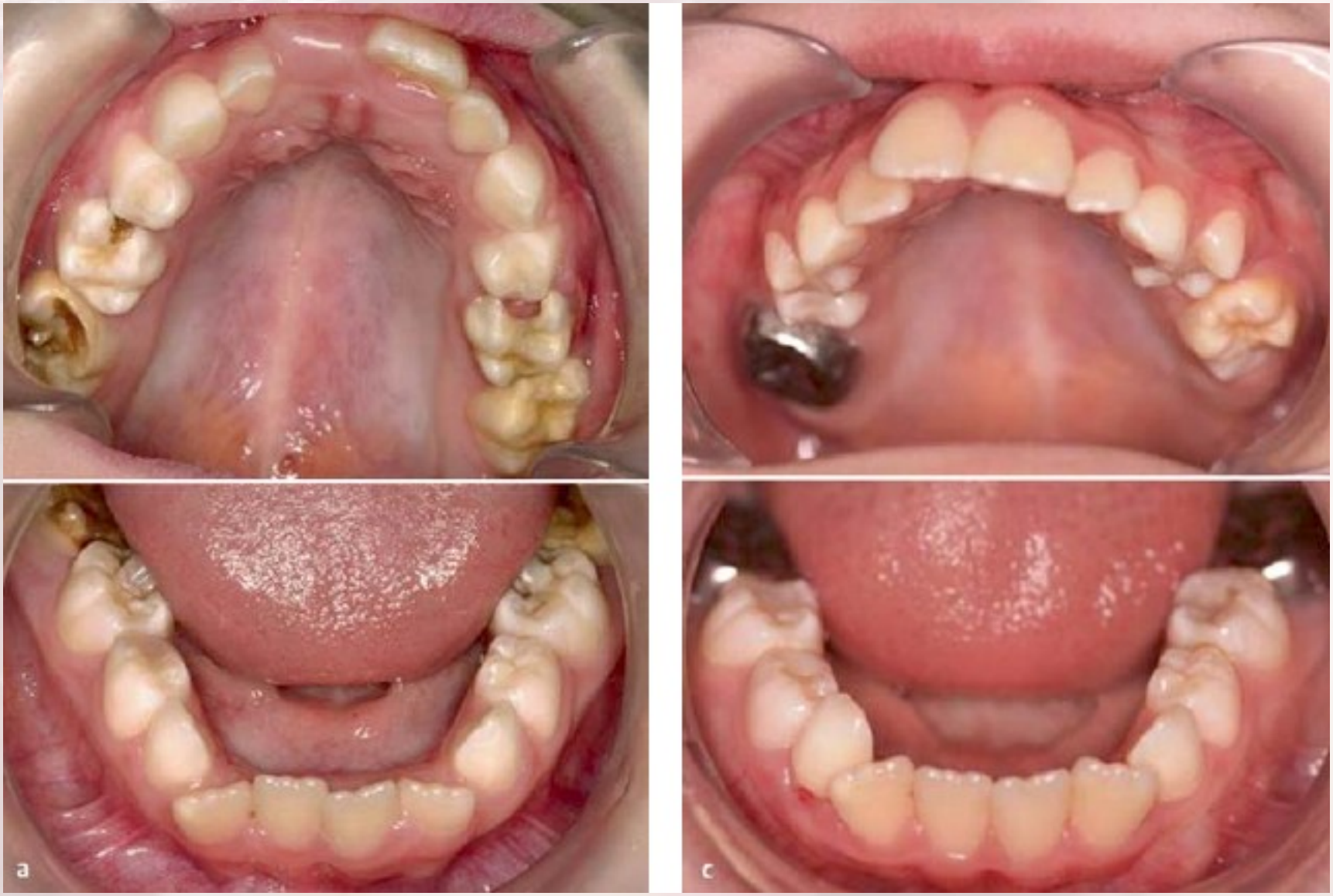
Case 2.



16; 36 és 46: 3-severe, Wetzel andReckel,
26: 1-mild, before treatment

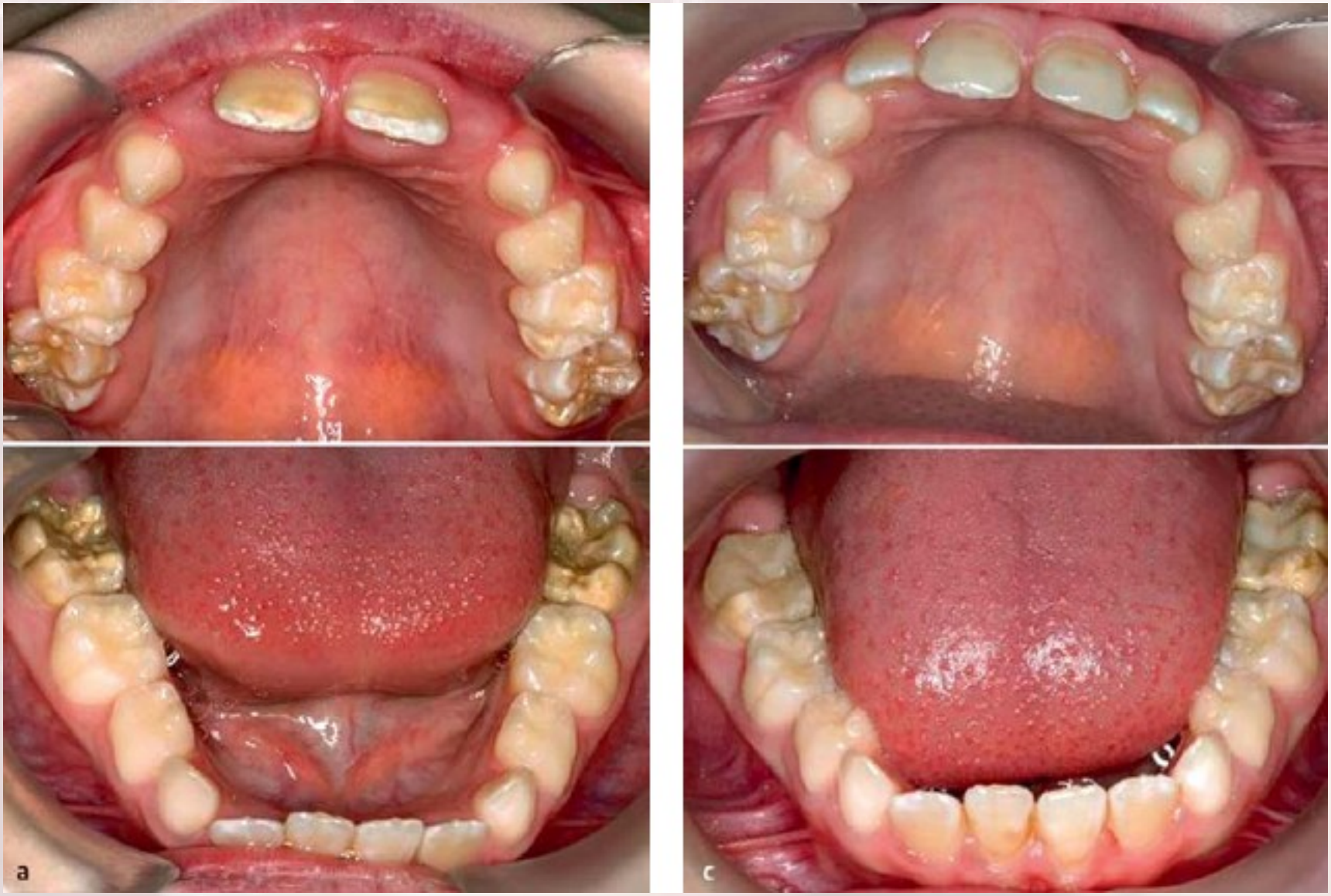
Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

Case 2.



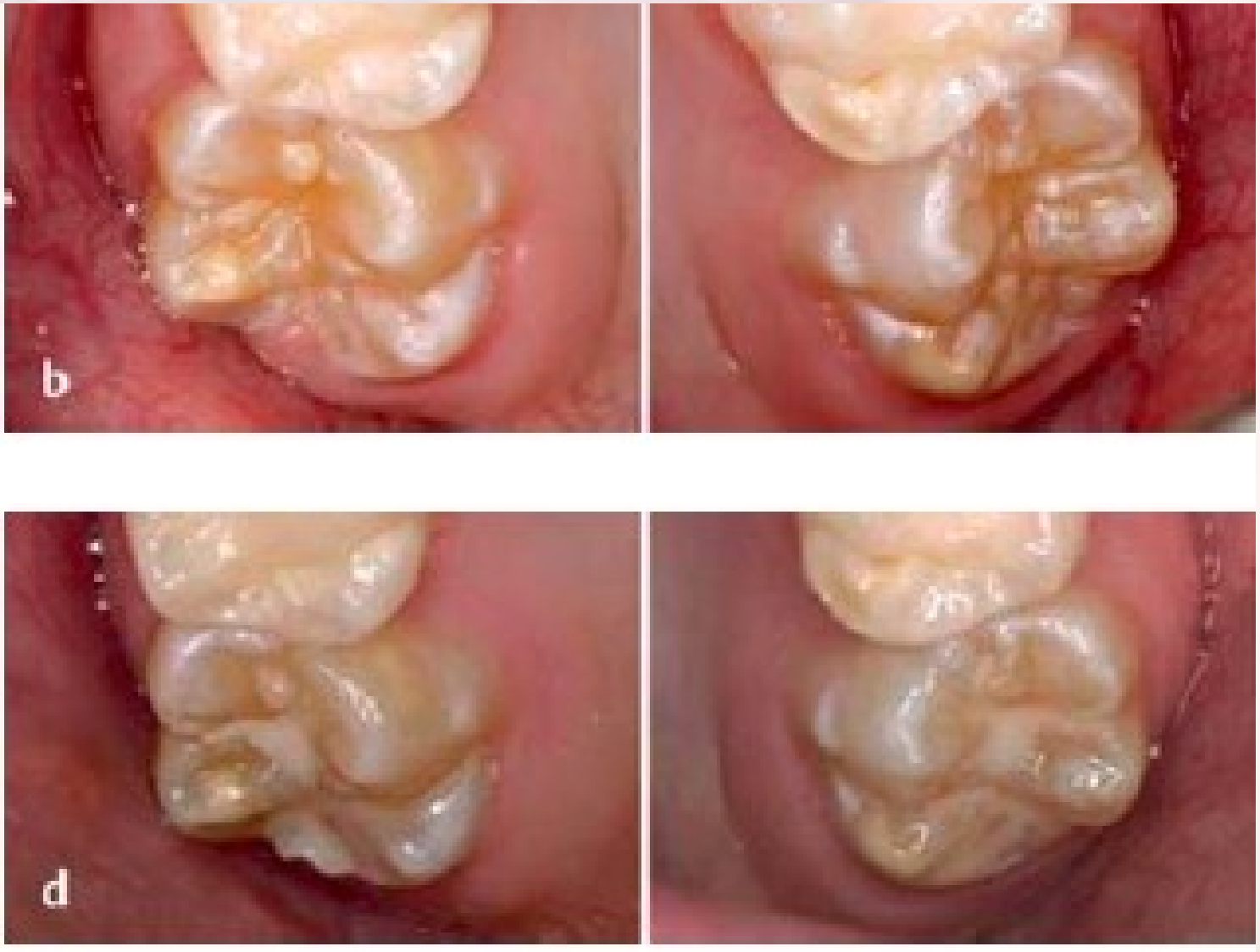
Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

Case 3. – 7½ years old ♀



Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

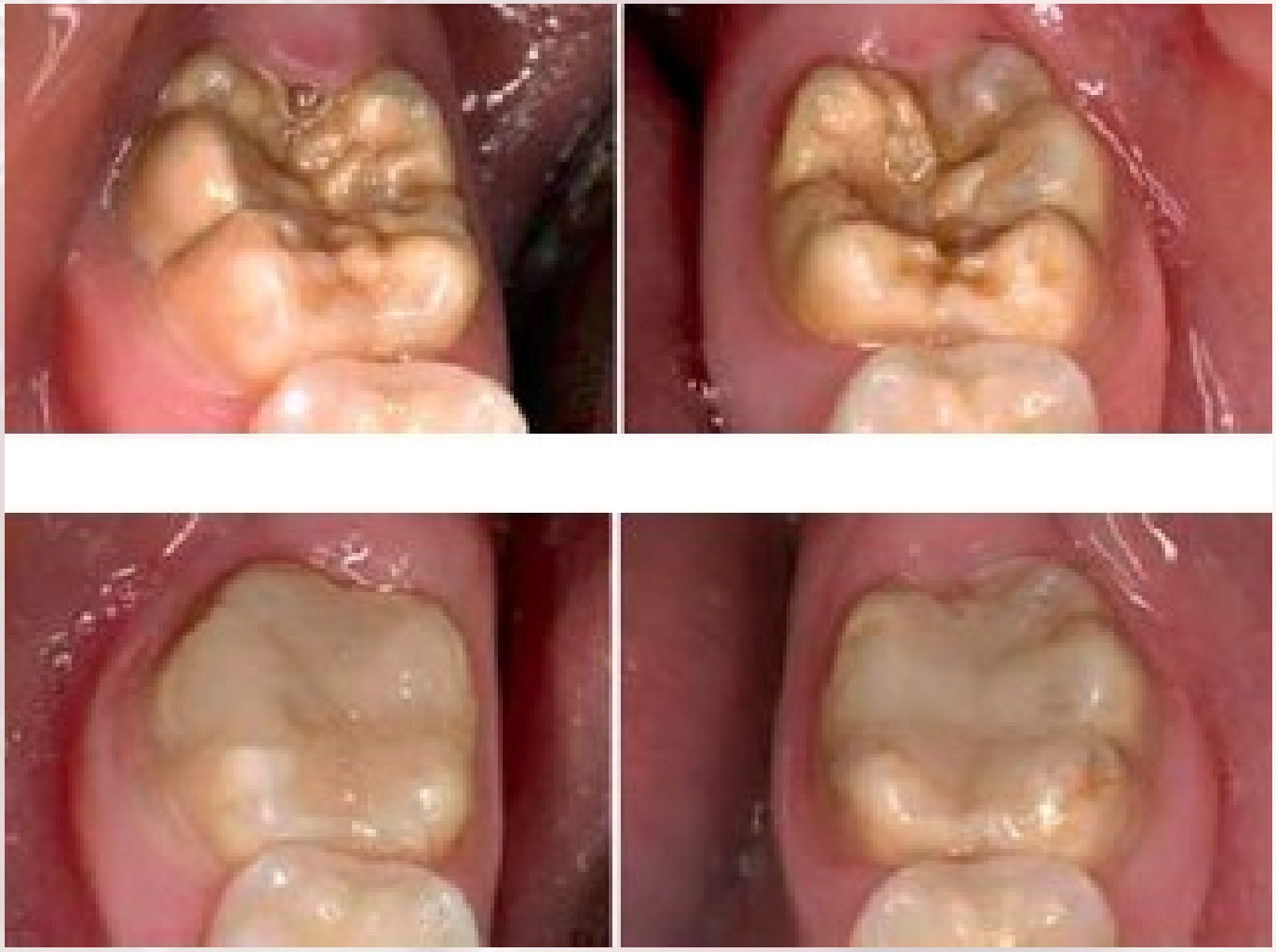
3. Case



16 and 26: 1-mild.

Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

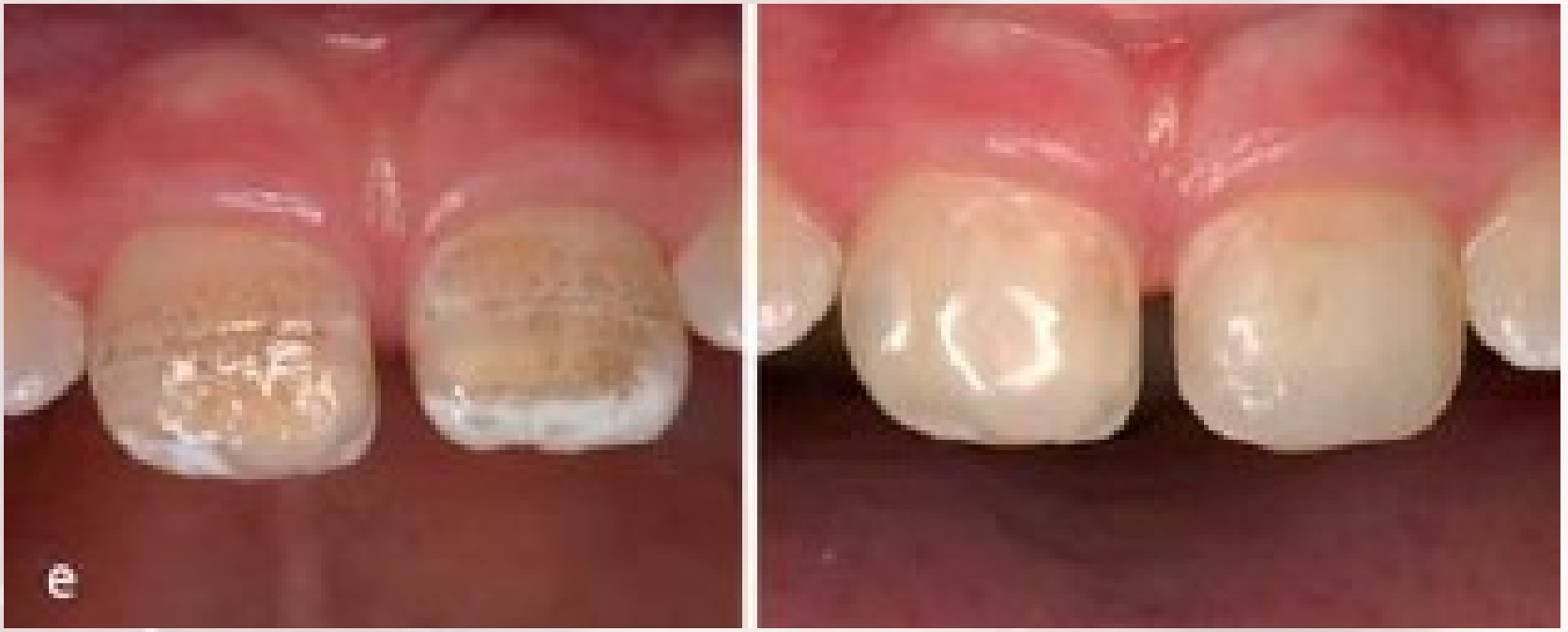
3. Case



36; 46 - 2. medium

Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

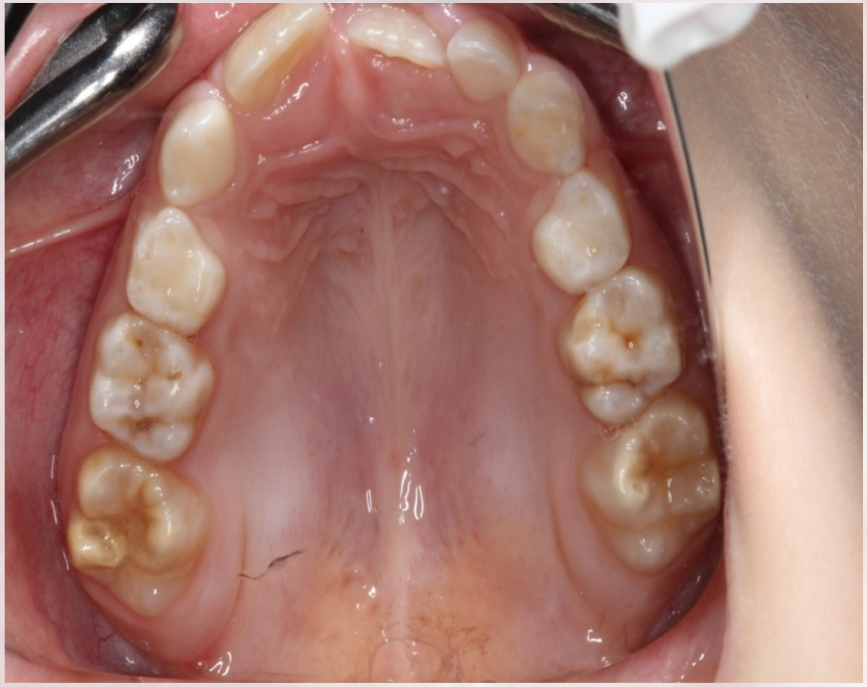
3. Case

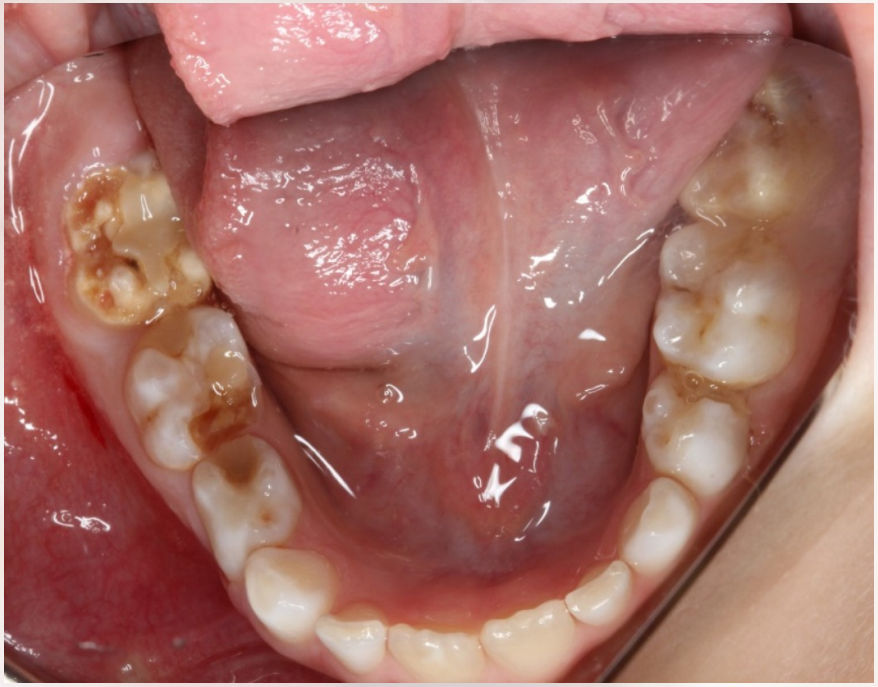


e. Hypomineralisatio: 11; 21.

Source: Knapp V, Nies SM. Molar-Incisor-Hypomineralization. Zahnmedizin up2date 5I2009I491-509.

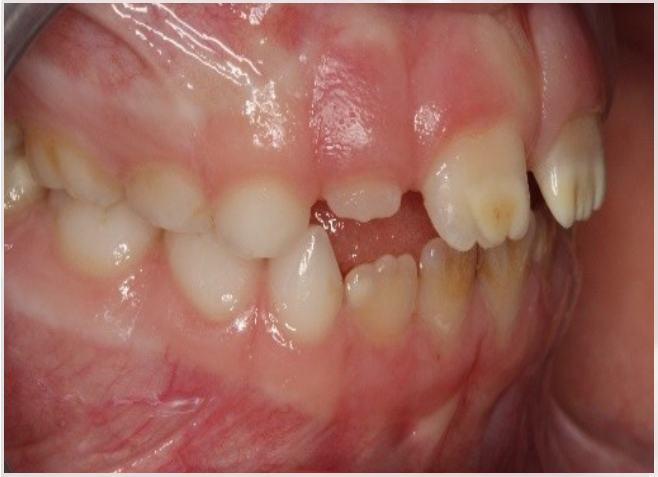
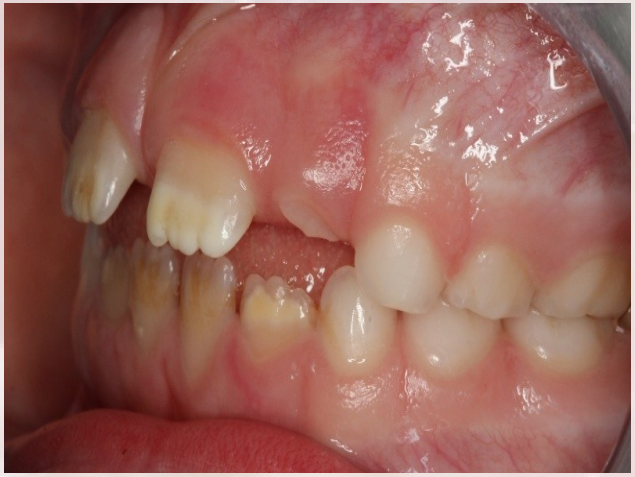
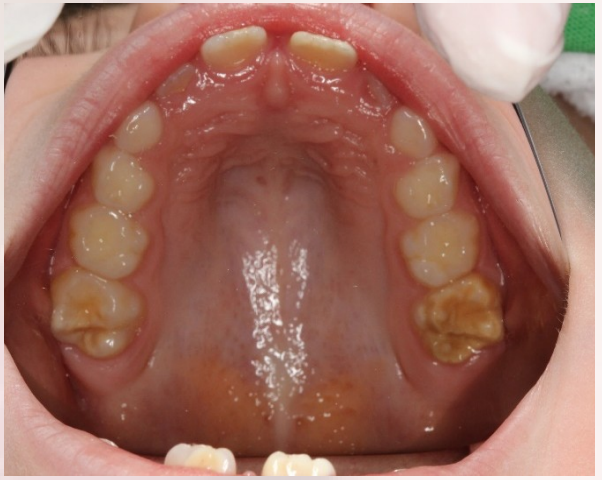
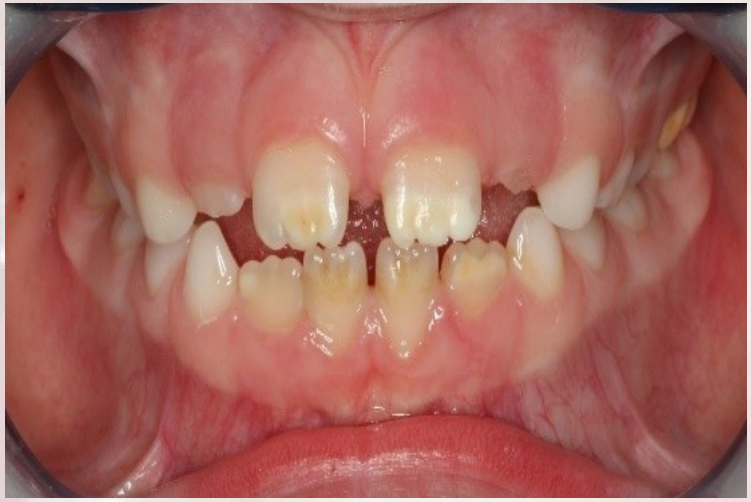
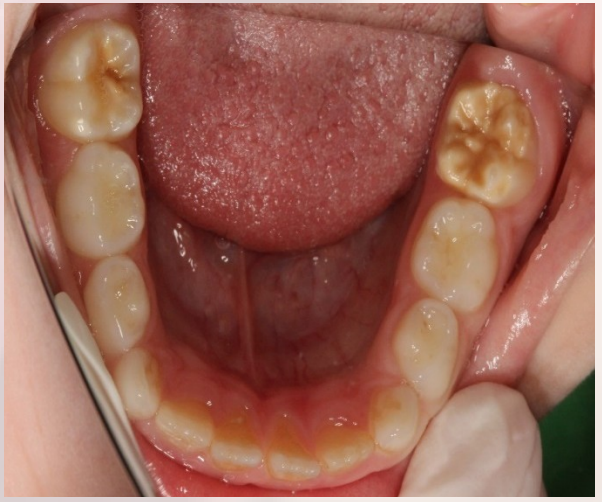
MIH, ♂ - 8 yeras old





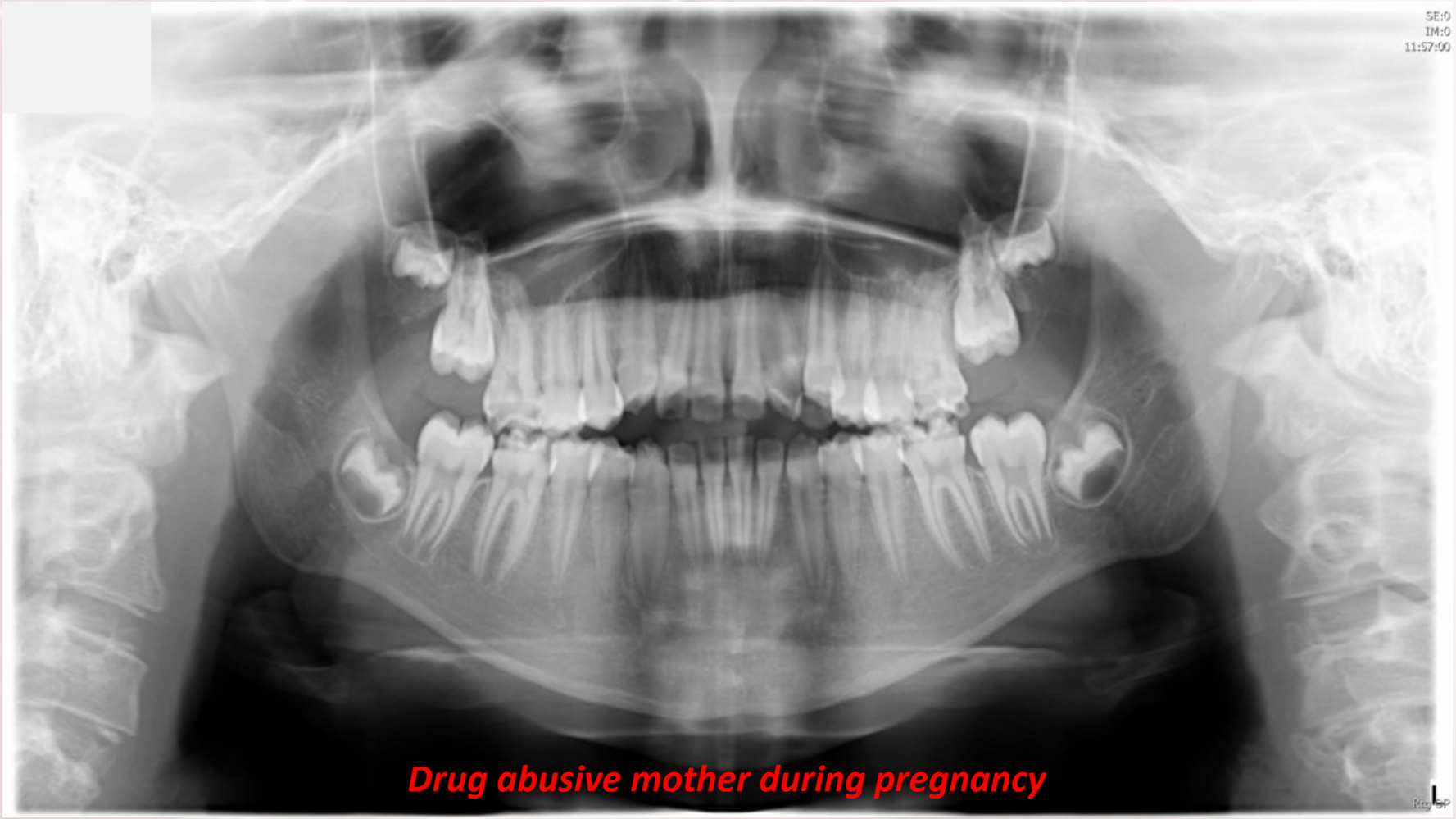
Source: SE FOK Gyermekfogászati és Fogszabályozási Klinika. E. Mlinkó

MIH, ♀, 8 years old

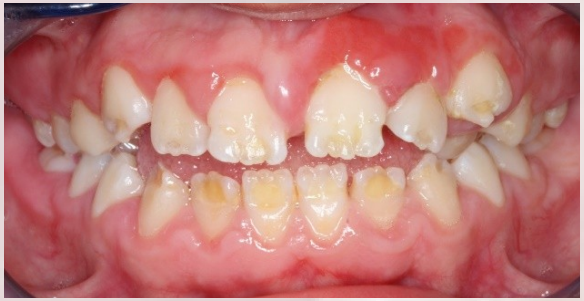






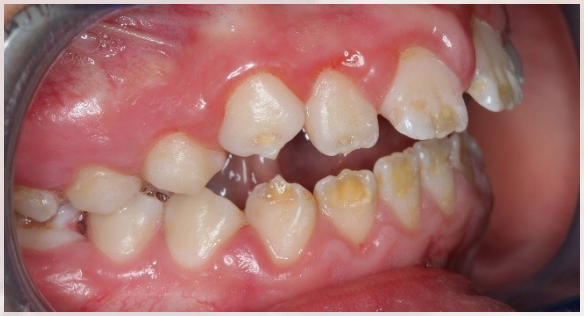
Source: SE FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga. E.Mlinkó

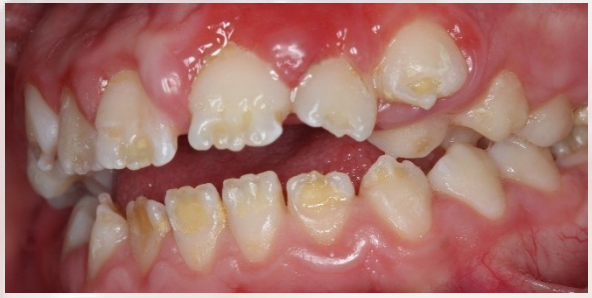
MIH, ♀11 years old

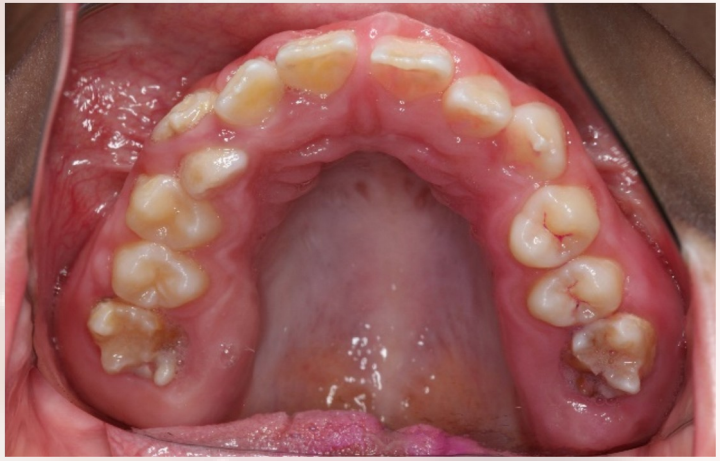


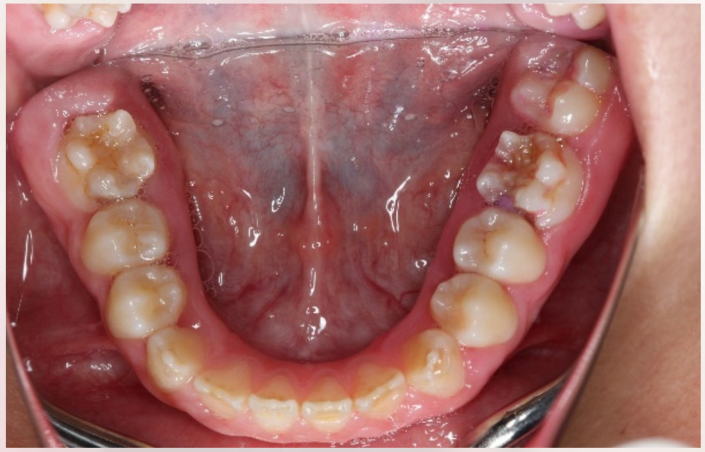
Forrás: SE FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga. D.Zs.Nagy



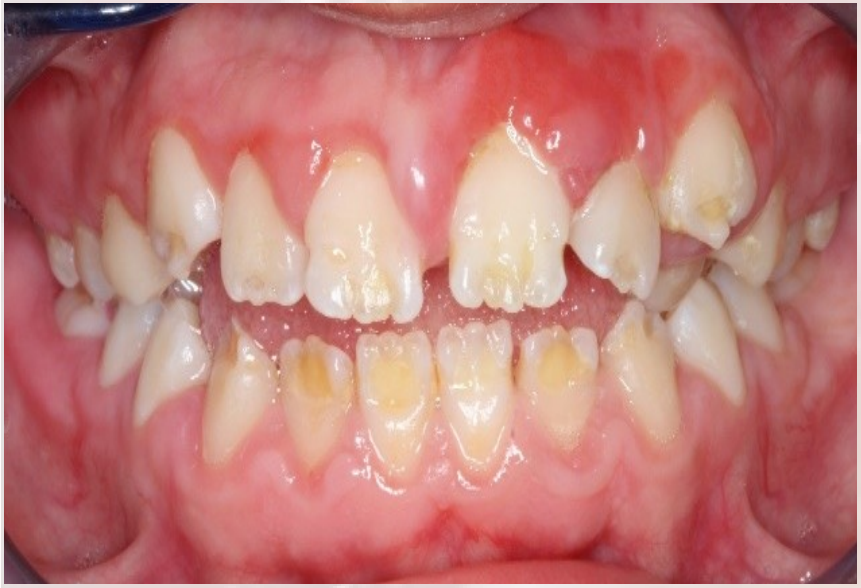




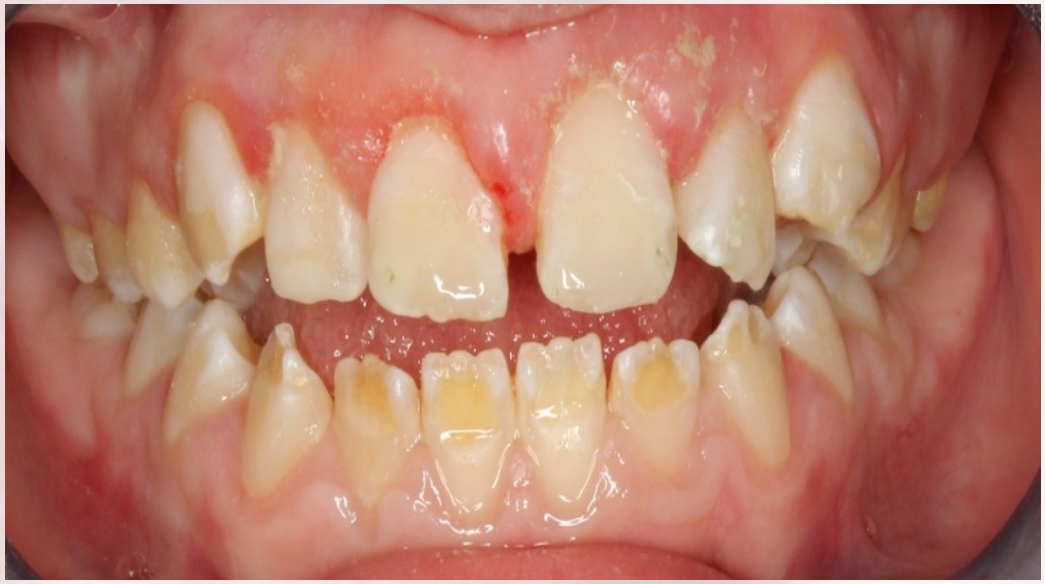




Source: SE-FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga. D.Zs.Nagy



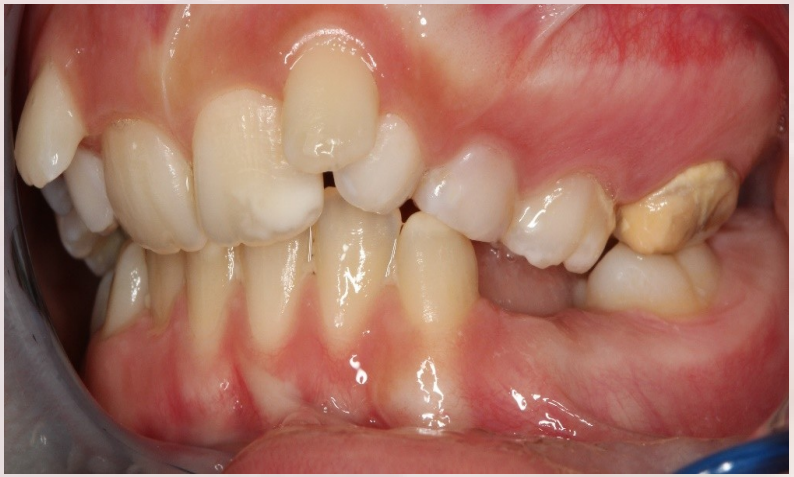
Before treatment

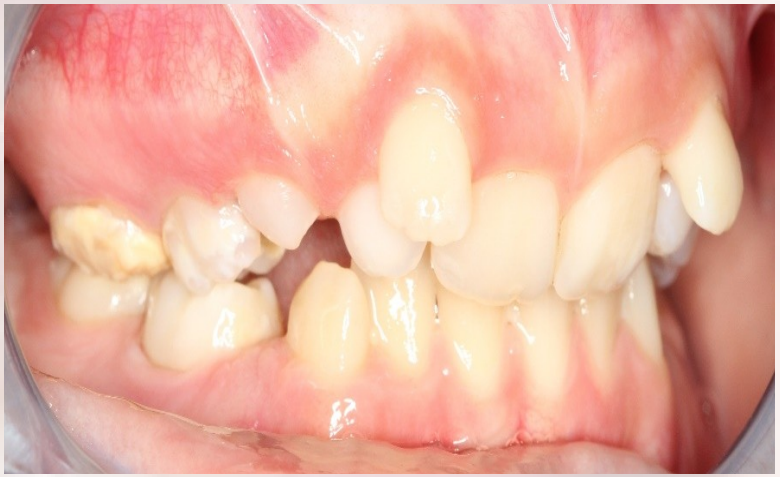


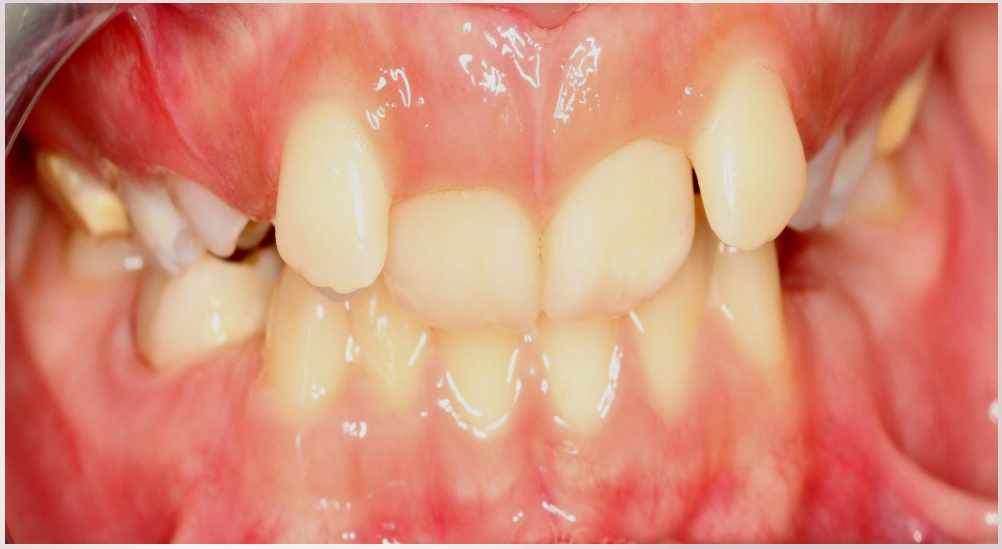
Kompomer fillings

Source: SE-FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga. D.Zs.Nagy

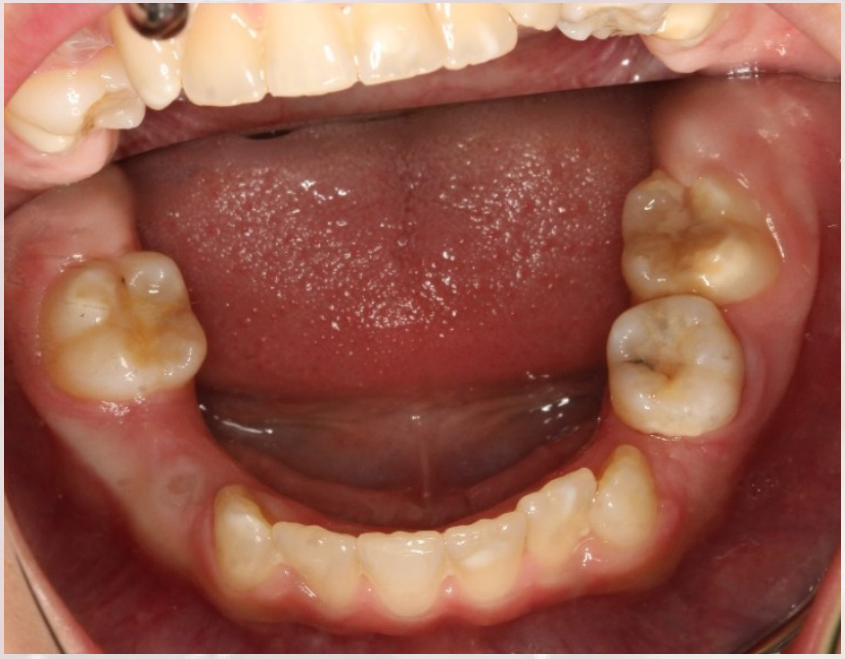
MIH, ♀ - 10 years old

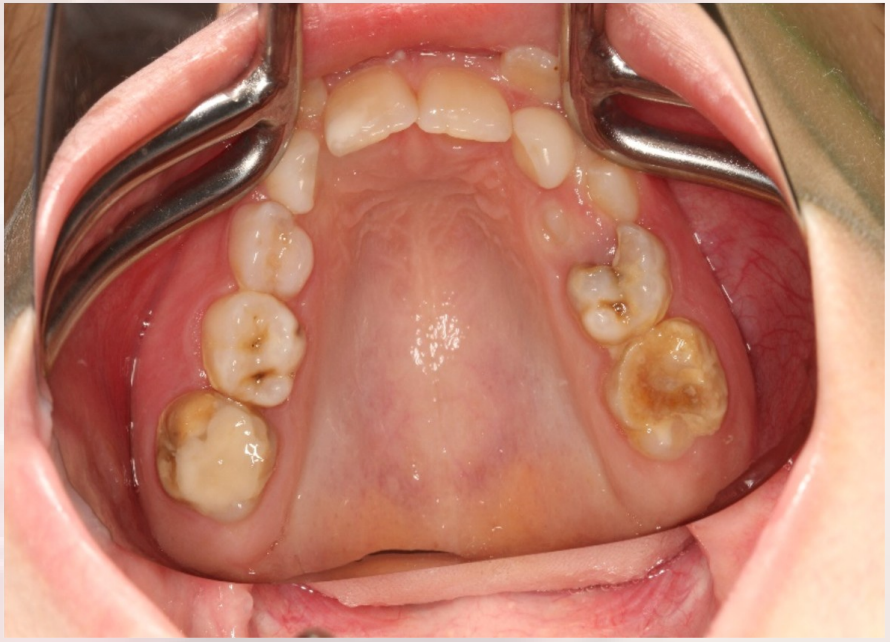






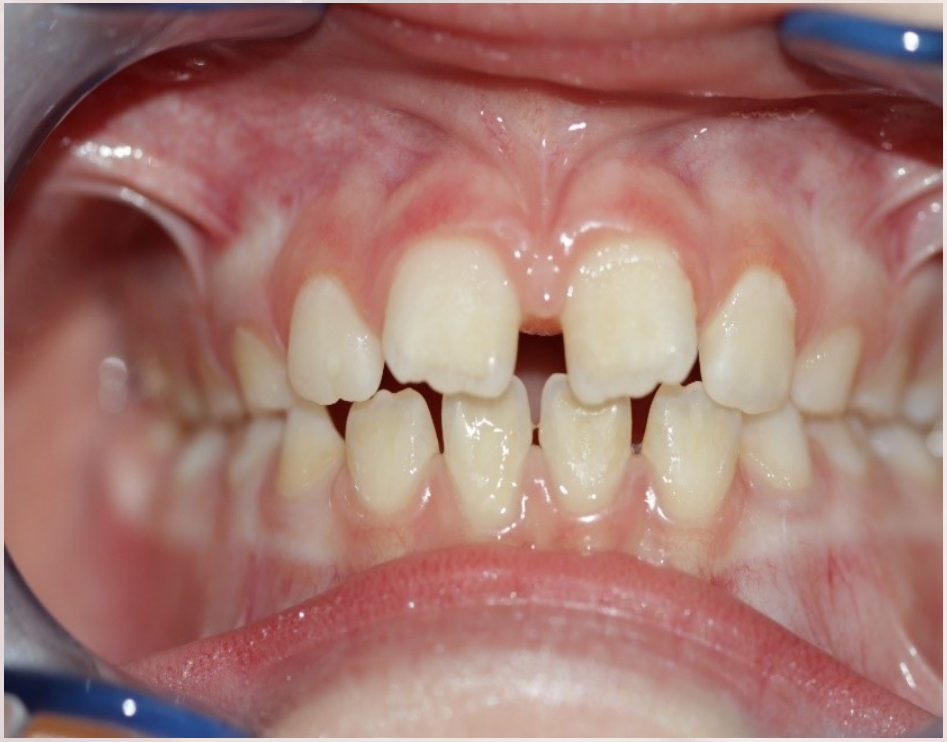
Source: SE-FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga. E. Mlinkó

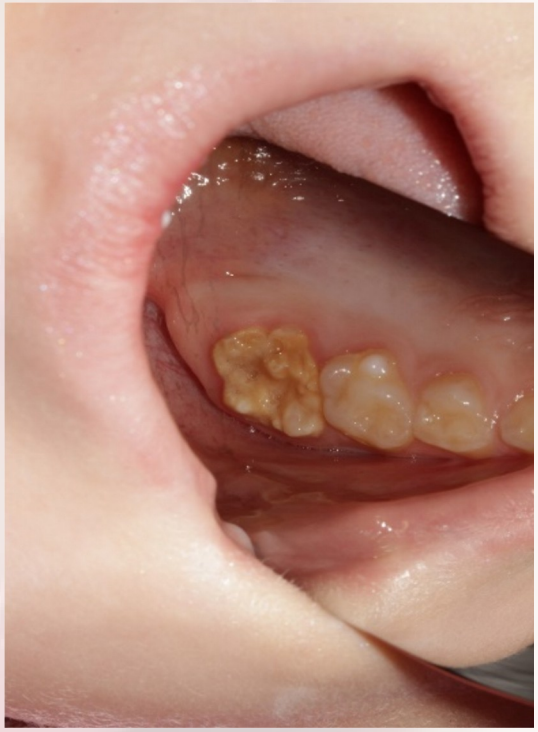




Source: SE-FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga. E. Mlinkó

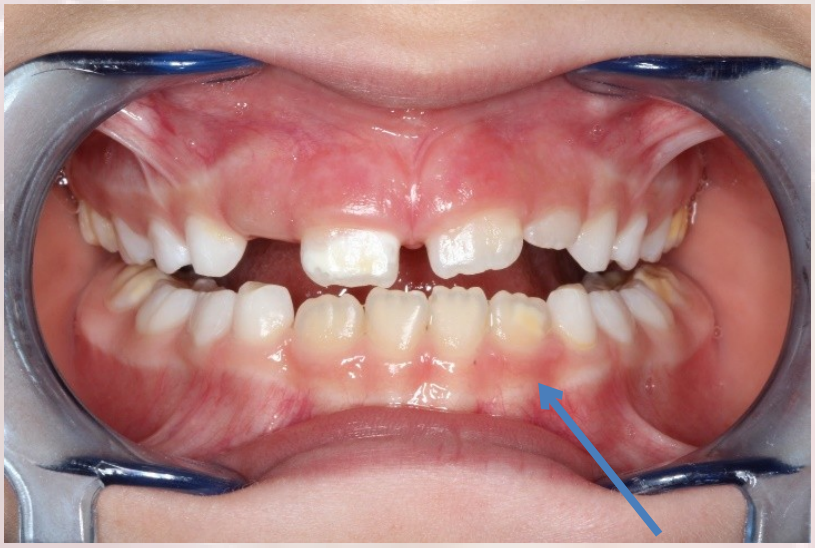
MIH, ♀ - 8 years old

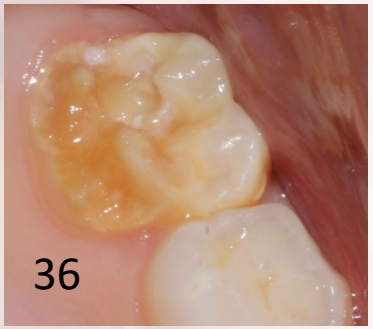


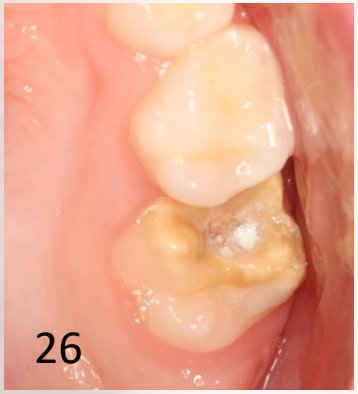


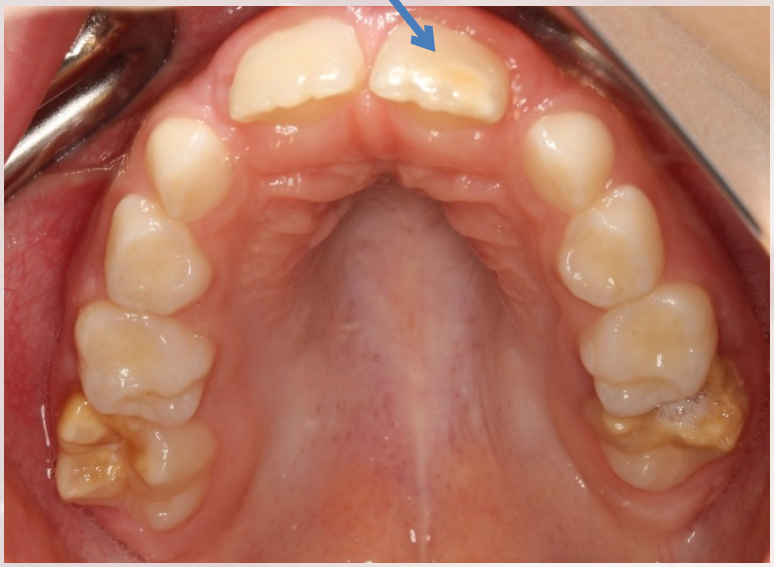
Source: SE-FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga. E. Mlinkó

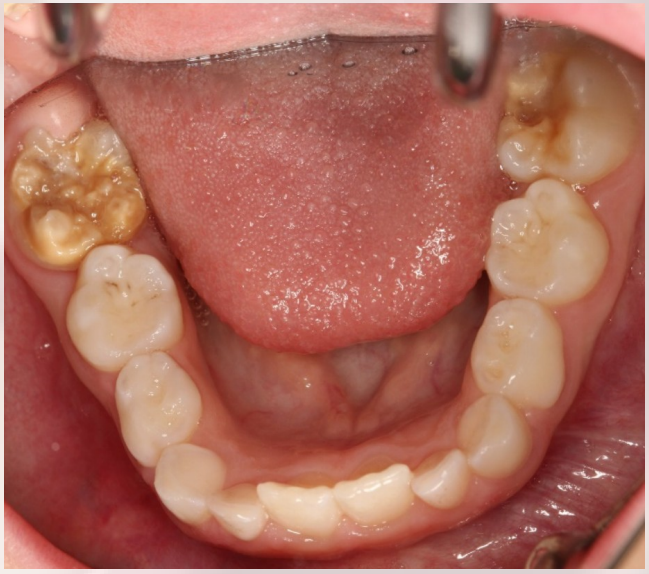
MIH











Source: SE-FOK Gyermekfogászati és Fogszabályozási Klinika képanyaga. E. Mlinkó


Thank you for your kind attention