DENTAL ANOMALIES: HYPOPLASIA AND TOOTH AGENESIS – A CASE REPORT

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ABSTRACT Background & Aims: A research shows that lower right second premolar was among the most frequently missing teeth (3.7%). Only (0.08%) of the research subjects had six or more missing tooth (Oligodontia). The purpose of this study is: Diagnosis of developmental defects of the dental hard tissues and their treatment. Case summary: In order to conduct the study, we received pre-declared informed consent from the parents of a patient who is 10 (ten) years old. The study was conducted in 2019-2020. Caries status was evaluated using both clinical and radiographic data and assessed as D1/D2 – enamel lesion and D3/D4 as dentin lesion. Dental status and treatment. We defined the diagnoses: Caries mediooclussalys d3b on tooth 65; Caries distooclussalys d3b on tooth 64. Caries was treated by fillings of the cavities with a Direct compomer. Deep non-mineralized fissure of tooth 36 with subsequent application of sealant. Orthopantomography also shows Hypodontia premolar dentis teeth (35, 44, 45); Hypodontia dentis (18, 28, 38, 48 D: Oligodontia). Diagnosis: 10-year-old boy with hypoplasia of the upper permanent incisors and Oligodontia. The patient was treated with non-invasive and invasive methods of treatment. The study was conducted at the University Medical and Dental Center. Conclusion: 1. Developmental anomalies associated with tooth agenesis include delayed tooth formation, prolonged primary tooth exfoliation, retained primary teeth, interdental spacing. 2. Enamel hypoplasia increases the risk of developing dental caries. 3. Early diagnosis allows optimal patient management and treatment planning and can reduce complications of the planned treatment.

KEYWORDS Tooth anomalies, Panoramic radiograph, Tooth agenesis, Oligodontia

Introduction

According to Laganà G. et al. 2017, the lower right second premolar was the most frequent missing teeth (3.7%) had only one tooth agenesis, and (0.08%) had six or more missing tooth (Oligodontia). The overall prevalence of dental anomalies was 20.9%. Approximately, 17.9% showed only one anomaly, 2.7% two anomalies, while only 0.3% had more than two anomalies. The most frequent anomalies were the displacement of maxillary canine (7.5%), hypodontia (7.1%) and tooth transposition (1.4%). The research done by the authors revealed significant associations among different dental anomalies and provide further evidences to support common etiological factors. [1].

Various dental anomalies of the dentition are frequently observed together in clinical practice [2]. If such associations of hereditary origin occur, they may be examined, as early diagnosis of one tooth developmental disturbance may reveal a potential risk of future position or eruption disturbances of other teeth [2].

According to Giuseppina Laganà et al. 2018, agenesis diagnosed in the two arches registered for 13.8% (n =45). Maxillary hypodontia was observed in 151 subjects and mandibular hypodontia was detected in 129 subjects. The prevalence of displacement of maxillary canines in the maxillary hypodontia group (14.8%) was higher than that in the mandibular hypodontia group (9.6%), with a chisquare value of 1.679 (P = 0.192) [3].

Oligodontia is a rare pathology and there are only limited data on its prevalence.
Researchers conducted a study with Danish students and found that oligodontia occurs in 0.16% of children (95% confidence interval, 0.07% to 0.30%). Two out of every three congenitally missing teeth were upper or lower second premolar or upper lateral incisors, and the condition was more common in girls than in boys ($P = 0.05$) [4].

From our known classification Dysplasiae dentales acquisitae (post rachitidem) refer to acquired dental dysplasias. They are the result of the influence of factors that have their manifestation after the birth of a child or during his first three years. The germs of almost all permanent teeth are laid from the stage of birth to the ninth month. Then only the germs of the premolars and the second molar are laid. Aplasia (lack of a tooth) of these teeth is possible during this important period. In most cases, aplasia is observed for the first premolar, which is furthest from the molar pattern. All permanent teeth except the first permanent molar build their organic matrix and mineralize it after birth. This process begins in the fourth month and lasts until the child’s 8th year. The beginning of the period is risky for hypoplasia and the whole period for occurrence of hypomineralization [5].

Risk factors for acquired dental dysplasias are metabolic disorders in infancy and early childhood. They occur as a result of malnutrition, disease and insufficient ultraviolet radiation. Such are nutritional deficiencies, hypovitaminosis - A, C, D, Ca and P deficiency, as a cause of rickets and others. Acquired dental dysplasias mainly damage the permanent dentition and a symmetrical group of teeth with close developmental dates. The most commonly affected groups of teeth are: first permanent molar, incisors and canine. These teeth develop approximately in the near future and the risk factors that have influenced them can also cause hypoplasia and hypomineralization. It is important to consider that the duration of action of pathological factors is also important for the severity of dental dysplasia. The longer they last, the more extensive the areas of defective construction of rigid dental structures [5]. Teeth with eruption have a corresponding defect. Patients sometimes report a feeling of sensitivity in the teeth. One of the causes of these dysplasias is vitamin D deficiency, leading to rickets. Vitamin D is important for the structural organization and mineralization of bones and teeth and for the formation of the organic matrix by participating in the transfer of proteins. It also regulates the absorption of Ca 2+ from the ileum. In cases where its amount is reduced, the formation of the organic matrix is disturbed and the clinical picture is of hypoplasia of the tooth enamel. Hypocalcemia is the reason for the simultaneous presence of skeletal deformities and dental defects - hypoplasia and hypomineralization of permanent teeth. Children having suffered rickets have the following clinical status: hypoplastic fields, furrows, dots, or mixed altered areas located along the incisal third of the front teeth and the occlusal surface of the first molars. Defects are observed on symmetrical permanent teeth [6].

More severe cases with changes in Ca / P (calcium-phosphorus) metabolism may also affect the mineralization of primary teeth. The differential diagnosis is determined by the anamnesis and changes in the skeletal system after rickets. Nowadays, hypoplasia of permanent teeth after suffering from rickets is rarely observed. There is prophylaxis against rickets with vitamin D drops prescribed by pediatricians in the first year after child birth with the exact period of intake and exact dosage [6]. According to Łanowy Patrycja et al., in 2019, the relationship between the level of vitamin D, which is involved in the proper mineralization of bones and teeth, and the presence of carious lesions in the dentition has been studied for years. In their publication, the researchers described that in Poland, the addition of vitamin D is recommended for children and adults from September to May and / or even throughout the year if the synthesis of the vitamin is insufficient. The analysis they perform is between caries disease and the level of vitamin D in the blood serum. In conclusion, Łanowy Patrycja et al. have proven that available research from the last seven years suggests a strong relationship between serum vitamin D levels and the prevalence of dental caries in children [7]. Sources of vitamin D are liver, egg yolk, fish oil. The function of this vitamin is its direct participation in the construction of enamel and dentin. Together with parathyroid hormone, it regulates the concentration of Ca and P in plasma and extracellular fluids and maintains the level of alkaline phosphatase in tissues. The daily requirement for children, pregnant and lactating women is 400 to 800 IU. In the treatment of rickets, intake is 1500-2000 IU. Vitamin D deficiency causes rickets in hypovitaminosis, which leads to: enamel hypoplasia, disturbed mineralization, dilation of the predentin area, wide interglobular spaces, irregular dentin and disturbed tooth eruption [8].

Case report

The purpose of this study is: Diagnosis of developmental defects of the dental hard tissues and their treatment. The study was conducted in 2019-2020.

Place of study: To conduct the study, we received pre-declared informed consent from the parents, respectively for the patient D. I. L, who is a child and started from his 9 (ninth) year, in 2019. The patient has been diagnosed, and the prevention and treatment of the child continue in his tenth year until now, in 2020.

Medical history. The anamnesis was taken according to data of the patient’s mother.

Passport part: History of the disease. The reason for the clinical visit of the patient with his parents is a preventive examination. The last visit of the patient to the clinic was a month ago for the purpose of extraction of temporary tooth 54 and due to the eruption of the permanent tooth 14, related to the dynamics of dental development and the time for eruption of the permanent first premolars. The scheduled visits of the patient for prophylactic examinations were every 4 months due to the assessment of a high risk of developing dental caries.

Social history and behavior. The patient has so far visited a dentist and is afraid of that. The behavior of the child with a score on the Frankl scale can be defined as slightly negative. The child has been taking sweet food and drinks four times a day.

Family and medical history. The mother’s pregnancy was normal by the end of the 9th month. The child was born at term in natural nonsurgical care in a hospital setting. The patient is a naturally fed child and in the first months after birth he was of normal weight and development. At one year and two months, the child began walking independently. The mother had no complications or injuries during the pregnancy. No medication was taken by the mother during pregnancy. The mother did not report diagnosed systemic diseases or food allergies. The patient has had established allergies to the following antibiotics: Sumetrolin, Augmetin. According to data of the patient’s mother, the child has preferences which do not include going out for sun walks during the day. The child suffered from a mild form of rickets. The mother did not report of the child having taken vitamin D in the first year of his childhood.
Oral hygiene habits The patient brushes his teeth on his own twice a day using an Elgydium Junior tooth program and an electric toothbrush and brush head is changed every two months as a period. The patient uses Elgydium Junior Ice Age, a 50 ml pack children’s toothpaste. It is a gel toothpaste designed for the prevention of temporary and first permanent teeth in children. ELGYDIUM Junior is specially designed to protect 7 to 12 year old permanent teeth enamel thanks to the adjusted fluoride concentration (1000 ppm fluoride ion). The low-abrasion formula protects tooth enamel and protects against caries formation. It is suitable for everyday use and has a pleasant tutti frutti taste. The composition of the paste used is: Hydrogenated starch hydrolysate, water (aqua), hydrated silica, anise alcohol, benzyl alcohol, cellulose gum, ci 42051, flavor (Aroma), nicotinyl alcohol hf, Peg-12, Peg-12 allyl ether, Peg -12 dimethicone, sodium benzoate, sodium lauroyl sarcosinate, sodium propionate, sodium saccharin [ELGYDIUM, 9]. The patient washes his mouth with daily use water for oral hygiene and additional oral hygiene products [Elgydium Junior mouthwash, 10]. Until this stage, the child has not undergone endogenous fluoride prophylaxis and currently does not take fluoride medications. The patient has no available bad habits. Temporary teeth and permanent teeth so far erupt on time. Family history. Parents visit their dentist regularly for treatment and for preventive examinations. Parents do not report allergies and chronic systemic diseases.

Clinical status The child has been attending our clinical practice for 1 year, when he was 9 years old. The patient at today’s treatment plan is 10 years old and has normal mental and physical development. The visible age corresponds to the actual age of the child.

Extraoral status. The patient has no facial asymmetry and visible scars in the face and neck. No rashes or swelling of the soft tissues were found. The lips are of normal pale pink color. The patient has not had orthodontic treatment so far. The child has not been in hospital and is not taking medication at the time of the test. The prophylactic examination of the patient showed reduced oral hygiene with the presence of mature plaque biofilm on the vestibular and occlusal surfaces of the teeth and the area of the teeth necks. Clinical oral prophylaxis was performed with a recommendation for the use of personal hygiene products at home. Detailed clinical status was taken and the gingiva and oral mucosa were assessed.

Dental status and performed dental treatment: Caries status was evaluated using both clinical and radiographic data and assessed as D1/D2 as enamel lesion and D3/D4 as dentin lesion. Dental status and treatment: Caries medioocclusalys d3b on tooth 65; Caries distoocclusalys d3b on tooth 64. Caries was treated by fillings the cavities with a comperomer Direct.

A pit is a small depression on the surface of the tooth, whereas fissures are the grooves that naturally occur on all biting surfaces of teeth. Deep fissure tooth 36 with subsequent application of sealant (Sealants render the pits and fissures easier to clean during brushing and chewing, as food particles do not get trapped as easily when you have deep fissures). The patient was treated with non-invasive and invasive (surgical) treatment methods.

The patient underwent remineralizing therapy in a clinical setting of all available teeth and surfaces with Tooth Mousse cream (GC Tooth Mousse remineralizing protective cream for sensitive teeth without fluoride) with 10 minutes application after pre-polishing, washing and drying of tooth surfaces. The composition of the cream is: milk protein RECALDENT™ (CPP-ACP) – provides the teeth with calcium and phosphate, strengthens tooth enamel, without fluorides, without sugar.

We prescribed the application to continue the application at home every night for 7 months, after oral hygiene and dried tooth surfaces. Remineralizing therapy was continued with prescribed dental agents [GC Tooth Mousse Mint, 11-15].

**Diagnosis**

Hypoplasia of permanent incisors and canines and Oligodontia teeth, tooth agenesis: 35, 44, 45, 18, 28, 38, 48, Table 1, Figures 1 and 2. Statistical analysis. All descriptive and comparative statistical analyses were performed using the SPSS software package (version 20.0, SPSS).

**Figure 1** Chronological and symmetrical hypoplasia of the permanent incisors and first permanent molars in a breast-fed 10-year-old boy who was not supplemented with vitamin D in the 1st year after birth.

**Discussion**

Tooth agenesis, the congenital absence of at least one tooth, is the most frequent dental anomaly [Laganà G. et al., 2011, 18]. The results obtained from this study demonstrate that in subjects with tooth agenesis teeth most often missing are upper lateral incisor, followed by lower second premolar; the female to male ratio is 5:3; upper and lower incisors are inclined lingually [16-18].

Gordon Nikiforuk et al. in 1981 described the etiology of enamel hypoplasia. In this study of children, researchers reported that there were scientific reports of enamel hypoplasia...
and other pediatric disorders in which hypocalcemia was a major symptom (e.g., vitamin D deficiency, prematurity, and neonatal tetany). The presence of enamel hypoplasia in a hypoparathyroid or rickets patient, when related to the chronology of enamel mineralization, helps to establish the time of onset of hypocalcemia. The results of these authors hypothesized that low serum calcium concentrations during enamel formation are a specific determinant of enamel hypoplasia. This hypothesis may be relevant for the etiology of linear enamel hypoplasia, lesions of temporary teeth in children from many countries, which predispose teeth to the development of caries [19].

Purvis et al. in 1973 concluded that enamel hypoplasia in childhood was a manifestation of vitamin D deficiency during pregnancy and was most likely the result of secondary hyperparathyroidism, which afflicted the mother [20].

If vitamin D levels are inaccurate for the mother (assessed using 25-hydroxyvitamin D [25OHD]) during pregnancy, they can affect the mineralization of teeth, predisposing to enamel hypoplasia and the development of dental caries in early childhood or developing ECC (Early childhood caries). The findings of authors such as Schroth RJ and co-authors in 2014 are that prenatal levels of 25OHD in the mother may affect the dynamics of development of temporary dentition teeth and the presence of ECC (Early childhood caries) [21].

In a long-term study, Nikiforuk G and coauthors in 1979 selected children patients with with three well-defined disorders of calcium and phosphate homeostasis, such as: hereditary vitamin D rickets, rickets associated with the X chromosome, hypophosphatemia, and hypoparathyroidism. Scientists report that they observe distinctive distributions of enamel and interglobular dentin hypoplasia, which provide conditions for the pathogenesis of these dental lesions. Each of the 25 patients with X-linked hypophosphatemia had extensive interglobular dentin in the temporary and permanent teeth, but had no enamel hypoplasia. These patients were normocalcaemic but very hypophosphatemic. Each in ten patients with vitamin D-related rickets had severe enamel hypoplasia in the permanent dentition. Moderate interglobular dentin was observed in each of the 7 patients whose teeth were examined histologically. These patients were hypocalcemic and also hypophosphatemic due to secondary hyperparathyroidism. Fifteen (out of 21) children with hypoparathyroidism had severe enamel hypoplasia, but none had interglobular dentin. The patients were hypocalcemic and hypophosphatemic. The hypothesis formulated by these authors is that enamel hypoplasia, in disorders of calcium and phosphate homeostasis, was caused by hypocalcemia, and interglobular dentin is caused by hypophosphatemia [22]. Finally, it is necessary to determine the consequences for dental health and to ensure the prevention of caries in early childhood through the synthesis of healthy dental structures, associated with ensuring the adequacy of vitamin D during pregnancy of the mother or taking the necessary poly-vitamins with providing their doses [23]. According to scientists such as Tsai PeiFen et al. in 1998, oligodontia was defined as a congenital absence of six or more teeth except for the third molars. Genetic factors play an important role in oligodontia, which can manifest as an isolated clinical finding or as part of a syndrome. Typical dental symptoms are reduced number of teeth, reduced tooth size, abnormal tooth shape and delayed tooth eruption. When 16 (sixteen) or more permanent teeth were missing, this pathology was corrected by orthodontic clinicians and removable partial dentures were placed to improve the patient’s chewing, speech function and aesthetic [24]. Vitamin D levels have been gaining growing attention in Oral Health [25]. There is growing evidence that vitamin D deficiency is associated with Iron Deficiency Anemia [26]. Vitamin D is a steroid hormone obtained mainly from exposure to sunlight and also from diet [27–31]. Foods naturally

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<th>Dental anomalies, Abnormal tooth</th>
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<td>Hypodontia 35</td>
<td>Hypodontia 12, 15, 25, 22, 45 Laganà G. et al., 2017</td>
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<td>Hypodontia 45</td>
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<td>Hypodontia 35</td>
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containing vitamin D and it can be found in oily fish (such as salmon, mackerel, and herring) and oils from fish (cod liver oil) [29].

Botelho J, Machado V et al., can conclude that vitamin D control levels prior to conception may be important to reduce the risk of enamel defects in primary teeth and should be controlled throughout pregnancy and after delivery [32].

Reed SG, Miller CS et al. 2020, conduct a detailed study. The aim of this study was to assess biomarkers of calcium homeostasis and tooth development, in mothers during pregnancy and their children at birth, for enamel hypoplasia in the primary maxillary central incisor teeth [33, 34]. In conclusion, the authors prove that the results suggest possible changing relationships of maternal and neonatal factors of calcium homeostasis during pregnancy and childbirth for enamel hypoplasia, contributing to the limit of knowledge about healthy dental development for the prevention of dental caries [35, 36]. According to Brook AH et al. 2014, understanding the process of morphogenesis and the variations in the outcomes is an important contribution to the multidisciplinary approach to new treatment [37].

Conclusion
1. Developmental anomalies associated with tooth agenesis include delayed tooth formation, prolonged primary tooth exfoliation, retained primary teeth, interdental spacing.
2. Enamel hypoplasia increases the risk of developing dental caries.
3. Early diagnosis allows optimal patient management and treatment planning and can reduce complications of the planned treatment.

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Conflict of interest
There are no conflicts of interest to declare by any of the authors of this study.

Patient informed consent
All patients have a pre-signed informed declarative consent for examination and treatment, signed by the parent accompanying the child.

References


