Case Report

The effects of idiopathic hypoparathyroidism on dental development

Zeki Arslanoglu[1]*, Halenur Altan[1], Osman Fatih Arpag[2], Ediz Kale[3], Fundagul Bilgic[4]
Departments of [1]Pediatric Dentistry, [2]Periodontics, [3]Prosthodontics and [4]Orthodontics, Faculty of Dentistry, Mustafa Kemal University, Hatay, Turkey

ABSTRACT

Hypoparathyroidism cases without etiology diagnosed in the first 10 years of life are called as idiopathic hypoparathyroidism (IHP). IHP is a rare endocrinological disorder among hypoparathyroidism cases, and the anomalies accompanying it are infrequently observed. Ca^{++} metabolism disorders occurring during the formation of tooth germs may cause enamel defects in teeth, enamel hypoplasia, enlarged pulp chambers, short roots, late eruptions, and missing teeth. The case presented in this article is a good example in terms of demonstrating how tooth damage ends with the commencement of treatment. Within this article, the results of a 9-year-old male IHP patient are discussed based on the literature. The said patient could not be diagnosed until the age of 18 months so developed enamel hypoplasia as well as enamel defects in this period due to abnormal mineralization, and no problems were observed in his dental tissue that formed after the ionic equilibrium was ensured following the diagnosis.

Key words: Dental development, Enamel defects, Enamel hypoplasia, Hypoparathyroidism, Idiopathic hypoparathyroidism

Access this article online

Website: www.jpediatrdent.org

DOI: 10.4103/2321-6646.174936

Quick Response Code:

INTRODUCTION

Hypoparathyroidism is an endocrinological metabolism disease characterized with hypocalcemia, in which endogenous parathormone is not released or is released inadequately, and accompanying hyperphosphatemia.[1,2] While this disease may be observed due to parathormone release disorders, surgical amputation of parathyroid glands, radiotherapy, after virus infections (such as measles) as well as alcoholism and intestinal absorption disorders (such as no intake of magnesium for a long time), it may also be seen idiopathically.[1-4]

When parathyroid glands do not function properly, blood calcium levels decrease while phosphor levels increase. In such a case, metabolic changes, dermatological changes, cataract, psychiatric disorders, neurological disorders, tetany as well as problems in the eruption, mineralization, and morphologies of teeth may occur.[5]

Hypoparathyroidism cases without etiology diagnosed in the first 10 years of life are called idiopathic hypoparathyroidism (IHP). IHP is a rare endocrinologic disorder among the hypoparathyroidism cases, and the anomalies accompanying it are infrequently observed. Dental symptoms may be seen only if there are parathormone release disorders during the formation of tooth germs as well as the eruption of teeth. Disorders of Ca^{++} metabolism during the formation of tooth germs due to late diagnosis may cause enamel defects in teeth,

How to cite this article: Arslanoglu Z, Altan H, Arpag OF, Kale E, Bilgic F. The effects of idiopathic hypoparathyroidism on dental development. J Pediatr Dent 2016;4:33-5.

***Address for correspondence**
Dr. Zeki Arslanoglu, Departments of Pediatric Dentistry, Faculty of Dentistry, Mustafa Kemal University, Hatay, Turkey.
E-mail: zekiarslanoglu@gmail.com

enamel hypoplasia, enlarged pulp chambers, short roots, late eruptions, and missing teeth.[5-9]

In this case report, it is aimed to share the defects that formed in the teeth of an IHP patient which have been mineralized before diagnosis, and the cessation of defect formation with the serum values balanced after the treatment.

CASE REPORT

A 9-year-old male patient applied to our clinic with the complaints of dental malformation and molar teeth damage. After the oral examination, enamel hypoplasia was observed in the enamels of all permanent first molars as well as permanent incisors [Figures 1 and 2]. According to his history, it was found out that the patient had IHP, and the disease had existed since his birth. However, it was detected that the muscle cramps and epileptic seizures he had experienced within the first 18 months following his birth had been misdiagnosed, and epilepsy treatment had been administered to him. It was detected that medical treatment was started after the diagnosis, and the muscle cramps, as well as the epileptic seizures, stopped with the commencement of medical treatment. It was understood that the blood values of the patient had not been followed until that day and that the hypoparathyroidism accompanying growth resulted in disorders of Ca^{++} metabolism affecting tooth mineralization negatively, and causing enamel hypoplasia in all of the first molars as well as permanent incisors.

The results of the laboratory tests performed after the patient's application to our clinic were as follows: Calcium 6.9 mg/dl (normal 8.50-10.60); phosphor 6.7 mg/dl (normal 2.30-4.70); alkaline phosphatase 121 IU/L (normal 30-90); and parathyroid hormone 7.6 pg/ml (normal 12-88). After the assessment of the results, blood ionic equilibrium was found to be unbalanced towards hypoparathyroidism, showing that the patient did not apply the recommended medical therapy properly. The patient was directed to a related specialist, and it was ensured that his values were taken under control again.

General health and height-weight status of the patient were appropriate regarding his age. Nevertheless, he had health problems associated with hyperthyroidism in terms of hypermetropia and dry skin.

The enamel defects and hypoplasia of the patient's incisor teeth were treated with composite laminate restoration. The defects and destruction in enamel were treated with composite restorations using resin modified glass ionomer based material. The patient is still kept under control, followed by our clinic with 6-month intervals.

DISCUSSION

Since 1966, only 7 articles have been reported indicating dental symptoms associated with IHP. In the literature, the first article was presented by Pisanty in 1966, in which hypoplasia in teeth, enamel defects, and unerupted persistent teeth were reported.[10] The largest number of patients was reported by Riley with 52 cases in the year 1969; however, dental symptoms were seen only in 21% of them.[7,10,11] In our case, tooth hypoplasia and enamel defects were observed; however, such disorders as unerupted teeth and short root morphology were not encountered. Just like other permanent damages occurring due to IHP, the formation of this condition also requires a long-term parathormone release disorder. The nonexistence of such release disorders prevented the formation of unerupted teeth as well as short root morphology. When the literature is reviewed, it can be seen that the mineralization of permanent first molars starts with birth, the mineralization of permanent canines starts in 5^{th} to 6^{th} (±2) month, and the mineralization of permanent incisors starts in 4^{th} to 5^{th} (±2) month according to Schour's odontogenesis scheme.[12] This explains the tooth mineralization as well

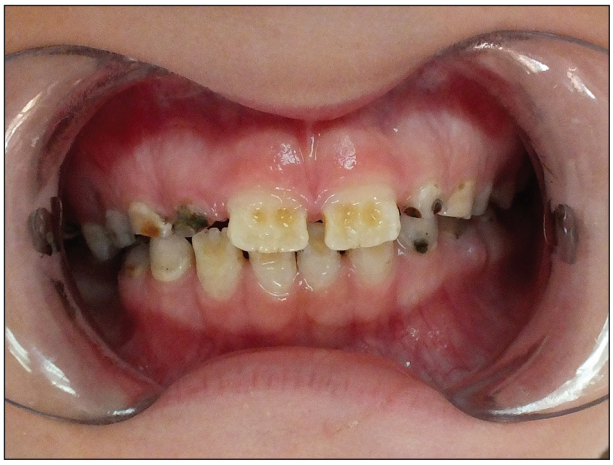

Figure 1: Enamel defects on incisors

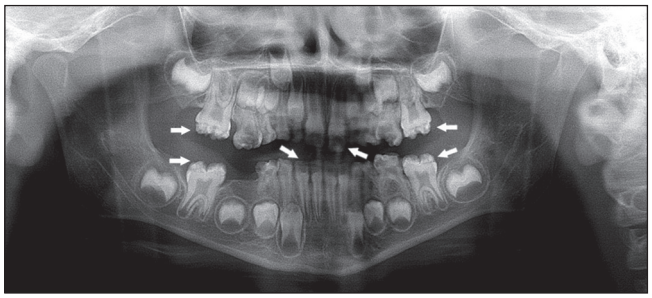

Figure 2: Radiographic view of severely affected teeth by the idiopathic hypoparathyroidism

as enamel hypoplasia and defects having formed in the first 18 months after birth.

Many conditions ranging from mental depression to psychosis may be seen in hypoparathyroidism patients.[13] Neurological seizures may be seen in almost 50% of all hypothyroidism patients.[13] In our case, the patient had neurological seizures after birth until the diagnosis and the commencement of medical therapy; the seizures stopped following the stabilization of ions. In our case, no permanent mental problem was observed, and it was found out that the patient had not experienced any psychiatric conditions. We think that this is because the ion destabilization lasted for a period of only 18 months. This period is short for a permanent neurological or mental damage formation. The risk of permanent damage would have been higher if such period had lasted much longer than 18 months.

Frequently observed dermatological abnormalities in hypoparathyroidism cases are dry skin, rash, and flat nails. Dry skin was observed in this case; however, problems related to nails were not seen.[11]

Frequently observed defects in cases of parathormone release disorders which are not treated for a long time include cataract and refractive errors.[14,15] In our case, hypermetropia was observed although cataract formation was not seen. Our patient has been experiencing hypermetropic problems since his early ages and using eye-glasses for therapy [Figure 3].

Consequently; dental symptoms associated with IHP, just like other permanent damages associated with this condition, become more frequent and diverse as treatment is delayed. Our case is a good example to demonstrate how dental damage can be stopped with the commencement of treatment. When the fact that patient may be a mentally recessive and depressive individual with disordered tooth development and morphology as well as vision disorders and cataract is taken into consideration, the vital importance of early diagnosis and treatment in idiopathic hypothyroidism can be better understood.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

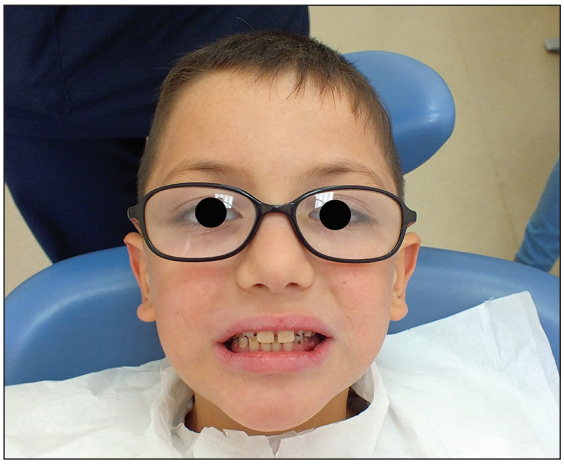

Figure 3: Facial view of hypoparathyroidism with hyperopic refraction error

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Özkara Ç, Atay T, Dalbayrak S, Arpaci B, Baybaş S. Common Cerebral Calcification and Neurological Findings Associated with Cases of idiopathic hypoparathyroidism. Dusunen Adam. J Psychiatr Neurol Sci 1992;5: 66-70.
2. Sharp C, Singer F, Krieger DT, Bardin CW. Current Therapy in Endocrinology. Philadelphia: The CV Mosby Co.; 1984. p. 266-72.
3. Baybaş S, Ozekmekçi S, Mumoğlu S, Denktaş F. Basal Ganglia Calcification-Hypoparathyroidism. Yeni Symposium, 1-2; 1988. p. 60-7.
4. Felig P, Baxter J, Broadus A, Frohman L. Endocrinology and Metabolism. New York: McGraw Hill Book Co.; 1981. p. 1057-74.
5. Kınoglu T, Omurlu H, Iscan HN. Dental Findings of Idiopathic Hypoparathyroidism and Maxillofacial Skeletal Morphology (A Case Report). Turkish J Orthod 1989;2:373-4.
6. Jensen SB, Illum F, Dupont E. Nature and frequency of dental changes in idiopathic hypoparathyroidism and pseudohypoparathyroidism. Scand J Dent Res 1981;89:26-37.
7. Riley DJ. Dental changes in patients with idiopathic hypoparathyroidism: Report of two cases. J Oral Surg 1969;27:44-7.
8. Greenberg MS, Brightman VJ, Lynch MA, Ship II. Idiopathic hypoparathyroidism, chronic candidiasis, and dental hypoplasia. Oral Surg Oral Med Oral Pathol 1969;28:42-53.
9. Pindborg JJ. Pathology of the Dental Hard Tissues. Copenhagen: Munksgaard; 1970. p. 182-4.
10. Pisanty S. Primary idiopathic juvenile hypoparathyroidism. Oral Surg Oral Med Oral Pathol 1966;21:294-8.
11. Kelly A, Pomarico L, de Souza IP. Cessation of dental development in a child with idiopathic hypoparathyroidism: A 5-year follow-up. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2009;107:673-7.
12. Schour I, Massler M. The Development of the Human Dentition. Chicago: Journal of the American Dental Association; 1941. p. 1153-60.
13. López-Villegas D, Kulisevsky J, Deus J, Junqué C, Pujol J, Guardia E, *et al.* Neuropsychological alterations in patients with computed tomography-detected basal ganglia calcification. Arch Neurol 1996;53:251-6.
14. Marx SJ. Hyperparathyroid and hypoparathyroid disorders. N Engl J Med 2000;343:1863-75.
15. Al Dhoyan N, Al Hemidan AI, Ozand PT. Ophthalmic manifestations of Sanjad-Sakati syndrome. Ophthalmic Genet 2006;27:83-7.