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Minerals and Vitamins Levels in Children with Enamel Hypoplasia

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Abstract

Objective: The present study aimed to estimate the deficiency levels of minerals and vitamins in children with enamel hypoplasia. **Materials and Methods:** Blood samples were collected from 100 children (50 with and 50 without enamel hypoplasia). The samples were then analyzed. **Results:** The mean differences of calcium, phosphorus and magnesium levels of children with enamel hypoplasia were significantly lower ($P \leq 0.05$) than those in the control group. Similarly, the mean differences of vitamin D3, A and K_2 in children with enamel hypoplasia were significantly less ($P < 0.01$) than children in the control group. **Conclusion**: Enamel hypoplasia could result from deficiency in one or more of some minerals and vitamins, including calcium, phosphorus, magnesium, vitamin D3, vitamin A, or vitamin K_2.

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Introduction

Because of genetic and general health conditions, a person may suffer from enamel dysplasia [1]. Pits or grooves may appear in the crowns of teeth afflicted by this condition, which is also referred to as "missing tooth structure," and the dentin under the enamel may be seen. One or a few teeth will be affected, or it might affect the whole mouth [2]. A defect's shape or location determines its classification. Classes of nativized enamel dysplasia include the following: pit-form; planeform; linear; as well as nativized enamel dysplasia [3]. A general or local disruption might lead to hypoplastic lesions in the teeth with enamel actively shaped. Dentin defects may also be limited to a specific location of the afflicted teeth, as they are formed over a long period of time [4]. People's susceptibility to enamel hypoplasia varies widely, and it may be used to infer information about their prior health and lifestyle [5]. The disease of ameloblasts—the cells that produce enamel—is thought to cause enamel hypoplasia, either temporarily or throughout the course of their lifespan [6]. There are several well-known causes of enamel dysplasia. Several of the reasons are inherited and environmental [7]. The degree and location of the defects depend on the timing and stage in which the enamel defect occurs [8]. Tooth decay and early childhood caries (ECC) are still a problem for many children with enamel hypoplasia [9]. Pits and missing enamel create a suitable environment for the adhesion and colonization of cariogenic microorganisms [10]. Enamel dysplasia has historically been overlooked as an ECC risk factor. It is possible for individuals to experience social humiliation or worry over their teeth's appearance because of biological process flaws in the enamel [11]. Environmental enamel hypoplasia has the same symptoms as hereditary enamel hypoplasia, but it can be triggered by various factors, including premature birth, malnutrition, infection with microorganisms and infectious agents, or trauma to the mouth and teeth that are still developing [12]. Therefore, this study aimed to investigate potential factors' deficiency caused by malnutrition and whether it directly results in enamel hypoplasia.**Error! Reference source not found.**

Material and Methods

An observational case-control approach was used in this study. Starting from January 2025 till July 2025, researchers gathered data for their investigation. The sample was collected from a private dental facility in Al-Hilla city, Iraq. A total of 100 children (50 children with

enamel hypoplasia and 50 healthy control children) were recruited in this research.

All children involved in this work were informed and the agreement was obtained from their parents before the collection of samples. Five ml of blood samples were collected from everyone. Two milliliters were deposited into EDTA, and the remaining three milliliters were gently pumped into gel tubes and left to clot at room temperature for 30 minutes before centrifugation at 3000 g for approximately three minutes [13]. Next, the sera were collected and kept in a freezer at -20°C until used in the analysis [14]. Calcium levels were measured by using Calcium kit (arsenazo III method). Phosphorus levels were measured by using standard colorimetric biochemical assays [15]. Magnesium levels were assayed by auto analyzer and compared to the reference value for normal children [16]. D_3 levels were measured by using VIDAS (Registered) 25 OH Vitamin D Total kit. Vitamin A levels were measured by using VIDAS 25 OH Vitamin A Total kit. Vitamin K_2 levels were measured by using VIDAS 25 OH Vitamin K_2 Total kit. Statistical analysis was done using SPSS version 23. To compare means between two groups, T-test was used.

Results

In this study, the mean differences of calcium level, phosphorus level and magnesium level between study groups including (children with Enamel hypoplasia and control group) was shown in Table 1 and Figure 1. The results showed that, there were significantly sever decrease ($P \leq 0.05$) in of calcium level, phosphorus level and magnesium level in children with Enamel hypoplasia compared with the control group.

In addition, the mean differences of vitamin D3, vitamin A and vitamin K_2 between children with enamel hypoplasia and the control or healthy group was shown in Table 2 and Figure 2. The results showed that, there were significant decrease ($P < 0.01$) in vitamin D3, vitamin A and vitamin K_2 in enamel hypoplasia children compared to the healthy group.

Discussion

Enamel dysplasia ensuing from severe metal deficiency affects esthetics, child self-esteem, and quality of life [17]. Enamel hypoplasia is that the results of an intermission within the method of enamel matrix formation inflicting defects in the quality and thickness of the enamel [18]. Enamel hypoplasia could also be delicate and should lead to corrosion of the enamel surface or the event of a horizontal line across the enamel of the crown [19]. There are cells called ameloblasts that are responsible for producing enamel. Morphogenetic, organizative, formative, maturative, protective, desmolytic are all parts of their life cycle, and they go through all six phases [20]. Throughout the ameloblasts' formative and maturative phases, the growth of enamel occurs. Mineralization of the enamel matrix happens in the maturation stage, when the enamel matrix is secreted in the formative stage [21]. Pathological amelogenesis is characterized by dysplasia, hypocalcification, or hypomineralization of the teeth [22]. Pitting, shape, or even a complete lack of enamel may be signs of enamel hypoplasia, which occurs when the matrix production process is impaired [23]. Fluorosis and growth defects are two of the most common manifestations of enamel development defects (DDE), which include dental anomalies such as hypoplasia and diffuse and defined opacities [24]. It's possible that an enamel crown defect called enamel dysplasia might be produced by an enamel matrix secretion problem, calcification problem, or maturation issue [18]. It's possible that hereditary and environmental factors, such as poor nutrition, exanthematous diseases like morbilli and chicken pox, inherent syphilis, hypocalcemia, or even a birth injury or premature delivery could trigger the development of enamel hypoplasia or hypo-mineralization. Native factors like infection or trauma from a primary tooth could also play a role [25]. Dysplasia is usually related to general disturbances that occur throughout the event of the permanent teeth, as well as exanthematous fevers [17]. However, enamel hypoplasia was shown to be the result of deficiencies in metal, phosphorus, and vitamins A, C, and D [26]. Blood calcium levels as markers of mineral loss are seldom effective since blood calcium levels remain relatively stable even in the most severe instances of calcium insufficiency with mineral missing from calcified tissues to maintain a liquid body substance physiological condition [27]. New research shows that children with bone mineral storage at their lowest levels are at greater risk of developing enamel dysplasia. A mineral shortage in liquid bodily material may also reduce or even stop the formation of calcification in dental tissues. This conclusion is based on the findings of this study [28]. Enamel hypoplasia is seen in children with a wide variety of metal balance problems, both genetic and acquired [29]. Hypovitaminosis (failure of bone matrix to mineralize) and severe enamel abnormalities are often associated with a lack of viosterol because of deficiency illness or inherited metabolic disorders [30]. Hypovitaminosis may be caused by a lack of vitamin D in children who don't get enough sunlight or who don't ingest enough viosterol [31]. Although nutritional rickets are assumed to be infrequent in affluent nations, the frequency may be growing in disadvantaged areas where milk is not supplemented with vitamin D and sunshine is scarce [32]. Toxic maternal smoking and vitamin D insufficiency during pregnancy, as well as neonatal tetany, are prenatal causes of enamel dysplasia, whereas postnatal causes include nutritional inadequacies [33]. Enamel dysplasia is more common in premature and low-birth-weight infants than in children who were born at a more conventional time and weight [34]. Premature delivery is associated with a wide range of health problems, including metabolic process immaturity, cardiovascular and nephritic abnormalities, brain damage, and anemia [35]. Additionally, preterm infants with hypocalcaemia, osteopenia, and hyperbilirubinaemia have a greater risk of developing enamel abnormalities [36]. In addition to a lack of metal and phosphorus mineral, a preterm child's digestive tract's inability to absorb minerals also contributes to the development of enamel dysplasia [37]. It has been shown that preterm infants who have laryngoscopy and endotracheal cannulation to treat metabolic distress are at increased risk of enamel damage in their main jaw dental teeth being damaged [18]. Teeth hypomineralization may occur in children with gastrointestinal illness and protein intolerance, resulting in a lack of absorption and mineral deficiency that can lead to tooth decay [38]. In children with chronic nephritic renal illness, mineralization pathways may be disrupted, putting them at risk for enamel abnormalities [39]. Either the errhine bacteria directly infect the ameloblasts, or their metabolic products or the high fevers caused in the patient may impair cellular performance [40]. Infections of the urinary system, redness, and greater illness have been linked to enamel abnormalities in clinical studies [41]. Enamel dysplasia in children was formerly thought to be caused by inherited VD, which could not be passed on via the mother's spirochete basal ganglia infection [42]. The enamel abnormalities was present in both the deciduous and permanent teeth is linked to a variety of infectious agent illnesses, including chicken pox, rubella, measles, mumps, grippe, and CMV [43].

Conclusion

Deficiency of essential minerals and vitamins responsible for normal enamel development including calcium, phosphorus, magnesium, vitamin D3, vitamin A, or vitamin K2 could have a direct association with enamel hypoplasia.

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Table 1. Mean difference of calcium level, phosphorus level and magnesium level between study groups.

Factors	Study groups		P-value
	hildren with enamel hypoplasia (N=50)	hildren without enamel hypoplasia (N=50)	
Calcium mg/dl	1.23 ± 0.27	4.42 ± 1.17	P≤ 0.05
Phosphorus mg\dl	1.93 ± 1.48	5.14 ± 1.34	P≤ 0.05
Magnesium mEq\L	0.38 ± 0.12	1.35 ± 1.49	P≤ 0.05

Table 2. Mean difference of vitamin D3, vitamin A and vitamin K_2 between study groups.

Vitamin	Study groups		P-value
	hildren with enamel hypoplasia (N=50)	hildren without enamel hypoplasia (N=50)	
Vitamin D3 nmol\L	26.18 ± 2.43	38.07 ± 6.18	P< 0.01
Vitamin A mcg\dl	13.07 ± 2.02	15.38 ± 2.53	P< 0.01
Vitamin K_2 mcg\dl	27.94 ± 2.20	61.45 ± 1.64	P< 0.01

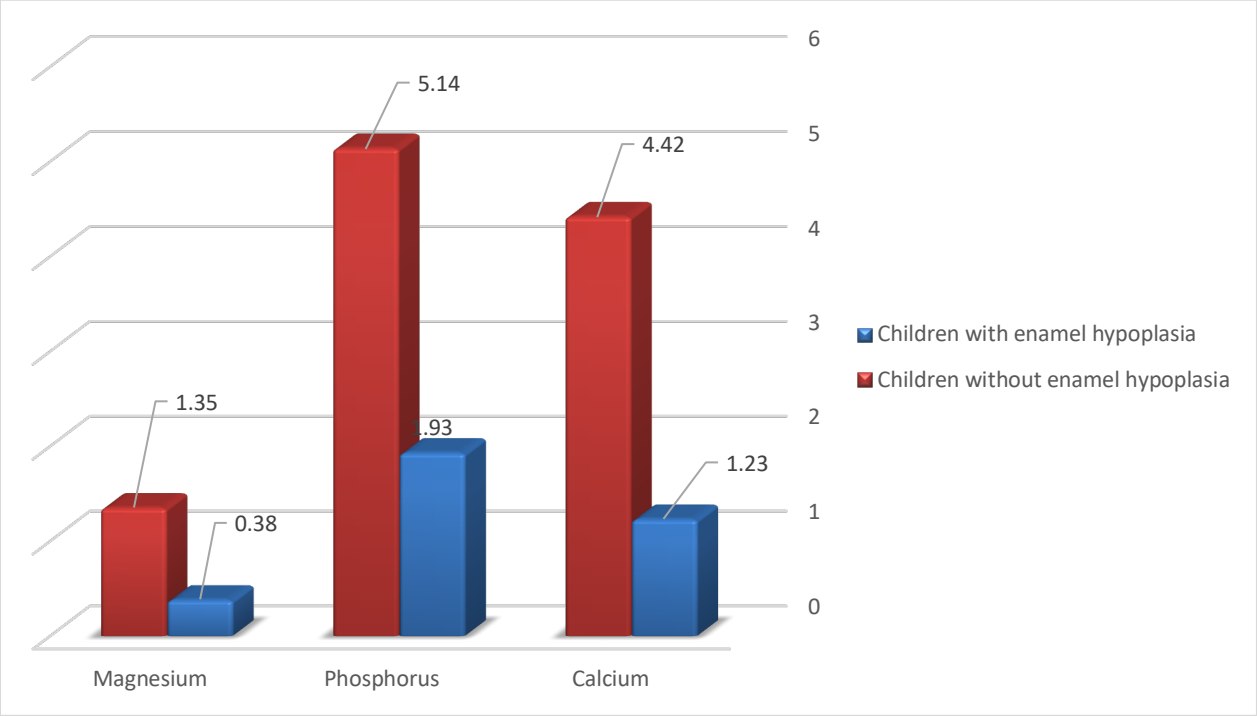

Figure 1. Mean difference of calcium level, phosphorus level and magnesium in children with enamel hypoplasia compared with control group.

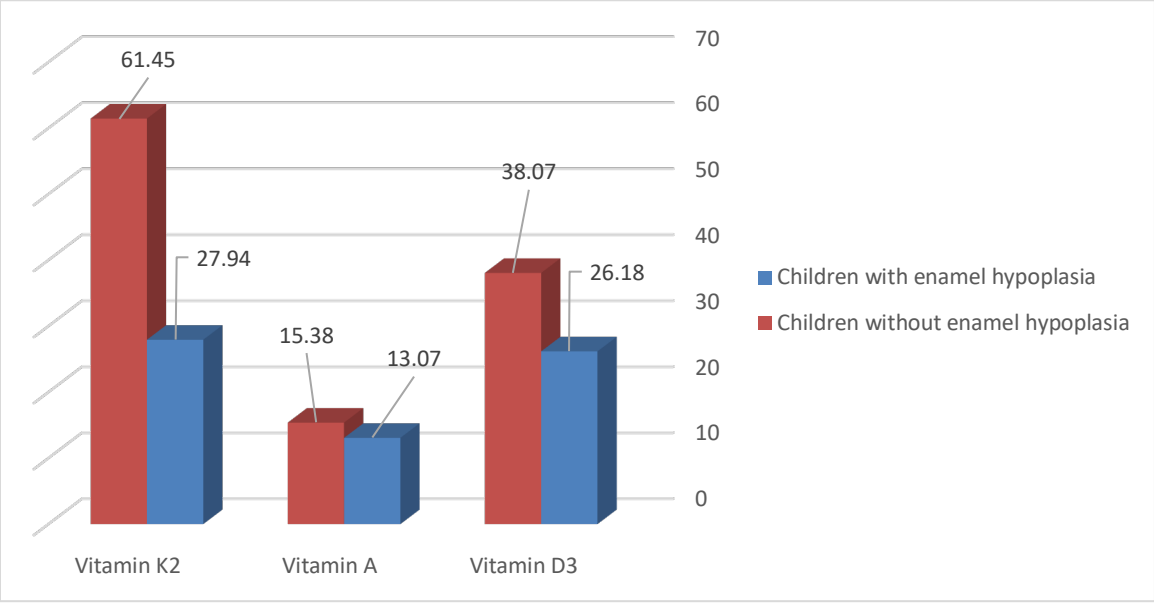

Figure 2. Mean difference of vitamin D3, vitamin A and vitamin K_2 in children with enamel hypoplasia compared with control group.