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Title:

Assessment of Vitamin D and Calcium deficiency as an etiological factor in delayed eruption of primary teeth

**A graduation research project
Presented to the council of collage of dentistry in partial
fulfilments for bachelor degree in dentistry**

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

"نرفعُ درجاتٍ من نشاءُ وفوقَ كُلِّ ذي علمٍ عَليمٌ"

صدق الله العلي العظيم

الآية (11) المجادلة

الإهداء

"الله نور السموات والأرض مثل نوره كمشكاة فيها مصباح المصباح في زجاجة الزجاج كأنها كوكب دري يوقد من شجرة مباركة زيتونة لا شرقية ولا غربية يكاد زيتها يضيء ولو لم تمسسه نار نور على نور يهدي الله لنوره من يشاء ويضرب الله الأمثال للناس والله بكل شيء عليم".

الى مشكاة بيت النبوة وموضع الرسالة ومهبط الوحي ومعدن الرحمة ومأوى السكينة ومُنْتَهَى الحُلُم، كوثر مُحَمَّدٍ المصطفى وبَضْعَتُهُ الكُبْرَى أم البُدُور المُنِيرَةِ والسُرْج المُضِيئَةُ والشُّهْب الثاقِبَةُ والأنجُم الزاهِرَةُ، أم الصديقين والشُّهداء فاطمة بنت مُحَمَّد عليها السلام الى مناراتِ العُلَى ومصابيحِ الخلود، أوتادِ الأرض يومَ اهْتَزَّتْ فَتَثَبَّتْها بإرادتهم الصَّالِبَةِ، إلى وَجْهِنَا النَّاصِعِ وهاماتنا المرفوعة في سماءِ العِزَّةِ. الى النفوسِ الأَبْيَّةِ، أساتذة الكرامة وحُماةِ مجدِ الأُمَّةِ، إلى مَنْ أَضَاءُوا دَرْبَ العِلْمِ والحياة بِشَهَبِ دِمَائِهِمِ الزَّاكِيَةِ، ووهبونا شرفَ المحاولة لنخطو خطاهم.. الى الشُّهداء السُّعْداءِ في مَقامِهِمِ الأَعْلَى، نَهْدِي هَذَا الجُهدَ المتواضعَ، راجين أن يكون وَفَاءً لِعِطائِهِمْ، وَخُطْوَةً تُضِيءُ دَرْبَ مَنْ يَأْتِي بَعْدَهُمْ .

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To all the professors at the College of Dentistry / University of Maysan, especially the one who supervised our research, this respected **Assist.Lecturer.**

Noor Abass AL-lamy We thank you all because you Nere sincere, dedicated, and generous with your giving. You have embodied the spirit of the virtuous teacher and have become an example to follow to all those who stood by us and supported us, we will remain grateful to you as long as we live, because you were quick to do good, and we find no reward for your benevolence except reciprocation with benevolence.

Abstract

Background:

Vitamin D deficiency is a condition that occurs when the body does not receive sufficient vitamin D for optimal vital functions. This study was conducted to evaluate vitamin D deficiency as a causative factor for delayed eruption of primary teeth.

Materials and Methods: The study included 74 infants of both sexes, aged 12 to 15 months, divided into two groups:

The first group (Group I) included 37 children with erupted teeth.

The second group (Group II) included 37 children with delayed eruption (no visible teeth).

Two milliliters of venous blood were drawn and placed in a test tube. Vitamin D levels were determined using an ELISA test kit, and the results were compared with the status of erupted teeth.

Results:

Group I included 17 males and 20 females, while group II included 21 males and 16 females. In the 12-month age group, there were 9 children without deficiency and 10 with deficiency.

At 13 months, there were 7 without deficiency and 8 with deficiency.

At 14 months, there were 11 without deficiency and 12 with deficiency.

At 15 months, there were 8 without deficiency and 9 with deficiency.

The results showed statistically significant differences ($P < 0.05$).

The mean vitamin D level in the first group was 32.5 ng/ml, while in the second group it was 13.8 ng/ml, and the differences were statistically significant ($P < 0.05$).

Conclusion:

There is a strong relationship between the timing of primary tooth eruption and vitamin D deficiency, and it can be concluded that vitamin D deficiency may be a causative factor for delayed tooth eruption.

Keywords:

Delayed tooth eruption, primary teeth, vitamin D deficiency.

Aim of the Study

The aim of study is to investigate the association between delayed eruption of primary teeth and deficiencies in vitamin D and calcium among children. The study seeks to identify whether insufficient levels of these essential nutrients contribute to altered eruption timing and to highlight the importance of early nutritional assessment in pediatric dental development.

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((Chapter one))
INTRODUCTION

1. 1 Vitamin D

Vitamin D (vitD) deficiency has essential effects on general health. It is known that oral and dental health is an integral part of public health, and there is a close relationship between them. From the development and eruption stages of the teeth to the formation of caries, vitD deficiency has accepted significant effects on oral health. It is essential to understand the role of vitD deficiency in early childhood caries (ECC), which is considered one of the most critical problems, especially in pediatric patients.

Low vitD levels during pregnancy have even been reported to increase ECC risk in infancy. For this reason, care should be taken to ensure that the mother's 25(OH)d level and later the child is in optimal conditions, starting from the pregnancy period, to improve the oral health status of children. Beyond just the mineralization of teeth, vitamin D deficiency (VDD) affects many other areas of overall health, especially immune system and bone growth(1).

In addition to being essential for the production of hydroxyapatite in teeth, vitamin D plays a critical role in calcium and phosphate homeostasis, which also helps to maintain bone density and prevent diseases like osteomalacia in adults and rickets in youngsters. Humans get vitamin D from exposure to sunlight, from their diet, and from dietary supplements (Table 1).¹⁻⁴ A diet high in oily fish prevents vitamin D deficiency.³ Solar ultraviolet B radiation (wavelength, 290 to 315 nm) penetrates the skin and converts 7-dehydrocholesterol to previtamin D₃, which is rapidly converted to vitamin D₃ (Figure 1).¹ Because any excess previtamin D₃ or vitamin D₃ is destroyed by sunlight (Figure 1), excessive exposure to sunlight does not cause vitamin D₃ intoxication. Few foods naturally contain or are fortified with vitamin D. The "D" represents D₂ or D₃ (Figure 1). Vitamin D₂ is manufactured through the ultraviolet irradiation of ergosterol from yeast. (Holick & Garabedian, 2006)

1.1.1 Source Of Vitamin D

Here are the main sources of vitamin D:

1. Sunlight: The body produces vitamin D when the skin is exposed to ultraviolet B (UVB) rays from the sun. About 10-30 minutes of sunlight a few times a week can help most people produce enough vitamin D.
 2. Fatty fish: Fish such as salmon, mackerel, sardines, and tuna are excellent sources of vitamin D.
 3. Fortified foods: Many foods are fortified with vitamin D, including:
Fortified milk (both dairy and plant-based options like almond, soy, and oat milk)
Fortified orange juice
Fortified breakfast cereals
 4. Egg yolks: Eggs, especially the yolks, contain small amounts of vitamin D.
 5. Cheese: Some cheeses, like cheddar and Swiss, provide small amounts of vitamin D.
 6. Liver: Beef liver is another food source, though it's not commonly consumed by everyone.
 7. Mushrooms: Certain types of mushrooms, such as maitake and shiitake, can contain vitamin D, especially when exposed to UV light
- For those with limited sun exposure or dietary preferences, vitamin D supplements are also widely available. (Bouillon, 2001)

1.1.2 Metabolism Of Vitamin D

It is now clear that vitamin D₃ can be produced in the skin or ingested in the diet. It accumulates very rapidly in the liver where it undergoes 25-hydroxylation, yielding 25-OH-D₃, the major circulating metabolite of the vitamin. 25-OH-D₃ proceeds to the kidney where it undergoes one of two hydroxylations. If there is a biological need for calcium or for phosphate the kidney is stimulated to convert 25-OH-D₃ to the 1,25-(OH)₂-D₃, a calcium and phosphate mobilizing hormone. If, however, the animal has sufficient supplies of calcium and phosphate, the 1-hydroxylase is shut down and instead the 25-OH-D₃ is converted to a 24,25-(OH)₂D₃. The role of the 24,25-(OH)₂D₃ remains unknown; it may be an intermediate in the inactivation-excretion mechanism. 1,25-(OH)₂D₃ proceeds to the intestine where it stimulates intestinal calcium transport and intestinal phosphate transport.

It also stimulates bone calcium mobilization and probably has other effects yet to be discovered in such tissues as muscle. The 25-OH-D₃-1-hydroxylase, which is located exclusively in renal mitochondria, has been shown to be a three component system involving a flavoprotein, an iron-sulfur protein (renal ferredoxin), and a cytochrome P-450. This system has been successfully solubilized, the components isolated, and reconstituted.

The 24-hydroxylase, however, has not yet been thoroughly studied. 1,25-(OH)₂D₃ is necessary for the appearance of the 24-hydroxylase; parathyroid hormone represses 24-hydroxylation. It is possible that the 24-hydroxylase represents the major regulated enzyme, so that its presence or absence may determine whether 1,25-(OH)₂D₃ is produced. Two metabolic pathways for 1,25-(OH)₂D₃ are known, conversion by the 24-hydroxylase to 1,24,25-(OH)₃D₃, and conversion of 1,25-(OH)₂D₃ to an unknown substance.

In the latter instance, there occurs loss of a side chain piece, including at least one of the 26 and 27 carbons. Whether 1,25-(OH)₂D₃ must be metabolized further before it carries out all of its functions has yet to be established. The primary excretion route of vitamin D₃ is via the bile into the feces. Urinary excretion appears small in magnitude and no

excretion products have yet been identified positively. Much remains to be learned concerning the metabolism and function of vitamin D and its metabolites.

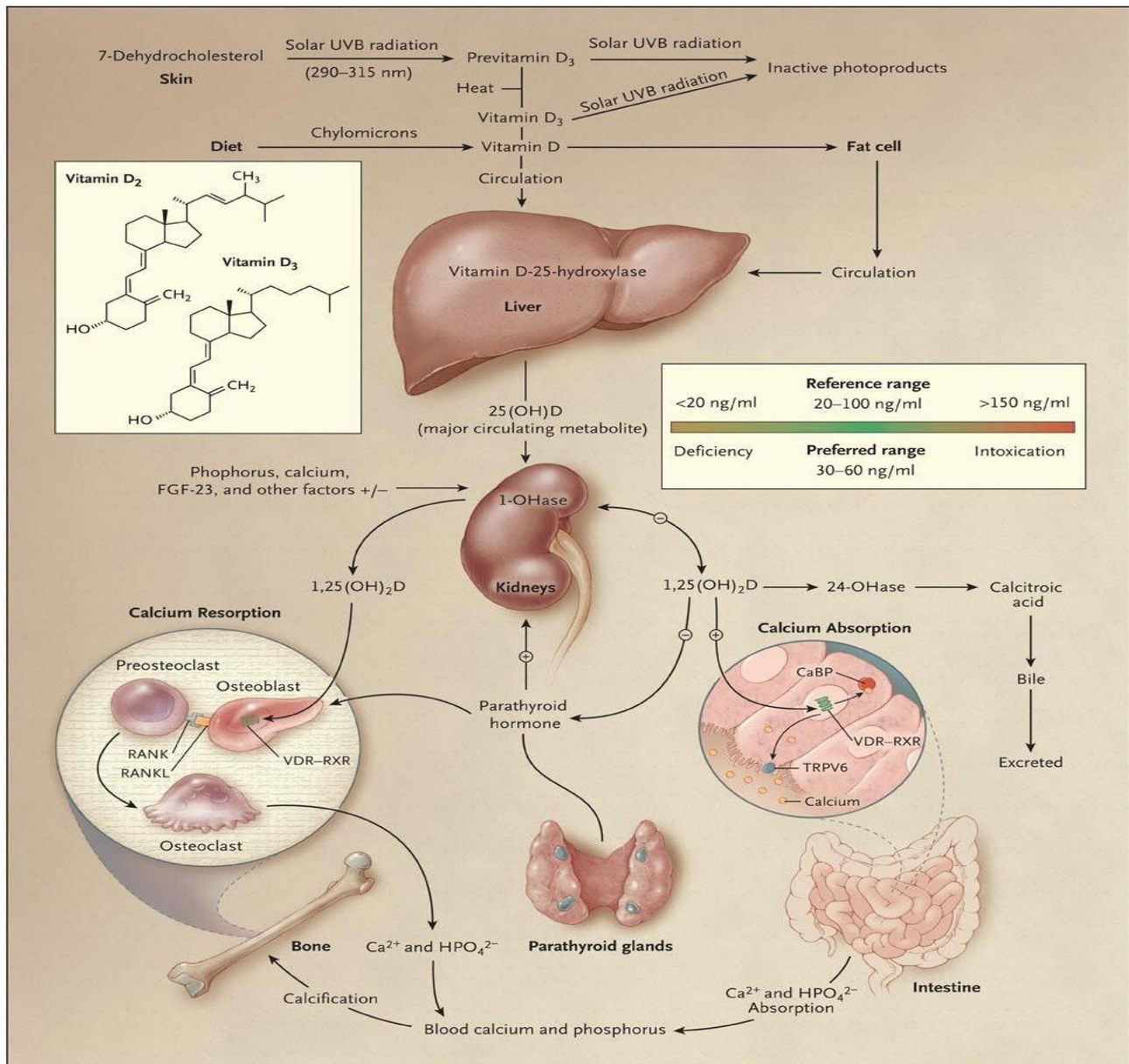
Source	Vitamin D Content
Natural sources	
Salmon	
Fresh, wild (3.5 oz)	About 600–1000 IU of vitamin D ₃
Fresh, farmed (3.5 oz)	About 100–250 IU of vitamin D ₃ or D ₂
Canned (3.5 oz)	About 300–600 IU of vitamin D ₃
Sardines, canned (3.5 oz)	About 300 IU of vitamin D ₃
Mackerel, canned (3.5 oz)	About 250 IU of vitamin D ₃
Tuna, canned (3.6 oz)	About 230 IU of vitamin D ₃
Cod liver oil (1 tsp)	About 400–1000 IU of vitamin D ₃
Shiitake mushrooms	
Fresh (3.5 oz)	About 100 IU of vitamin D ₂
Sun-dried (3.5 oz)	About 1600 IU of vitamin D ₂
Egg yolk	About 20 IU of vitamin D ₃ or D ₂
Exposure to sunlight, ultraviolet B radiation (0.5 minimal erythral dose)†	About 3000 IU of vitamin D ₃
Fortified foods	
Fortified milk	About 100 IU/8 oz, usually vitamin D ₃
Fortified orange juice	About 100 IU/8 oz vitamin D ₃
Infant formulas	About 100 IU/8 oz vitamin D ₃
Fortified yogurts	About 100 IU/8 oz, usually vitamin D ₃
Fortified butter	About 50 IU/3.5 oz, usually vitamin D ₃
Fortified margarine	About 430 IU/3.5 oz, usually vitamin D ₃
Fortified cheeses	About 100 IU/3 oz, usually vitamin D ₃
Fortified breakfast cereals	About 100 IU/serving, usually vitamin D ₃
Supplements	
Prescription	
Vitamin D ₂ (ergocalciferol)	50,000 IU/capsule
Drisdol (vitamin D ₂) liquid supplements	8000 IU/ml
Over the counter	
Multivitamin	400 IU vitamin D, D ₂ , or D ₃ ‡
Vitamin D ₃	400, 800, 1000, and 2000 IU

* IU denotes international unit, which equals 25 ng. To convert values from ounces to grams, multiply by 28.3. To convert values from ounces to milliliters, multiply by 29.6.

† About 0.5 minimal erythral dose of ultraviolet B radiation would be absorbed after an average of 5 to 10 minutes of exposure (depending on the time of day, season, latitude, and skin sensitivity) of the arms and legs to direct sunlight.

‡ When the term used on the product label is vitamin D or calciferol, the product usually contains vitamin D₂; cholecalciferol or vitamin D₃ indicates that the product contains vitamin D₃.

Table .1.Dietary, supplemen and pharmaceutical sources of vitamin D2 and D3



(1.2) Synthesis and Metabolism of Vitamin D in the Regulation of Calcium, Phosphorus, and Bone Metabolism.

1.1.3 Vitamin D Normal Ranges in Children and During Pregnancy

Vitamin D plays a critical role in calcium and phosphate metabolism, bone mineralization, and immune function. In both children and pregnant women, maintaining adequate levels of vitamin D is essential for proper growth, development, and overall health.

In Children:

The optimal serum concentration of 25-hydroxyvitamin D [25(OH)D], which is the best indicator of vitamin D status, is generally considered to be in the range of 30–50 ng/mL (75–125 nmol/L).

Levels below 20 ng/mL (50 nmol/L) are considered deficient and are associated with an increased risk of rickets and other developmental issues related to poor bone mineralization.

Concentrations between 20–30 ng/mL (50–75 nmol/L) are often classified as insufficient.

During Pregnancy:

Pregnancy increases the demand for vitamin D due to fetal skeletal development and maternal physiological changes. The recommended range for pregnant women is 30–60 ng/mL (75–150 nmol/L).

Levels below 20 ng/mL may increase the risk of complications such as preeclampsia, gestational diabetes, and low birth weight.

Maintaining sufficient vitamin D levels during pregnancy supports both maternal bone health and fetal development. (Anderson et al., 2016)

1.2. calcium and their Source

Calcium is an essential mineral with critical functions in the skeletal, cardiovascular, endocrine, and neurological systems. Approximately 99% of total body calcium is in bone, where it provides rigidity and structure to the skeletal system and acts as a calcium reservoir. The remaining fraction participates in metabolic processes, including vascular and muscle contraction, nervous system transmission, transmembrane transport, enzymatic activation, and hormonal function. The majority of studies of long-term consequences of inadequate calcium intake are related to bone health, especially rickets in children and fractures, osteopenia, and osteoporosis in older adults.

Calcium can be found in a variety of food sources, including:

1. Dairy products: Milk, cheese, yogurt.
2. Leafy greens: Kale, collard greens, spinach, bok choy.
3. Fortified foods: Fortified plant-based milks (almond, soy, oat), fortified orange juice, and breakfast cereals.
4. Fish with edible bones: Sardines, salmon (canned with bones).
5. Tofu: Especially if prepared with calcium sulfate
6. Nuts and seeds: Almonds, chia seeds, sesame seeds.
7. Beans and lentils: White beans, black beans, chickpeas.
8. Fruits: Oranges, figs, and blackberries (in smaller amounts).
9. Cruciferous vegetables: Broccoli, Brussels sprouts.
10. Seaweed: Wakame, nori.

These foods can help meet your calcium needs, depending on your dietary preferences (Bjarnason & Finnbogason, 1991)

]

1.2.1 Bioavailability of calcium

Calcium is a large mineral and not so easy to break down in the gut. The amount of calcium listed on the Nutrition Facts label of a food product is the measure of calcium in the food, but not necessarily the amount the body will absorb. The amount that is actually absorbed and used by the body is called “calcium bioavailability.” Some foods have higher calcium bioavailability than others. For example, dairy foods have a bioavailability of about 30% absorption so if a food label on milk lists 300 mg of calcium per cup, about 100 mg will be absorbed and used by the body. Plant foods like leafy greens contain less calcium overall but have a higher bioavailability than dairy. For example, bok choy contains about 160 mg of calcium per 1 cup cooked but has a higher bioavailability of 50%, so about 80 mg is absorbed.

Therefore, eating 1 cup of cooked bok choy has almost as much bioavailable calcium as 1 cup of milk. Calcium-fortified orange juice and calcium-set tofu have a similar total amount of calcium and bioavailability as milk, while almonds have slightly lower total calcium and bioavailability of about 20%. This may be useful information for those who cannot eat dairy foods or who follow a vegan diet. A downside to some plant foods is that they contain naturally occurring plant substances, sometimes referred to as “anti-nutrients.” Examples of anti-nutrients are oxalates and phytates that bind to calcium and decrease its bioavailability. (Nelson et al., 1983)

Spinach contains the most calcium of all the leafy greens at 260 mg of calcium per 1 cup cooked, but it is also high in oxalates, lowering the bioavailability so that only 5% or about 13 mg of calcium can be used by the body. The takeaway message is not to avoid spinach, which contains other valuable nutrients, but not to rely on spinach as a significant

source of calcium since most of it will not be absorbed by the body. You can also schedule your meals so that you do not eat “calcium-binding” foods like spinach at the same meal as calcium-rich foods or with calcium supplements. Also keep in mind that the exact amount of calcium absorbed in the body will vary among individuals based on their metabolism and what other foods are eaten at the same meal. In general, eating a variety of calcium-rich foods can help to offset any small losses. (Cochrane et al., 2008)

1.2.2 Calcium deficiency

Calcium deficiency can have several negative effects on teeth, as calcium plays a crucial role in the formation and strength of teeth and bones. The main effects of calcium deficiency on teeth include:

1. **Weakened Tooth Enamel:** Calcium is a key component of tooth enamel, which is the outer protective layer of the teeth. A lack of calcium can lead to weakened enamel, making teeth more susceptible to decay and sensitivity.
2. **Increased Risk of Cavities:** With weaker enamel, teeth are more prone to cavities (tooth decay) because the enamel is not as strong to protect the inner structures of the teeth from harmful acids and bacteria.
3. **Delayed Tooth Development in Children:** In children, calcium deficiency can lead to improper or delayed tooth development, resulting in weak, poorly-formed teeth.
4. **Tooth Loss:** Over time, a severe calcium deficiency can contribute to gum disease and bone loss, which can lead to tooth mobility and, eventually, tooth loss.
5. **Increased Sensitivity:** Without enough calcium to maintain the structural integrity of the teeth, the nerves within the teeth become more exposed, leading to increased tooth sensitivity

Ensuring an adequate intake of calcium through food or supplements is essential for maintaining healthy teeth and overall bone health. (NIH, 2021)

1.2.3 Normal Calcium Ranges in Children and During Pregnancy

In Children:

The normal serum calcium range for children is typically between 8.5–10.5 mg/dL (2.12–2.62 mmol/L).

Levels below or above this range can indicate problems with calcium metabolism.

During Pregnancy:

The normal serum calcium level during pregnancy is also 8.5–10.5 mg/dL (2.12–2.62 mmol/L), similar to non-pregnant adults. However, pregnant women may need more calcium from their diet for fetal development. (ADA, n.d.)

1.3 Tooth eruption

The word erupt has origins in the Latin word eruptus, which mean breakout, it refers to the axial or occlusal movement of the tooth from its developmental position within the jaw to its functional position in the occlusal plane (Bhaskar ,1991). In common usage, eruption signifies the cutting of the tooth through the gingiva. owever, the eruption of even a single tooth has to be understood in the broad context of various changes that occur in the jaws in preparation to, simultaneous with, and subsequent to the eruption of that tooth.

**These overlapping phases of
physiologic tooth movements are studied:**

1.3.1 Pre-eruptive tooth movement

It includes all movement of the primary and permanent tooth germs through tissues of the jaw prior to their eruption. Bony remodeling of crypt wall occurs to facilitate movements of growing tooth germ and its movement. In bodily movement in a mesial direction, bone resorbs on the mesial side and forms on the distal side of the crypt. There is a considerable change in position between the permanent incisor tooth germ and its deciduous predecessor in the first 2 years of life. The permanent molars, which have no deciduous predecessors, also exhibit movement (Nanci , 2009) The pre-eruptive movements of primary and permanent tooth germs place the teeth in a position within the jaw for eruptive movement (figures 3 &4) . (Nishida et al., 2000)

The movements are a mixture of total bodily movement of the tooth germ and a5.1. Pre-eruptive tooth movement:

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1.3.2 Eruptive tooth movement:

During the phase of eruptive tooth movement the tooth moves from its position within the bone of the jaw to its functional position in occlusion, and the principal direction of movement is occlusal or axial. However, as in the case of preeruptive tooth movement, jaw growth is still occurring while most teeth are erupting so that movement in planes other than axial movement is superimposed on eruptive movement (Srinath et al., 2013). The term ‘prefunctional’ eruptive tooth movement is used to describe the movement of the tooth after its appearance in the oral cavity till it attains the functional position. The actual eruption of the tooth, when it breaks through the gum, is only one phase of eruption, the final position of tooth in the oral cavity is determined by environmental factors, forces of muscles (the tongue, cheeks, and lips) that acts on the tooth, also the contact with other erupted teeth. Thumb sucking which is a childhood habit is an example of the environmental influence of tooth position (Avery and Steel, 1992) (Mayo Clinic, 2021)

1.3.3. Post eruptive tooth movement

This tooth movement is a complicated series of events to move the tooth in three-dimensional space. It is a passive process (unlike the pre eruptive and eruptive movement) that continues throughout the life time of the tooth. It results from attrition of the occlusal/incisal and proximal surfaces of the tooth which then allows for continued occlusal movement and mesial drift of the teeth (Craddock and Youngson, 2004). Post-eruptive tooth movements are those that (Nanci, 2013):

- (1) maintain the position of the erupted tooth while the jaw continues to grow.
- (2) compensate for occlusal and proximal wear (WebMD, 2021)

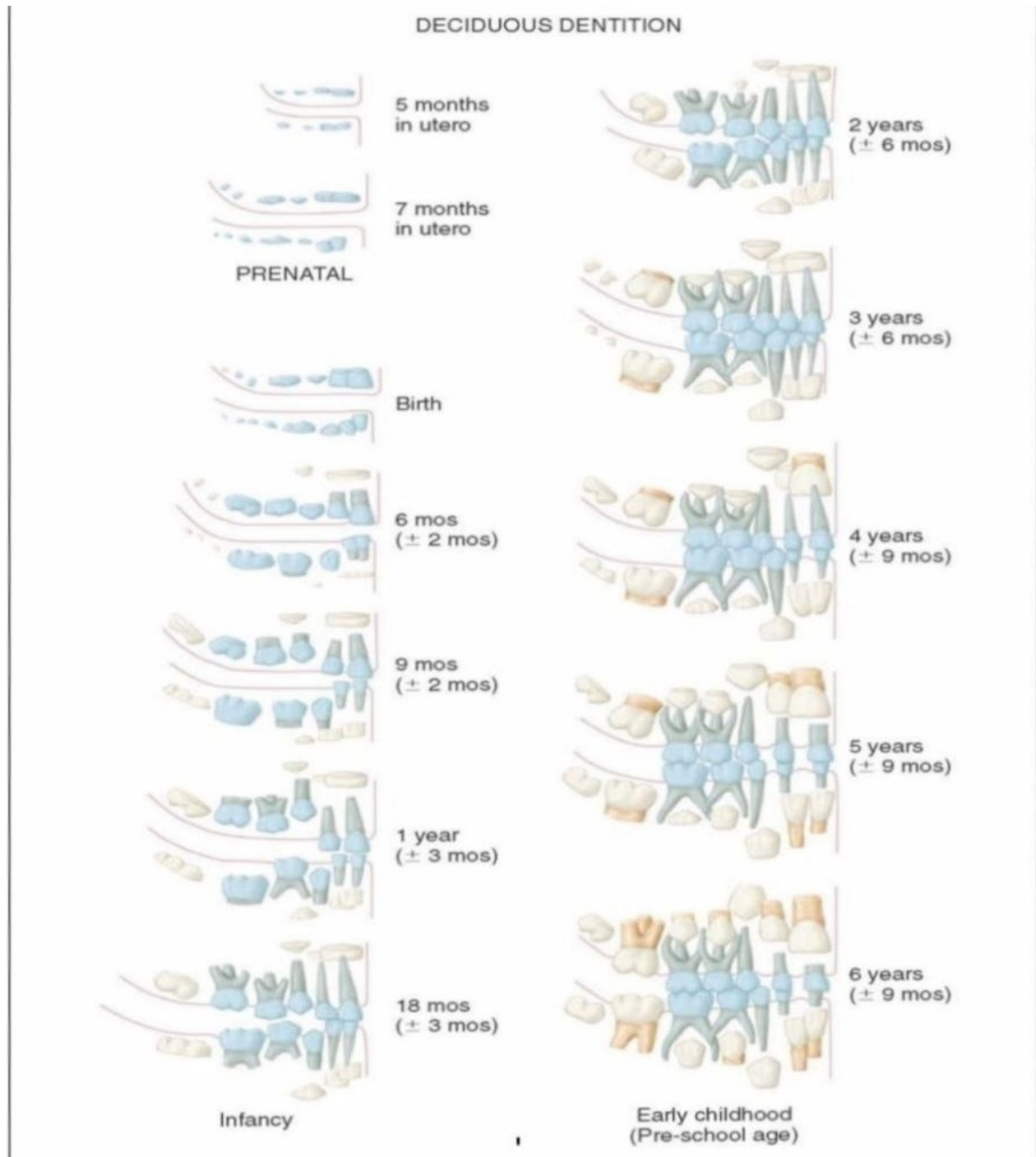


Figure 3 : chronology of development of deciduous dentition (Nelson, 2015)

1.4 There are several reasons that may lead to delayed tooth eruption, including:

1. Genetics: Certain genetic traits, such as delayed tooth eruption, can be passed from parents to children.
2. Nutritional Deficiency: A lack of essential nutrients, such as calcium and phosphorus, may affect tooth development.
3. Hormonal Disorders: Some hormonal disorders, such as pituitary gland deficiency, may lead to delayed tooth eruption
4. Genetic Disorders: Certain genetic conditions, such as Down syndrome, may cause delayed tooth eruption.
5. Growth Delay: Some children may experience overall growth delays, which could result in delayed tooth eruption.
6. Gum Inflammation: Severe gum inflammation may cause delayed tooth eruption.
7. Tooth Formation Disorders: Some disorders, such as hypercalcification, may lead to delayed tooth eruption.
8. Vitamin D Deficiency: A lack of vitamin D may cause delayed tooth eruption.
9. Thyroid Disorders: Certain thyroid disorders may lead to delayed tooth eruption.
10. Protein Deficiency: A lack of protein may result in delayed tooth eruption.

(Benabe & Martinez-Maldonado, 1978)

1.5 relationship between the parathyroid gland, vitamin D, and calcium:

relationship between the parathyroid gland, vitamin D, and calcium:

The Relationship Between the Parathyroid Gland, Vitamin D, and Calcium The parathyroid glands, although small in size, play a significant role in regulating calcium levels in the body, which is essential for maintaining various physiological functions such as muscle contraction, nerve signaling, and bone health. The parathyroid glands, in conjunction with vitamin D and calcium, work together to maintain homeostasis and ensure that calcium levels remain balanced. The complex interaction between these components is crucial for maintaining bone density, muscle function, and overall health.

Parathyroid Gland and Calcium Regulation

The parathyroid glands, four small glands located behind the thyroid gland, produce parathyroid hormone (PTH). PTH is the main regulator of calcium levels in the blood. When calcium levels in the blood fall below the normal range, the parathyroid glands secrete PTH to increase calcium levels. PTH achieves this by stimulating the release of calcium from bones, increasing calcium reabsorption in the kidneys, and activating vitamin D to enhance calcium absorption in the intestines.

Role of Vitamin D in Calcium Absorption

Vitamin D is crucial for the absorption of calcium from the gastrointestinal tract. When the body lacks vitamin D, the intestines cannot absorb sufficient calcium, which leads to lower calcium levels in the blood. To compensate for this, the parathyroid glands secrete more PTH to mobilize calcium from bones. Vitamin D is activated in the liver and kidneys, converting it into its active form, calcitriol, which increases calcium absorption in the intestines. Thus, vitamin D is essential for optimal calcium absorption and balance in the body. (Haussler & McCain, 1977)

Calcium Homeostasis and Bone Health

Calcium is vital for various functions in the body, including maintaining bone density and muscle contraction. The parathyroid glands play a direct role in regulating calcium homeostasis by releasing PTH, which influences calcium levels in the blood. When calcium levels are low, PTH stimulates bone resorption, leading to the release of calcium from bones into the bloodstream. In contrast, when calcium levels are high, the parathyroid glands reduce PTH secretion, which helps prevent excessive calcium release from the bones.

Interactions Between the Parathyroid Gland, Vitamin D, and Calcium

The relationship between the parathyroid glands, vitamin D, and calcium is tightly regulated. Vitamin D facilitates calcium absorption from the intestines, and if vitamin D levels are low, calcium absorption is impaired. In response, the parathyroid glands increase the secretion of PTH to maintain calcium levels. Excessive secretion of PTH can lead to bone loss, as calcium is released from bones into the bloodstream. In contrast, insufficient PTH secretion can result in low calcium levels, leading to conditions such as hypocalcemia. (McLean & Hastings, 1935)

Aim of the study:-

Disclosure of the relationship of parathyroid gland hormones with Calcium and Vitamin D regulation

((Chapter two))
Material and Methods

2.1 Method of Calcium Sample Collection:

The procedure for calcium sample collection is a standard laboratory practice aimed at determining serum calcium levels. This process is essential for diagnosing and monitoring various metabolic and endocrine disorders, particularly those related to bone health and parathyroid function.

2.1.1 Test Focus:

The laboratory may conduct testing for either total calcium or ionized calcium. Total calcium measures the overall calcium in the blood, including both bound and unbound forms, while ionized calcium reflects the biologically active form that is not bound to proteins.

2.1.2 Preparation Requirements:

Generally, no specific preparation is required before collecting a calcium sample. However, in certain clinical situations, patients may be advised to fast or temporarily discontinue calcium supplements to ensure accurate measurement of serum calcium levels.

2.1.3 Sample Collection Tubes:

Blood samples for calcium testing are typically collected in heparin or EDTA tubes. In some cases, a plain (red-top) tube may be used, particularly if the test requires serum separation for specific biochemical analysis. The choice of tube depends on the test type (ionized vs. total calcium) and the requirements of the laboratory protocol.

(Mallette et al., 1974)

2.1.3.1 Working Reagent Preparation

Mix equal volumes of Buffer (1) and Color Reagent (2).

Allow the mixture to stand for 10 minutes at room temperature before use.

2.1.3.2 Storage and Stability

The reagents and standard are stable up to the stated expiry date when stored at 15–25°C.

The combined working reagent is stable for:

- 7 days when stored at 22–28°C
- 3 days when stored at 15–25°C

2.1.3.3 Specimen

The preferred specimens are serum or heparinized plasma.

Calcium in serum remains stable for up to 10 days when stored at 2–8°C.

2.1.3.4 Precaution

To avoid contamination:

- Use clean laboratory wares.
- Avoid direct exposure of the reagent to light.

2.1.3.5 Assay

Wavelength: 587 nm

Cuvette Light Path: 1 cm

Temperature: 37°C

Measurement: Against reagent blank (Nordin, 1976)

2.1.3.6 Calcium OCPC Method – Procedure

Components	Blank	Standard	Sample
Pipette into cuvettes			
Working Reagent	1000 µL	1000 µL	1000 µL
Standard		10 µL	
Sample			10 µL

Procedure:

Mix the contents.

Measure the absorbance of the sample (As) and standard (Ast) against the reagent blank within 5–10 minutes.

2.1.3.7 Calculation

Serum Calcium (mg/dL) = $(\Delta A_{\text{sample}} / \Delta A_{\text{standard}}) \times 8$ (std. conc.)

To convert serum calcium from mg/dL to mmol/L, divide the result by 4.

2.1.3.8 Linearity

The test is linear up to 15 mg/dL (or 3.75 mmol/L).

Samples with higher values should be diluted 1+1 with distilled water.

Re-estimate calcium, then multiply the result by 2.

2.1.3.9 Normal Range (Serum/Plasma)

8.1 to 10.4 mg/dL

2.02 to 2.6 mmol/L

2.1.3.10 Quality Control

Any control serum with calcium determined by this method may be used. (Parfitt & Kleerekoper, 1980)

2.1.3.11 NOTES

1. Contaminated glassware is a common source of error. Use disposable plasticware for this test.
2. The test is not affected by:
 - Hemoglobin up to 20 mg/dL
 - Bilirubin up to 20 mg/dL
3. Lipemic and hemolytic samples:
 - Prepare a sample blank by mixing 0.05 mL of sample with 100.5 mL distilled water.
 - Measure absorbance against distilled water.
 - Subtract this value from the absorbance of the sample.
4. Buffer and Standard contain 0.1% sodium azide as a preservative.
 - Do not swallow.
 - Avoid contact with skin and mucous membranes. (Robertson & Marshall, 1979)

2.2 Method of Vitamin D Sample Collection:

The procedure for collecting a vitamin D sample closely resembles that used for calcium testing, with minor differences in test focus and patient preparation. These distinctions are essential for ensuring accuracy and reliability in laboratory results, particularly when comparing levels of different micronutrients.

2.2.1 Test Focus:

Calcium: Laboratory testing may be performed to assess either total calcium or ionized calcium concentrations.

Vitamin D: Testing is specifically conducted to measure the serum level of 25-hydroxyvitamin D, which is the most accurate indicator of vitamin D status in the body.

2.2.2 Preparation Requirements:

Calcium: No specific preparation is typically required prior to sample collection. However, in certain cases, patients may be advised to fast or to refrain from taking calcium supplements for a brief period before the test.

Vitamin D: Patients are generally instructed to avoid taking vitamin D supplements for a certain duration prior to testing to avoid falsely elevated serum levels.

2.2.3 Sample Collection Tubes:

Calcium Samples: These are usually collected in heparinized or EDTA tubes. In some cases, plain tubes may be used depending on the nature of the test being performed.

Vitamin D Samples: Blood samples for vitamin D assessment are commonly collected in heparin or EDTA tubes, with the primary goal of analyzing 25-hydroxyvitamin D levels. (Schneider & Sherwood, 1975)

2.2.3.1 Vitamin D (25-Hydroxyvitamin D) – ELISA Method

Principle

This method is based on a competitive enzyme immunoassay. Vitamin D in the sample competes with enzyme-labeled vitamin D for binding to specific antibodies coated on a microplate. The amount of bound enzyme is inversely proportional to the concentration of vitamin D in the sample.

2.2.3.2 Specimen

Serum or heparinized plasma is preferred. Store samples at 2–8°C and analyze within one week.

2.2.3.3 Assay Conditions

- Wavelength: 450 nm
- Temperature: Room temperature (20–25°C) or 37°C (based on kit instructions)
- Incubation: Multiple steps, typically 30–60 minutes each
- Measurement: Microplate reader

2.2.3.4 Calculation

Use the absorbance values of the standards to plot a standard curve. Determine the vitamin D concentration of the samples by comparing their absorbance to the curve (interpolation).

2.2.3.5 Normal Range

Vitamin D Level	ng/mL	Interpretation
< 20	< 20	Deficient
20 – 50	20 – 50	Sufficient
> 100	> 100	Toxic

2.2.3.6 Procedure Table

Step	Action	Time
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1	Add standards, controls, and samples to wells	—
2	Add enzyme-conjugated vitamin D reagent	—
3	Incubate	30–60 min
4	Wash wells (3–5 times)	—
5	Add substrate (TMB)	—
6	Incubate	15–30 min
7	Add stop solution	—
8	Read absorbance at 450 nm	Immediately

2.2.3.7 Materials and Equipment

Item	Description
ELISA kit (Vitamin D)	Includes coated plate, reagents
Micropipettes & tips	For accurate liquid handling
Microplate washer or manual	For washing wells
Distilled water	For dilution and washing
Microplate reader	Set to 450 nm
Incubator (optional)	For 37°C incubation if required
Timer	To monitor incubation steps
Disposable gloves	For safety and hygiene

((Chapter three))
Result and discussion

3.1 Discussion

The eruption of primary teeth is a highly regulated biological process that reflects the overall growth and development of the child. It is influenced by a variety of local and systemic factors including genetics, endocrine function, and nutritional status. Among these, the roles of Vitamin D and calcium have gained significant attention due to their established impact on skeletal and dental development. This study aimed to explore the relationship between delayed eruption of primary teeth and deficiencies in serum levels of Vitamin D and calcium.

Vitamin D, a secosteroid hormone, is essential for the maintenance of calcium and phosphate homeostasis, both of which are necessary for bone and tooth mineralization. It functions by enhancing the intestinal absorption of calcium and phosphorus, thereby ensuring sufficient availability for incorporation into developing bones and teeth. A deficiency in Vitamin D reduces calcium absorption, leading to hypocalcemia, which in turn can interfere with odontogenesis and eruption of teeth. In our study, a significant proportion of children with delayed primary tooth eruption were found to have suboptimal levels of serum Vitamin D, highlighting a clear association between hypovitaminosis D and dental eruption delay.

The biological mechanism behind this association can be explained by the role of Vitamin D in stimulating the expression of osteocalcin and other proteins involved in bone matrix formation and mineralization. During the eruption process, the dental follicle and surrounding bone tissues undergo remodeling, a process that is heavily dependent on the presence of adequate Vitamin D. Without sufficient levels, this remodeling is impaired, resulting in delayed eruption timelines. Several studies in the literature corroborate these findings, reporting delayed eruption, enamel defects, and increased prevalence of early childhood caries in children with Vitamin D deficiency. (Stamp, 1971)

Calcium, the most abundant mineral in the human body, is another critical element required for the structural integrity of bones and teeth. It serves as a primary component of hydroxyapatite crystals, which make up the hard tissue of teeth. In our findings, a significant number of children with delayed eruption exhibited low serum calcium levels, suggesting that calcium deficiency may be an independent or synergistic risk factor alongside Vitamin D deficiency.

Calcium also plays a vital role in cellular signaling, neuromuscular function, and enzymatic processes necessary for tooth development. An inadequate supply of calcium during the critical phases of tooth bud formation and mineralization may disrupt the sequence and timing of eruption. In addition, low calcium levels may lead to compensatory hyperparathyroidism, which alters bone metabolism and may further delay eruption by affecting the eruption pathway of the tooth through the alveolar bone. The combination of Vitamin D and calcium deficiencies has been shown to produce more pronounced skeletal and dental abnormalities than either deficiency alone. Our data supports this, as children with concurrent deficiencies demonstrated more severe eruption delays compared to those with isolated deficiencies. This synergistic interaction suggests the importance of evaluating both nutrients in pediatric assessments, especially when signs of developmental delay are observed.

It is also important to consider that the period of primary tooth eruption overlaps with rapid phases of physical growth, during which the demands for both calcium and Vitamin D are substantially increased. Children in this phase are particularly vulnerable to nutritional deficiencies due to factors such as inadequate dietary intake, limited sun exposure, malabsorption syndromes, and certain cultural practices that may limit Vitamin D synthesis. (Marshall & Bangert, n.d.)

((Chapter Four))

Conclusion and Recommendation

4.1 Conclusion:

although the cause-effect relationship between delayed eruption of primary teeth and deficiencies in vitamin D and calcium remains unclear, our study has found that the prevalence of delayed eruption, as well as lower serum levels of vitamin D and calcium, are significantly higher in affected children compared to the control group. This suggests that nutritional deficiencies may play a key role in the timing of tooth eruption and could be a contributing factor. Therefore, it will be helpful to routinely assess vitamin D and calcium levels in children with delayed eruption and consider supplementation therapy if necessary.

4.2 Recommendations

1. It is recommended to conduct routine screening for vitamin D and calcium levels in infants and young children, particularly those presenting with delayed eruption of primary teeth, to enable early detection and intervention of nutritional deficiencies (Kühnisch et al., 2020).
2. Public health awareness campaigns should be implemented to educate parents and caregivers about the importance of sufficient vitamin D and calcium intake during pregnancy and early childhood, as these nutrients are essential for proper dental and skeletal development (Holick, 2017).
3. Nutritional interventions should be encouraged through pediatric healthcare providers by promoting the intake of vitamin D- and calcium-rich foods, safe sun exposure, and the use of supplements when necessary, especially in at-risk populations (Moynihan & Kelly, 2014).

4. Further longitudinal and controlled studies are needed to explore the potential causal relationship between vitamin D and calcium deficiencies and delayed tooth eruption, and to assess the long-term dental and systemic health implications (Prakash et al., 2016).
5. It is necessary to develop standardized clinical guidelines for the assessment and management of children with delayed primary tooth eruption, including recommendations for laboratory testing, nutritional evaluation, and appropriate referral procedures (American Academy of Pediatric Dentistry [AAPD], 2022).
6. Interdisciplinary collaboration among pediatricians, dentists, and nutritionists should be strengthened to ensure a comprehensive approach in the prevention, diagnosis, and treatment of delayed tooth eruption related to systemic nutritional deficiencies (Arora et al., 2018).

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- 33.. *Tietz Textbook of Clinical Chemistry and Molecular Diagnostics* (Carl A. Burtis, David E. Bruns). It discusses the methods for blood sample collection and the proper handling of samples for various analytes, including calcium and its different forms.
- 34.. *Manual of Laboratory Diagnostics* (Diana M. Fraser). This manual covers the specifics of collecting blood samples for various laboratory tests, including ionized calcium.
- 35.. *Clinical Chemistry: Principles, Techniques, and Correlations* (Michael L. Bishop, Edward P. Fody, Larry E. Schoeff). This textbook provides an in-depth explanation of the laboratory tests for calcium and other electrolytes, as well as the different collection methods.
- 36.. "Tietz Textbook of Clinical Chemistry and Molecular Diagnostics" (Carl A. Burtis, David E. Bruns) – This textbook offers comprehensive guidelines on laboratory testing and the proper procedures for calcium analysis, including ionized calcium.
37. "Clinical Chemistry: Principles, Techniques, and Correlations" (Michael L. Bishop, Edward P. Fody, Larry E. Schoeff) – This resource provides details on clinical chemistry testing, including calcium tests and sample collection protocols.

38. *"Laboratory Medicine: A Handbook for Use in the Clinical Laboratory"* (W. J. Marshall, S. K. Bangert) – Discusses laboratory testing methods, including calcium levels, and provides guidance on appropriate sample handling.
39. *"Manual of Laboratory Diagnostics"* (Diana M. Fraser) – This book covers the specifics of blood sample collection for various laboratory tests, including calcium analysis.
40. *"Fundamentals of Clinical Chemistry"* (William J. Marshall, Stephen K. Bangert) – Offers a detailed explanation of clinical chemistry tests, including calcium measurements and the procedures for proper sample collection.
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46. *"Calcium and Bone Disorders: A Comprehensive Guide to Diagnosis and Treatment"* (M. Ian McCollum, Donald S. Boulanger) – This source addresses the interplay between calcium metabolism, parathyroid hormone, and vitamin D in bone health.
47. *"The Parathyroid Gland and Calcium Homeostasis"* (Henry C. K. Wong) – A detailed article focusing on how the parathyroid gland regulates calcium levels and its relationship with vitamin D.
48. *"The Role of Vitamin D in Calcium Homeostasis"* (Christopher M. O'Neill, et al.) – A research paper discussing the role of vitamin D in calcium absorption and how parathyroid hormone interacts with these processes.
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51.. Laboratory Medicine: A Handbook for Use in the Clinical Laboratory (W. J. Marshall, S. K. Bangert). This source provides detailed information on various types of laboratory tests, including calcium measurements and the appropriate tubes for sample collection.

52.. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics (Carl A. Burtis, David E. Bruns). It discusses the methods for blood sample collection and the proper handling of samples for various analytes, including calcium and its different forms.

53.. Manual of Laboratory Diagnostics (Diana M. Fraser). This manual covers the specifics of collecting blood samples for various laboratory tests, including ionized calcium.

الملخص:

الخلفية:

يُعد نقص فيتامين D حالة تحدث عندما لا يحصل الجسم على الكمية الكافية من فيتامين D اللازمة لوظائفه الحيوية بالشكل الأمثل. أُجريت هذه الدراسة لتقييم نقص فيتامين D كعامل مسبب لتأخر بزوغ الأسنان اللبنية.

المواد والطرق:

شملت الدراسة 74 رضيعاً من كلا الجنسين، تتراوح أعمارهم بين 12 إلى 15 شهراً، وتم تقسيمهم إلى مجموعتين:

(المجموعة الأولى) المجموعة I تضمنت 37 طفلاً لديهم بزوغ أسنان.

(المجموعة الثانية) المجموعة II تضمنت 37 طفلاً يعانون من تأخر البزوغ (لا يوجد أي سن ظاهر في الفم).

تم سحب 2 مل من الدم الوريدي ووضعها في أنيوب اختبار، وتم تقدير مستوى فيتامين D باستخدام مجموعة اختبار ELISA، وتمت مقارنة النتائج مع حالة بزوغ الأسنان.

النتائج:

تضمنت المجموعة الأولى 17 ذكراً و20 أنثى، بينما تضمنت المجموعة الثانية 21 ذكراً و16 أنثى.

في الفئة العمرية 12 شهراً، كان هناك 9 أطفال بدون نقص و10 يعانون من النقص.

وفي عمر 13 شهراً، كان هناك 7 بدون نقص و8 يعانون من النقص.

أما في عمر 14 شهراً، فكان هناك 11 بدون نقص و12 يعانون من النقص.

وفي عمر 15 شهراً، كان هناك 8 بدون نقص و9 يعانون من النقص.

وقد أظهرت النتائج وجود فروقات ذات دلالة إحصائية ($P < 0.05$).

كان متوسط مستوى فيتامين D في المجموعة الأولى 32.5 نانوغرام/مل، بينما كان في المجموعة الثانية 13.8

نانوغرام/مل، وكانت الفروقات ذات دلالة إحصائية ($P < 0.05$).

الاستنتاج:

توجد علاقة قوية بين توقيت بزوغ الأسنان اللبنية ونقص فيتامين D ، ويمكن الاستنتاج أن نقص فيتامين D قد يكون عاملاً مسبباً لتأخر بزوغ الأسنان.

الكلمات المفتاحية:

تأخر بزوغ الأسنان، الأسنان اللبنية، نقص فيتامين D.



جمهورية العراق
وزارة التعليم العالي والبحث العلمي
جامعة ميسان
كلية طب الاسنان

تقييم نقص فيتامين د والكالسيوم

كعامل مسبب لتأخر بزوغ الأسنان البينية

**مشروع بحث مقدم الى مجلس كلية طب الأسنان كجزء من متطلبات درجة البكالوريوس في طب
الأسنان**

مشروع مقدم من

فاطمة جبار شنين

زينب حمدان جاسم

اشراف الأستاذ

أ. م نور عباس اللامي