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Assessment of Caries Experience, Enamel Defects, Feeding Types and Area of Residency in Children with Nutritional Rickets

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ABSTRACT

Background: *Vitamin D insufficiency is a public reason of rickets, a situation that distresses bone development in children and that can have severe dental obstacles. VDD rickets happens utmost usually through infancy, beginning in the primary months of life. The absence of vitamin D through gestation can cause enamel hypoplasia of deciduous teeth. Deficiency over premature babyhood can upset permanent teeth and resulting caries can sometimes hint to tooth damage at a young age.* ***Aim of study:*** *To reveal the effect of nutritional rickets on the occurrence of dental caries and enamel defect in children, also to estimate the influence of nutritional rickets by feeding types and area of residency.* ***Materials and methods:*** *Total 30 children with vitamin D nutritional deficiency rickets with an age range from 1.5 years to 3.5 years of both genders were compared with 30 healthy children as a control group. Dentition status was measured according to WHO 1997, feeding type and area of residence were recorded while enamel defect was registered according to enamel defect index WHO 1997.* ***Results:*** *Caries experience represented by dmfs was significantly higher among control group compared to study group, while enamel hypoplasia was higher in the study group than the control group. Exclusive breastfeeding children are more prone to nutritional rickets than mixed and bottle feeding. Rural area children were at high risk to nutritional rickets.* ***Conclusion:*** *There is an obvious effect of nutritional rickets on caries experience and enamel hypoplasia. The 25-hydroxyvitamin D was lower in exclusive breastfeeding children.*

Keywords: Healthcare workers, Dietary habits, Workload, Weight

INTRODUCTION

Rickets is an unstiffening of bones in children owing to shortage or diminished metabolism of Vitamin D, possibly leading to breakages and abnormality [1]. A disease that happens throughout babyhood, rickets is the let-down of growing bone to mineralize [2]. The prevalence of vitamin D deficiency and nutritional rickets stays to be emphasized in a number of publications. Rickets is graded amongst the top 5 infantile diseases in developing countries [3].

Most cases of rickets in advanced nations are described to be in infants wholly breastfed without adequate packing of vitamin D, spending most of their lives in towns or houses with air toxic waste, and without sufficient exposure to sun, the number of aspects have been specified as being accountable for an extraordinary occurrence of vitamin D deficiency and rickets comprising area of residency and parental vitamin D deficiency [4-7]. Afterward birth, exclusively breastfed new-born may produce vitamin D using the UV radiation or obtain it directly by human milk [8]. Breast milk of well unenhanced lactating ladies comprises somewhat slight amounts of vitamin D and 25(OH) D and is regularly reflected inadequate to avert vitamin D deficiency in exclusively breastfed babies if their contact to sunlight is restricted [9]. Nutritional rickets typically presents at 6-24 months of age [10]. Since this is a serious time period of growth of teeth, the dental manifestation embrace enamel hypoplasia, delayed the formation of teeth and increased the incidence of cavities in teeth (dental caries) [11]. Deficiency throughout initial childhood can disturb permanent teeth and resultant caries from time to time can hint to tooth loss at a young age in adding to malocclusion [12].

PATIENTS AND METHODS

Total 30 children with vitamin D nutritional deficiency rickets were considered as a study group with an age range from 1.5 years to 3.5 years of both genders. They were examined at Paediatric Central Teaching Hospital in Nutritional

Rehabilitation Care Center (N.R.C) in Baghdad, Iraq with a duration of at least two months of illness. They were all with a confirmed diagnosis of vitamin D deficiency rickets according to the laboratory and physical examination compared with 30 healthy children. The diagnosis was confirmed by a special pediatrician at the same hospital as a control group. Ages of children were registered according to the last birthday [13]. Approval was achieved from the Ministry of Health for examining the children and all the parents of children were told about the purpose of this research and agreed with the research protocol.

Dental caries experience (dmfs) index including decayed surface (ds), missing surface (ms) and filling surface (fs) were diagnosed and recorded for all children according to the criteria of WHO, 1987 [14]. All the dental surfaces involving carious lesions were recorded. If there were missing teeth, they were recorded as (ms), filled surfaces as (f) and decayed surfaces as (d). According to WHO and UNICEF in 2002, feeding types of children study and control group were determined into 3 groups as exclusively breastfeeding group, bottle feeding group and mixed feeding group. Area of residency was recorded by questionnaire whether urban or rural area the children are living. Enamel defects were examined according to the criteria of the enamel defect index of WHO 1997 as shown in Table 1 [15].

Table 1 Criteria of enamel defect index (WHO 1997)

Code	Criteria
0	Normal
1	Demarcated opacity: the thickness of enamel is normal and have an intact surface but there is an alteration in the enamel translucency in variable degree. It was demarcated from the normal adjacent enamel with clear, distinct boundaries and could be white, creamy, yellow or brown in color
2	Diffuse opacity: an abnormality involving alteration in the enamel translucency that is variable in degree and white in color. There was no clear boundaries between the normal adjacent enamel and opacity, which can be linear or patchy
3	Hypoplasia: a defect that involve the enamel surface and associated with localized reduction in enamel thickness. It could occur in form of: a. Pits: single or multiple, deep or shallow, arranged in a row horizontally across the tooth surface or scattered b. Grooves: single or multiple, narrow or wide c. Partial or complete enamel absence over considerable region of tooth dentin. The affected enamel may be opaque or translucent
4	Other defects
5	Demarcated and diffuse opacity
6	Demarcated opacity and hypoplasia
7	Hypoplasia and diffuse opacity
8	All the three conditions
9	Not recorded

RESULTS

Caries experience for primary dentition (dmfs index) (mean and standard deviation) by fractions among children with nutritional rickets and control group was showed in Table 2.

Table 2 Caries experience of primary dentition by fractions (mean and standard deviation) among study and control group

Variable	Study group		Control group		T-test	p-value
	Mean	+SD	Mean	+SD		
d	1.030	1.220	2.870	3.810	-2.510	0.017
m	0.267	0.583	0.470	1.010	-0.940	0.352
f	0.167	0.379	0.400	0.855	-1.370	0.180
dmfs	1.470	1.910	3.570	4.540	-2.340	0.025

Results revealed that caries experience represented by dmfs was significantly higher among the control group compared to study group ($P<0.05$). Concerning caries experience fractions among study group, the same table illustrates decay component (ds) of dmfs index represented the highest proportion and statistically significant difference, followed by missing fraction (ms) while filled fraction (fs) constituted the least proportion. The control group (ds) component of dmfs index also represents the highest proportion followed by missing fraction (ms) and then filled fraction (fs) that constituted the least proportion. The data of the present study showed that the decayed surface value was significantly

higher among the control group than the study group (t= -2.51, P<0.017, df=34). The same result was found concerning missing and filling surfaces but the difference was not significant (p>0.05).

Regarding enamel defect among children with nutritional rickets and healthy children (Table 3) illustrates the scores of enamel defect according to the severity of enamel defect (score 0 = normal),(score 1=demarcated opacity),(score 2= diffuse opacity) and (score 3= hypoplasia). The current study revealed that the percentage of children with enamel defect score 0 (free from enamel defect) was more in the control group than in the study group.

Concerning score 1, a higher percentage of the study group affected with enamel defect were cited under score 1 followed by score 2. In addition, children with nutritional rickets complain from score 3 were higher % when compared with the control group (Table 3).

Table 3 Distribution of children study and control group with enamel defects by scores

Groups	Descriptive	Dental anomalies scores			
		Score 0	Score 1	Score 2	Score 3
Study	N	14.000	10.000	3.000	3.000
	%	0.460%	0.333%	0.100%	0.100%
Control	N	26.000	3.000	0.000	1.000
	%	0.860%	0.100%	0.000%	0.033%

Table 4 illustrate the distribution of children, study and control group, according to the area of residency which represented by urban and rural area. Regarding urban area, a low percentage of children with nutritional rickets was recorded in this area compared with control healthy children. The same table shows an opposite reading in a rural area, a high percentage was recorded of children with rickets in this area compared with healthy children.

Table 4 Distribution of children study and control groups according to area of residency

Groups	Urban area		Rural area	
	N	%	N	%
Study	14.000	0.460%	16.000	0.533%
Control	20.000	0.666%	10.000	0.333%

Table 5 illustrates the distribution of children, study and control groups according to the feeding type, which represented by breastfeeding, bottle feeding, and mixed feeding. Regarding breastfeeding type, current investigation recorded a high percentage of children with nutritional rickets under breastfeeding type compared with healthy children. Concerning bottle feeding type, the same table showed a low percentage of a study group under bottle feeding compared with the control group. At the same table, the finding of the current investigation revealed that a low percentage of rickets children under mixed feeding type compared with healthy children.

Table 5 Distribution of children study and control group according to feeding type

Groups	Descriptive	Feeding types		
		Breast feeding	Bottle feeding	Mixed feeding
Study	N	17.000	8.000	5.000
	%	0.566%	0.266%	0.166%
Control	N	10.000	12.000	8.000
	%	0.333%	0.400%	0.266%

DISCUSSION

Caries- experience in the current study was recorded using dmfs index. Vitamin D deficiency is a valuable indicator of dental caries, but it can also foretell a child's nutritional status [16]. The mean dmfs for study group was (1.47) which is lower than the mean dmfs of control group which was (3.57), This result may be attributed to the fact that nutritional rickets children selected for the study were under treatment regime until they became healthy so they had calcium level higher than healthy children, or due to caries expansion is typically a unhurried development with numerous years of delay before a cavity is detected, consequently, the serum 25(OH)D level at the time of caries counting may or may not be illustrative of the period once caries signs established [17]. Comparing with other studies, Schroth et al., showed that caries free kids have perfect 25(OH)D concentration (≥75ng/mL) matched to children with premature childhood caries which display lacking levels of 25(OH)D (<14ng/mL) [18].

While the results of the current investigation disagreed with Hofilena 2015 by illustrating that vitamin D ranks of children suffering from early childhood caries were statistically lower than control children [19]. This may be owing to, as soon as vitamin D levels are little, calcium is not completely absorbed and the body releases Parathyroid Hormone (PTH) to stimulate the mobilization of vitamin D from skeletal tissues.

Concerning enamel defect the result of this study showed that the percentage of teeth with score 1 (demarcated opacity), score 2 (diffuse opacity) and score 3 (enamel hypoplasia) in nutritional rickets children was higher than the percentage in healthy children, while normal teeth percentage (score 0) appeared higher in healthy children than nutritional rickets children. The results related to that vitamin D deficiency causes mineralization faults in teeth, leading to poorly mineralized and hypoplastic dentin involving calcospherites rather than appropriately mineralized dentin. This mineralization blemish may disturb dental development and development [20]. In addition, this may be explained by a fact that enamel is somewhat thin, hypocalcified or hypoplastic, as enamel and dentin formation occurs between 4 months in utero and 11 months of age, failings in primary dentition cannot be disallowed [21].

Schroth et al., observed that enamel are concomitant with complications through the prenatal and primary postnatal times especially, an in utero lack in vitamin D marks in a metabolic insult to ameloblasts and accordingly, results in the development in enamel hypoplasia [22]. In addition, the findings of the present study may be related to several contributing issues and risk factors which not involved in this study are proposed to cause or improve primary enamel faults, some of these are nutrition, hypocalcemia and low birth weight (<1.0 Kg) [22].

The result of the current study concerning enamel defects is in agreement with Surushi et al., and Zerofsky et al., when found that nutritional rickets cases of children had a larger amount of enamel defects than the healthy children and the difference was significant [23,24]. Nutritional rickets was related to defects in enamel which is reliable that one of the symbols of rickets in children is hypoplasia of enamel, leading to a superior vulnerability to dental caries [25]. In addition, another study was in agreement with the result of the current investigation, Galhotra et al., found enamel hypoplasia was noticed in 75% of children had nutritional rickets [26]. A deficiency in vitamin D levels adversely impacts tooth development by making noticeable developmental defects, such as enamel hypoplasia [27]. Breastfeeding informs numerous benefits to both mothers and infants [28,29]. Breast milk is broadly predictable as the most suitable food for babies, but it comprises very slight vitamin D [30], and protracted breastfeeding is connected with vitamin D deficiency rickets [31]. Breastfed infants are commonly safe from vitamin D deficiency rickets through the first few months of life, since vitamin D metabolites, specifically 25-hydroxyvitamin D [25(OH)D], do cross the placenta, such that neonatal 25(OH)D concentrations are approximately two-thirds of maternal values. It is estimated that the half-life of serum 25(OH)D is □3 wk; therefore, even if neonates do not receive an exogenous source of vitamin D throughout the first weeks of life, 25(OH)D concentrations should be reduced to values related with vitamin D deficiency solitary near the end of the second month, provided that the maternal vitamin D status is sufficient in pregnancy. An opposite association between the length of time of exclusive breastfeeding and 25OHD concentration [32].

Nevertheless, the results of the current investigation revealed that the percentage of breastfeeding type was higher in the study group than the alone, but this difference was not significant. Infants who are breastfed but do not receive supplemental vitamin D or suitable daylight coverage are at enlarged risk of developing vitamin D deficiency rickets [33]. In addition, the study by Soskic, et al., found a association between the risk of vitamin D deficiency and control group. In contrast, the percentage of bottle feeding and mixed feeding types were higher in control group than the study group. This study was agreed with Dawodu, et al., that exclusively breastfed infants are at bigger risk of vitamin D deficiency if vitamin D supplementation is missing and sun exposure is inadequate [34]. Taylor et al found no significant difference was detected in vitamin D intake by feeding type [35]. The infants receiving predominantly breast milk had lower vitamin D intake than those who received more formula or formula absence of supplementation amid breastfed infants [36]. The median period of exclusive breastfeeding was with an converse relationship between the span of a period of exclusive breastfeeding and 25OHD [37].

Taylor, et al., found that vitamin D intake is great in those who established more formula or formula alone, but this difference was not significant. Breastfeeding lacking supplementation was powerfully related with vitamin D deficiency amongst children, with a more than 10-fold increase in risk compared to children who were exclusively bottle-fed [38]. The result of the present study revealed the distribution of study and control group according to area

of residency showed that the percentage of nutritional rickets children living in a rural area were higher than healthy children, the finding may be due to the nature of rural children diet which is high content of unrefined cereals so that raises the possibility of dietary constituent such as phytate that impairing calcium absorption [39]. Studies are currently underway to assess intestinal calcium absorption among children with and without rickets and to determine the effects of phytate on absorption, therefore, in developing countries where calcium intakes are characteristically low and the population relies heavily on cereal-based staples, dietary calcium deficiency appears to be the major cause of rickets among children outside the infant age group [40].

Pettifor revealed in his study in South Africa that rickets among rural children was attributable to low dietary calcium consumptions and not vitamin D3 deficiency [3]. These children presented with active rickets, their diets were typically devoid of dairy products and high in phytate. Their calcium intakes were estimated to be □200 mg/d and were significantly lower than those of age-matched control subjects living in the same community.

CONCLUSION

There is an obvious effect of nutritional rickets on caries experience and enamel hypoplasia. 25-hydroxy vitamin D was lower in exclusive breastfeeding children.

DECLARATIONS

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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