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ANALYSIS OF COMPARISON OF VITAMIN D LEVEL OF CHILDREN WITH SEVERE EARLY CHILDHOOOD AND CONTROL GROUP

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ABSTRACT

A significant modifiable risk factor for dental caries in children is vitamin D deficiency. Dental caries can therefore be prevented by supplementing vitamin D in youngsters and avoiding vitamin D deficiency. To compare levels of vitamin D and relation of vitamin D deficiency with SECC in children with severe early childhood caries and children who have no caries. A total of 300 children (144 girls, 156 boys) with SECC aged 5 and compared to 300 caries-free, case-specific (gender and age) participants were investigated. Dental development was evaluated using the DA technique estimated by Demirjian et al.[1973] and a 2-sided t-test and chi-squared test were used to statistically analyze the data. The case group had a mean serum 25(OH) vitamin D of 12.19 ng/mL [4.37 SD standard, 95% 10.5–13.8 confidence range] whereas the control group had a mean serum vitamin D of 20.11 ng/mL (4.12 SD, 95% 18.56–21.65 trust interval). There was a statistically significant variation (p-value <0,0001) in the mean level of vitamin D serum 25(OH) between case and control groups.

INTRODUCTION

Early childhood caries (ECC) is a severe form of caries. One or more decayed, missing, or filled teeth for children from three to five years of age, or a decaying or missing score or filled score of more than four (three years) or > 5 (four years old) and >6 (five years) are serious caries from the early childhood (SECC) [AAPD 2016a].

Early childhood caries is defined as the occurrence in the child in 71 months of age or younger of one or more decaying (uncavitated or cavitated) lesions, missing (caries), or filled tooth surfaces.

Caries are a major public health issue in early childhood. 60% of children and 40% of adults have cavities in general. It is the most frequent, contagious infectious infection of children and difficult to control and can begin as soon as the teeth of the infant sprout. Multiple variables, such as microbes, food, oral health, medical disorders, or lack of vital minerals, such as vitamin D, contribute to the development of tooth decay. One billion individuals worldwide are vulnerable to a lack of vitamin D. In absorbing both calcium and phosphorus, 9,10 and vitamin D play a key role.

ECC is the most commonly reported chronic condition in children but remains mostly untreated in many countries under the age of three [aPD 2016a]. The prevalence of ECC is between 3 and 74 percent globally. ECC starts on the border of the gingiva in the upper primary incisors [Kawashita et al. 2011]. SECC affects not only the teeth but also more health-related and health-related issues, including restricted activity, reduced quality of life, increased treatment and cost, loss of days at school, reduced learning capacity, emergency visits, as well as delayed growth and development [AAPD, 2016b]. The underdevelopment of both height and weight was seen for children with SECC [Vania et al., 2011]. The World Health Organization (WHO) consequently recognizes that growth is an important indicator for the identification of child nutrition and health as the best single measure. In several studies, the association between BMI and dental caries has been assessed for children [Vania et al. 2011] but no dental development studies for children with ECC are available. The purpose of the present study was thus to explore dental growth in children with SECC by estimating dental age compared to age and gender-specific controls (DA).

BACKGROUND

Along with the traditional caries etiological triad, ECC is a complex illness influenced by environmental and health determinants[16]. Multiple risk factors including lack of oral hygiene, absence of dental homes, early colonization by cariogenic bacteria, extended use of bottles, frequent snacks, and socio-economic status have all been identified (SES). Another risk factor is enamel hypoplasia, as these deficiencies predispose to cariogenic bacterial colonization. The genetic condition, hereditary syndromes, and metabolic abnormalities, or localized causes can cause enamel hypoplasia. Systemic factors include mother or baby illnesses, metabolism, nutrition and deficiency diseases, preventative birth, and chemical compound exposures.

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It can be linked to the nutritional status of a youngster. For example, SECC may be an iron deficiency anemia risk sign. There are also other links between oral health in infancy and nutrition. A study assessing the influence of high-dose vitamin D surgery on oral babies showed that those with no surgery developed subsequent dental eruptions. Furthermore, the supplementation with prenatal vitamin D may minimize enamel hypoplasia in babies, an identified caries risk factor, as otherwise caries can easily colonize these enamel abnormalities.

In the 1920s and 1930s, May Mellanby first suggested that vitamin D immunity from both endogenous and exogenous sources may potentially be linked to caries. Her work has led to the idea that vitamin D is vital in avoiding and arresting cavities and not just needed for the normal growth and calcification of dentin and enamel. Insufficient vitamin D can also raise the risk of decay during times of tooth development.

Exogenous or synthetic exposure of skin to UV light may result in vitamin D enhancement in body. Special sources of food include fortified milk, fish, eggs, liver, cereals fortified, and margarine. The major circulating form is 25-hydroxyvitamin D [25(OH)D]. 25(OH)D is generated in the liver and acts as storage for vitamin D in our bodies. In addition, 1,25-dihydroxy vitamin D is hydroxylated into the kidney in its most active form. In absorbing dietic calcium from the gut, 1,25-dihydroxy vitamin D is important. The bone and other calcified tissues, including the teeth, should be developed normally and mineralized. Calcium metabolism is affected if deficiencies of vitamin D exist and the body cannot adequately absorb calcium. The low calcium content of parathyroid hormone (PTH) stimulates and mobilizes calcium from the skeletal source. It also encourages 25(OH)D to be converted into its most active form, 1,25-dihydroxy vitamin D, which enhances intestinal calcium absorption.

METHODS

Study was conducted in Riyadh elm Univeristy, Riyadh, Kingdom of Saudi Arabia.25(OH)D is the prime circulating form and is the finest overall status indicator since it represents an endogenous and exogenous measurement of overall achievements. Additional measures of other serum metabolites, particularly parathyroid hormones, are recommended to help with the adherence of vitamin D. (PTH). PTH is an indication of adequate adequacy through the inverse association between PTH and 25(OH)D. Consequently, PTH analysis was also conducted[1].

In the days from July to September 2008, the Misericordia Health Centre (MHC) in Winnipeg, a fluoridated city, recruited healthy children < 72 months old with SECC on their dental operation day. Either in response to poster ads or after a reference from a local pediatrician age-matched cavity-free controls were recruited from the community. After the examination was done to ensure that caries was free and that there was no current or previous caries experiment (dmfs=0 and dmft=0) check-ups were registered. The site of residence for all the participants was checked, and all the participating youngsters were either Winnipegs or communities in southern Manitoba to control the disparities in solar exposure which might vary according to the geographical latitude.

To get information on nutrition, diet, vitamins, and sun exposure, parents performed were interviewed. Questions included whether a youngster had visited a dentist, and whether the family could afford dentistry. Also noted were socioeconomic factors, including education levels, employment status, and whether government help had been granted to the family. Some factors have also been selected to evaluate their possible effects on levels of 25(OH)D.

In a proforma related to the dental caries status for both groups, a questionnaire designed to identify the medical history of the kid, their oral health, their food habits, their oral health, and their socio-economic position, was produced and filled out with parents of both the case and control groups' children. After parental permission, a clinical pathology laboratory technician estimated serum 25(OH) vitamin D was acquired for the venous blood sample (about 2.5 mL). It was done using mean, paired t-test, and basic linear regression that all obtained data was collated and statistical analysis.

RESULTS

Based on the reliability analysis a high-reliability factor has been discovered (0.999; p=0.001), and the descriptive data for all children in this study are presented in Table 1. The study comprised 300 youngsters (144 females and 156 males) with mean cAs, respectively, aged 5,0684±0,3892 and 5,0656±0,4155. Within 0.1 years of the CA of the kid in the study group, each case-control kid was matched to allow the direct comparison of the DAs. There were considerable disparities of mean DA-CAs between the children with SECC and case-controls, as compared with the DA-CA in the control group, and the DA-CA of the children with SECC was lower. Figure 1 shows an example panoramic x-ray for each group.

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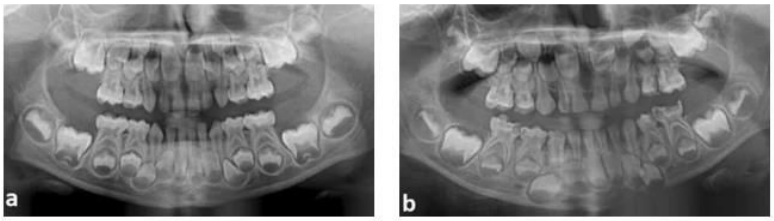

Fig. 1: A sample panoramic radiograph for each group

The girls and the boys showed a mean difference of DA-CA that was less than the case controls. Table 2 indicates this. Figure 2 compares the mean DA-CAs for the case controls between females with SECC, males with SECC, and all children with SECC. As Table 3 indicates, the early childhood gender-adjusted SECC is an independent predictor of the eventual difference between DA and CA. Table1

	Gender	C-AGE (mean± SD)	D-AGE (mean± SD)
Children with sECC	Females	5.0684±0.3892	5.7458±0.7936
	Males	5.0656±0.4155	6.0485±0.7144
Case controls	Females	5.1512±0.3808	6.6007±0.7427
	Males	5.0967±0.3729	6.6673±0.6943

Table 2

Group	N	Mean (DA-CA)	Std. Deviation	p
sECC group	300	0.8364	0.6716	
Control group	300	1.5125	0.6479	0.0001
sECC group-Females	144	0.6778	0.6714	
Control group-Females	144	1.4495	0.6844	0.001
sECC group-Males	156	0.9828	0.6398	
Control group-Males	156	1.5706	0.6087	0.001

Data analysis

The parameters for descriptions were presented as mean±SD. We have analyzed the data with a two-sided t-test. In addition, the 95% trust intervals were established using linear regression analysis in gender models. We thought of a value of p.

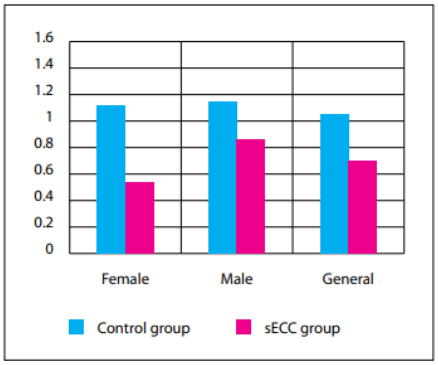

Fig 2: compares the mean DA-CAs between the females with sECC

ECC continues to impact children around the world as a prevalent childhood disease. In children with ECC, dental decay can have a substantial influence on overall health, quality of life, and development if it continues untreated. Caries if left untreated affects growth and development in various respects; for example, untreated caries can lead to reduced intake of food and malnutrition [Benzian et al. 2011]. Moreover, diminished quality of life may include decreased sleep, limited activity, and deficiencies in attention due to the suppression of growth hormone-releasing (GH). Increased production of GH can, more frequently, result in inhibiting GH release [Vania et al., 2011; Jenni et al. 2007] as a result of physiological reactions to stresses and suffering. Increased cytokine production could also occur since

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the development of an SECC could be linked to Chronic Inflammation from odontogenic infections. [Benzian et al., 2011; Means, 2003]. It was also hypothesized that SECC might be a risk to the development of iron deficiency anemia [Clarke et al., 2006].

Conclusion

This is the first study that shows an important relationship between ECC and dental development. In comparison to children without a cavity, children with ECC had decreased dental development. Clinicians should take care of their therapy by looking at the fact that these youngsters are more advanced in their dental development than youngsters with regular teeth. In general, children should not be overlooked for the necessity of immediate intervention and management of caries. Comprehensive programs for the improvement of children's oral health must be created and further investigations are needed to verify the conclusions of this research.

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