NUTRIENTS IN PRE- ERUPTIVE PHASE OF TEETH- A REVIEW

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ABSTRACT

Dental traumatic injury is a challenging health problem as these injuries could result in displacement, fracture, or loss of tooth and if not properly managed can have serious esthetic and emotional consequences.The difficulties associated with Calcium Hydroxide apexification such as prolonged treatment time, risk of fracture and improper apical bridge formation were overcome by the introduction of Mineral trioxide aggregate (MTA). This case report presents with the application of MTA for apexification followed by anatomic fiber post for maxillary central incisors reconstruction. Advantages of this technique include fewer visits, can be used in canals which are not round and are ideal for teeth with wider canals and thin dentin.

Key words: Apexification, Anatomic Fiber Post, MTA, Open Apex.

INTRODUCTION

The food consumed plays a large role in the development of dental tissues and oral environment. The diet, nutritional factors, frequency of consumptions, the quality of the food and its quantity alongside the microbial flora, all contribute to the overall health and disease of the teeth and oral cavity. While we are aware of which nutritional factors are necessary for the specific growth of the development of the oral cavity, the specific effects of malnutrition has not been adequately investigated. Two phases exist that are most influenced by the dietary factors affecting dental tissues.[1]

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1. The *pre-eruptive period* being the initial bell stage where tooth development begins until the time the mineralization occurs and the tooth breaks through the epithelium into the oral environment.
2. The *post-eruptive period* spans through the complete calcification and maturation of the tooth and its enamel which continues for a while after the tooth is present in the oral environment.

The purview of this review will cater to a discussion that is limited to an understanding of the data that exists so far about the effects of malnutrition on the development of teeth confined to the pre-eruptive phase of the same. In particular, protein- energy malnutrition and calorie deficiency along with vitamin and trace element deficiencies and their effect on tooth formation, maturation, eruption and susceptibility to disease specifically, dental caries will be considered.

Role of Protein- Energy Malnutrition and Carbohydrates

Menaker et al [2] in 1973 studied rats that were fed an ad libitum diet of 67% sucrose from tooth eruption at about 16-17 days of age and that the intensity of such highly caries-inducing diets can in fact neutralize the benefits of a good nutritious diet during the developmental stages of tooth thus leading to an inverse relationship between caries susceptibility and adequate nutrition. Besides, just like every other hard tissue and soft tissue in the human body, teeth also require a certain fulfilling environment to develop and grow fully in, during which nutrients have to be available.

Malnutrition in the early stages of eruption of teeth can affect enamel formation, saliva secretion, saliva flow and its composition and the antibody formation by the salivary glands.

A study was conducted by Holloway et al[3] wherein varying concentrations of sucrose/casein ratios were fed to female rats for about 4 weeks continuing into pregnancy and lactation. The various effects of altered ratios of sucrose and casein were studied on the teeth and structures within the oral cavity. It was concluded that rats born to females that were fed a higher sucrose and lower protein diet showed slower growth and that the eruption of their molars was also delayed. The molars that were seen were smaller than usual in size and there was a reduction in the thickness of dentin and also displayed missing cusps.

Shaw et al [4,5,6,7] conducted various studies in 1963, 1969 and 1972 on the effect of a protein deficient diet during reproductive phase in relation to tooth development. Shaw and Griffiths in 1963 investigated the impact of various sucrose/casein ratios fed during the perinatal period to female rats and concluded that the offspring born to these mothers showed abnormal cuspal pattern and low weight babies. Eruption of teeth was also seen to be delayed in the teeth. When a complete protein deficiency was imposed for 5 days at different times during lactation, third-molar eruption was delayed proportionally to how late the protein deficiency was imposed during the lactation period.

Studies by Larson et al[8] also concluded that prior to the emerging of the tooth into the oral cavity, any form of undernutrition could affect enamel structure, morphology and tooth eruption. DiOrio et al [9] (1973) confirmed that while undernutrition does affect tooth development at a later stage, the deficit caused had to be identified and categorized as under protein or calorie malnutrition. In an experiment done on five groups of rats- A, B, C, D and E, rat pups in groups A and B were fed protein rich diets while the other groups were fed protein deficient diets. Pups in group D were received true protein malnutrition while pups in group C received isocaloric supplements as well. Pups in group E received a complete calorie and protein deficit.

The results obtained indicated that a specific protein malnutrition during mineralization, stages of tooth development for the molars was seriously retarded. On administering protein supplements this change could be reversed to an extent, while administration of calories alone did not rectify the damage that the malnutrition had already caused by feeding the rats a limited amount of milk that also contained vitamins and minerals as well.

The results obtained by these animal trials pointed towards the next question being, determining if a specific malnutrition trait during the pre-eruptive phase of teeth development would affect the susceptibility to caries. This kind of study is difficult to do with human trials and there has been no such human trial owing to ethical issues. This review compiles all of the data concerning this discussion as seen concluded by animal experiments.

Menaker and Navia (l973a) designed a similar experiment to that of DiOrio et al wherein they fed rats diets containing 25% protein and 8% protein. A sub-group was also given milk supplements all of which were stopped at 19 days of weaning. The addition of protein alone counteracted the caries index and restoration of body weight as opposed to calorie substitution for the same and it was found that protein and not calories; was the primary contributory factor in overcoming the adverse effects of malnutrition on caries susceptibility in the rats. Table 1 highlights the various studies conducted with regard to the role assayed by calorie and protein deficiency in the pre-eruptive phase of teeth.

Fluoride, Dental Development, and Caries

Fluorides has proved its importance in dental development and maintenance of teeth to the least as its role in overall essentiality in the human body is still to be established. The reasons why fluorides are indispensable to the oral environment and teeth is because of their tooth morphology altering properties, causing a decreased solubility of enamel and bacterial enzymatic activity, formation of larger fluoropatite crystals in enamel and the stimulation of remineralization.

There are several studies proving that the benefits of fluorides are exerted on the tooth structure post the eruption of tooth into the oral cavity. However, studies done on cohorts of children evaluated the pre-natal administration of fluorides to expecting mothers. Horowitz and Heifetz (1967) and Carlos et al[19] (1962) studied several groups children that were prenatally exposed to fluoridated water in different patterns. Their results maintained that administration of fluorides in the form of fluoridated water to the mother did not transfer any major benefit to the offspring in the prenatal period. Carlos et al in 1962 and Howowitz and Heifitz[20] in 1967 also confirmed that the provision of fluorides does not bestow on any special properties to the primary teeth. Since the development of teeth takes place over a prolonged period of time, fluorides have to be given at a critical stage of tooth development in order to counteract mottling of teeth and induce cariostatic effects.

Arnold et al[21] in 1962 claimed that the administration of fluorides after the teeth have begun to erupt in the oral cavity is more beneficial in the control of caries. Investigations still need to confirm absolutely if fluoride intake in a specific dose from birth to about 6 years of age is advantageous or not.

The pre- and post-eruptive effects of fluorides was studied by Navia et al[22,23,24] in experimental rats on caries. It was found that when rat pups were given high doses of fluorides in the pre-eruptive phase, the molars were found to be hypoplastic and there were changes in the appearance of enamel and they were also more susceptible to caries. They also confirmed that when fluoride was fed post-eruptively, the molars displayed a significant decrease in caries susceptibility and did not show any mottling or brown stains.

Dental Dysplasias and Malnutrition

Baume et al [25,26,27]conducted several studies in 1966, 1968 and 1969 on Polynesian children of the school going age and noticed that two types of dental dysplasias existed. The first was an odontoclasia like phenomenon in primary teeth while the second was seen in permanent dentition in the form of 'yellow teeth.' He blamed these findings on a possible malnutrition state in the early stages of life when the teeth were undergoing mineralization and formation that may have led to the alterations seen in the enamel structure and morphology.

A certain hypoplasia was seen by authors in the deciduous dentition of 3-6 year olds wherein the defect was seen as a straight line of defected enamel across the incisors. Such a phenomenon was reported by Sweeney et al[28] in 1971 in Guatemalan children and termed as 'bar decay', by Nicholls et al[29] (1961) in Asiatic underprivileged children. The timing of this incidence seemed to match to birth or post-natal periods indicating that a state of inadequate nutrition may have been a causative factor.

Jeliffe et al [30,31] conducted several studies and called attention to the fact that a characteristic 'carved out erosion' in maxillary incisors was seen bilaterally in rural Jamaica (1954) and Haiti (1961) and a higher prevalence of it was seen in Cuna Indians in Panama being 31%. (Jelliffe et at., 1961).[32]

Vitamin A Deficiency and Dental Development

The influence of Vitamin A on the development of teeth is essential for tooth development and maturation. Wolfbach and Howe[33] in 1925 reported a study that involved young rats that were fed a vitamin A deficient diet and the diet mainly consisted of casein, starch, Brewer's yeast, McCollum's salt mixture and lard. While the experiment was being conducted no major changes were noticed in the teeth but the eruption rate seemed to have slowed down and changes were detected only microscopically. Table 2 contains a compilation of the studies assessing the role of vitamins and minerals in the maternal nutrition and its impact on tooth eruption and development.

The histopathological changes that were seen were irregular down growths of odontoblasts that indicated unusual deposits of dentin. The osteoblasts in the pulp were surrounded by islands of osteoid-like tissue and as the inadequacy of Vitamin A increased, the enamel formation seemed to have ceased. A narrow layer of stratified and non-keratinized epithelium had replaced the ameloblastic layer which was atrophic and shrunken in nature. One of the main investigators with regard to the role of nutrients in pre-eruptive phase of teeth was May Mellanby[34] who performed various experiments in relation to Vitamin A and its effects on teeth development and susceptibility to caries. Most of her work evaluated Vitamin A deficiencies alongside calcium and phosphorus. Mellanby used young dogs that were fed an array of differently chartered diets that were deficient in one or more nutritious factors to study distinct defects produced in teeth from pigmentation of enamel, induction of interglobular spaces in highly irregular and disorganized dentin. Her work was reviewed by Shaw and Sweeney,[35] 1973 and it was consistent with other related studies done by McCollum and Toverud who also suggested that an inadequacy of Vitamin A in the formative stages could eventually lead to an increased caries index afterwards.

Wolfbach and Howe (1933)[36] also conducted studies on the effects of deficiency of Vitamin A on the incisor teeth of guinea pigs and albino rats. Tooth formation takes place over a long period of time and teeth are subject to changes at any given stage of development when exposed to nutritional deficiency. It was concluded that Vitamin A deficiency in the pre-eruptive phase in particular, led to atrophic enamel epithelium, depolarization of ameloblasts and mostly affected the enamel organ.

A case was reported by Boyle[37] in 1933 in a 3 and a half year old child who showed an atrophic enamel organ, the histopathological findings of which displayed a replacement of the ameloblasts and stellate reticulum by a layer of non-keratinized squamous epithelium instead. Boyle and Bessey[38] also reported that the predentin was wider than usual and showed cell and capillary inclusions and abnormal calcification. Dinnerman et al[39] worked with post mortem infants to conduct these experiments that were aged between 3-7 months and observed that enamel hypoplasia and unsatisfactorily calcified dentin was seen but could not point out at a certain established connection of these findings with that of Vitamin A levels.

Schour and co-workers (1941)[40,41] established that Vitamin A level exerted influences on the odontogenic epithelium and its differentiation that led to altered morphologic and microscopic changes. This was consistent with the presence of abnormal epithelial cords found in the pulp by a continued proliferative activity of the odontogenic epithelium and its invasion into the pulp. This cellular disorganization translated into a gross distortion of the morphologic outline of the teeth, the replacement of which re-established the rate of dentin apposition and a faster differentiation of the peripheral cells next to the pulp into odontoblasts.

Mellanby (1941 a,b) was aware that nutritional deficiencies could be imposed upon the offspring through the mother and affect the growth of teeth, especially incisors as they are the first to erupt. Mellanby administered a natural diet that was scarce in Vitamin A levels, to rats for about 12-34 weeks after which they were bred with male rats. The offspring born from this breeding were killed at various ages. The young ones of the rats that were fed this diet for about 12 weeks produced progeny with normal dentition as compared to the progeny of mothers that were fed the diet for about 15-19 weeks. The latter displayed minor tooth alterations. The offspring of mothers to whom this diet was administered for even longer (24 weeks) showcased gross abnormalities in the shape and size of incisors and their growth. It was clear that a nutritional deficiency in the early stages of life led to detrimental and irreversible changes in the tooth histology and structure. Mellanby drew the conclusion that Vitamin A was essential for orderly bone remodelling and osteoblastic and osteoclastic function.

Similar results were obtained by Burn et al[50] who evaluated the teeth of rats that were maintained on a chronic Vitamin A deficiency for about a year. Atrophic and eventually absent ameloblasts and lingual odontoblasts were observed along with incisor teeth that presented loss of pigmentation, ridging, constrictions, and misalignment. Irving[51] evaluated the effects of avitaminosis A as well on the upper incisors of rats and observed excessive aggregates of abnormal dentin on the buccal surfaces of incisors and the presence of osteoid masses within the pulp.

The effects of minerals was also studied with relation to tooth development and caries susceptibility by Brown et al, Becks et al and Kruger et al to conclude that zinc, magnesium and other trace elements were essential for proper amelogenesis.

Conclusion and Summary

To ensure that the genetic information is expressed normally, nutrients have to be provided by the trophic environment. This period of nutrient exchange is critical to the development of oral tissues. Developmental dysplasia and similar developmentally anomalies may result, which are most often irreversible, if the required nutrients are not provided. This particularly holds true for tissues that are formed prior to the eruption of teeth, such as enamel. Ameloblasts that are responsible for its formation are highly sensitive cells and are lost soon after development. Thus, repair mechanisms at the cellular level fail to occur in these tissues, once the tooth has completely erupted in the oral cavity. In such tissues, the only reparative mechanism that can be relied up on for maintenance of its integrity, is the remineralizing property of saliva, and certain protective factors that are available and provided by food substances and water, for example, fluoride.

Pre- and postnatally, nutritional adequacy particularly in the form of nutrients such as vitamin A, protein and the mineral elements, is of primary importance to oral tissues. Other nutrients also may be necessary for maintaining the integrity of teeth within the oral cavity. However, an inference can be made that these nutrients, as mentioned above, have been experimentally proven to play a momentous role not only in the development of oral tissues, but also in the maintenance of oral health.

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Conflicts of interest

No conflicts of interest present.

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