

Consequences of premature birth on dental health

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ABSTRACT

Preterm infants are a special category of patients known for high rates of associated morbidities. These morbidities range from breathing problems to motor and neurologic impairments. The survival rates are improving along with better neonatal care, but the effects of preterm birth can still be seen in this category in patients. Even though dental health problems are not a largely debated subject, one should take into consideration that teeth, like other tissues are influenced by prematurity, and preterm babies are more likely to develop dental problems.

Keywords: premature birth, teeth, mineralisation problems, dental health

INTRODUCTION

The incidence of preterm birth ranges from 5% to 10% in Europe, North America, Australia and parts of South America, while in many countries from Africa and Southeast Asia the rate is between 10% and 30%. It correlates with differences in living conditions between developing and developed countries. Prematurity has a multifactorial aetiology and may be related to diseases of foetus and mother, but almost in half of the cases the aetiology is unknown. Epidemiological, microbiological and immunological recent studies suggested that periodontal disease default can be a risk factor for preterm birth and low birth weight (LBW). Evoked mechanisms include translocation of periodontal pathogens into the placenta and amniotic fluid and the action of a periodontal reservoir of lipopolysaccharides or inflammatory mediators (1).

Newborns are classified as PT if they are born at gestational week 33-36, very PT (VPT) if they are born at gestational week 29-32 and extremely PT (EPT) if they are born before the 29th week of gestation (2). The great improvement in neonatal intensive care over the last three decades has led to an increase in the survival of preterm infants, especially for EPT / ELBW (extremely low birth weight).

The short-term and long-term effects of premature birth on the child's physical and psychological growth and development are topics of current interest. Most studies have shown that premature infants have significant delays in many areas of physical and psychological growth and development. Although “catch-up” growth was reported later in childhood, some studies have indicated that there may be delays in the long term in adolescence (3). Like other tissues of the body structures of the mouth are also affected by the premature birth.

LONG-TERM COMPLICATIONS OF PREMATURITY

Morbidities associated with preterm birth often have physical, psychological and economic costs and are considered a global burden. Adolescents born with low GA or ELBW may have persistent neurological developmental problems, high blood pressure, and respiratory problems (4-6). Long-term morbidity did not decrease in the 1990s, but there has been an improvement in neurodevelopment in ELBW children over the past two decades (7).

The frequency and severity of these morbidities are higher the lower the GA, and the more common are the disabilities in males (8,9). A worse outcome can be predicted by major neonatal morbidities, but the frequency of morbidity does not increase with age (6). Chronic health conditions, including functional limitations such as cerebral palsy (5-17% vs. 1%), asthma (20% vs. 6%) and hearing and hearing impairment (9-27% vs. 1%), as well as cognitive impairment (40-50% vs. 5%) and neuropsychiatric disorders (10% vs. 5%), are more common among preterm infants with GA <26 weeks, compared to newborns older than this threshold (6,8,10,11). Motor capabilities are also affected in preterm infants, and these are correlated with lower IQ, lower academic ability and behavioural issues (12).

Cognitive function in children and adolescents / ELBW is lower, and children performed poorer in school compared to full-term children (12,13).

Eating and drinking habits develop later in prematurity and eating disorders have been reported (14-17). 42% of children with early feeding problems were born prematurely and the risk for a subsequent diagnosis of anorexia nervosa was three times higher among preterm infants with GA below 33 weeks (18,19).

EMBRYONIC DEVELOPMENT OF DENTITION

The development of dentition begins in the uterus around the 4th week of gestation and continues until the age of 20 years. Primary teeth start developing at 2 months of gestation, and the development of permanent teeth begin a few months before birth. Premature birth can affect those teeth that are in a critical stage of development at that time.

Teeth are created by the mineralization of a matrix mainly made of proteins. The beginning of this process is around the 4 month of gestation and the completion is in adolescence (20). Calcium, phosphorus, fluorine and vitamins A, C and D play a major role for dental development. The protein in dentin, collagen needs vitamin C for normal synthesis. Keratin from the enamel needs vitamin A to form. Phosphorus and calcium and are the basic chemical

elements of teeth, and vitamin D is needed for phosphorus and calcium to be deposited in the protein matrix.

The mineralization of the deciduous incisors is almost complete at 40 weeks gestation, and the mineralization of the other deciduous teeth crowns has begun. The calcification of the first permanent molar crown takes place at about 28 to 30 weeks of gestation and is not complete until the age of 3 to 4 years. The formation of all the anterior permanent teeth begins in the uterus, but the time to full development is very variable (21).

Maternal diseases can affect the development of teeth and can be associated with dental abnormalities. High blood pressure, preeclampsia, maternal diabetes, infections and nutritional deficiencies have all been linked to foetal size and oral structures abnormalities (22,23). Maternal smoking is associated with LBW and has associated with dental defects (21).

CRANIOFACIAL AND DENTAL FEATURES IN PRETERM INFANTS

Premature babies are facing growth deficits, but periods of “catch-up” growth during childhood seem to diminish this condition (24). It is not fully understood whether premature infants follow normal growth patterns in the growth of the facial skeleton. However, the circumference of the head is smaller at birth and remains smaller without the “catch-up” growth until the age of 11 years (25). It has been shown that craniofacial morphology in premature infants at the age of 8-10 years differs from full-term infants. They have a shorter anterior cranial base, less convex profile, shorter jaw length and more malocclusion features (26,27). Differences in craniofacial size in schoolchildren may be associated with a growth hormone deficiency (28,29). When growth deficiency was treated with hormones, these effects were reduced (30).

There are significant changes in the dental arches in the early period of mixed development, with an increase in the anterior segment. When the primary molars are exfoliated, the posterior segments decrease. The total circumference of the spring also depends on the increase in the width of the spring. When all these developmental changes are taken into account, no changes in the total perimeter of the arch are observed between the first permanent molars between the ages of five and thirty one (31). Dentoalveolar growth is predominantly genetic, but is also affected by external factors such as would be muscle activities associated with head positioning, finger sucking and predominantly oral breathing. A compensatory increase was observed, both in the development of the skeleton and in the dento-alveolar development in these cases (32-34).

CHARACTERISTICS OF JAW MORPHOLOGY AND MALOCCLUSIONS

In the postnatal period, preterm infants often require prolonged orotracheal intubation and gavage feeding, resulting in asymmetry and deepening of the palate (35,36). Palatine grooves and palate deepening due to pressure of the laryngoscope and intubation probe have also been demonstrated (37,38). These defects may persist, but have also been shown to disappear with age remodeling process (36,39,40). The narrow palate effect has been shown to be a common cause of cross-bites in school children (39). Both deep and open bites are more common in premature babies (39,41,42).

TEETH SIZE IN PREMATURE BABIES

The effects of premature birth, morbidity, and postnatal treatment during tooth development, such as mineralization defects, altered crown size and morphology, and eruptive disorders have been highlighted (43,44). There is mainly a genetically determination when it comes to teeth size (45). The size of the teeth in the primary and permanent dentition can be affected during the developmental stage given the fact that the matrix is secreted both prenatally and postnatally (46). An association between tooth size and maternal health, BW, GA, sex (47-51) has been demonstrated. However, there is no consensus that premature birth affects the size of the tooth.

PREMATURE BIRTH RELATED TO MINERALIZATION DISORDERS AND THE QUALITY OF TOOTH ENAMEL

The effects of preterm birth and low birth weight upon enamel development were demonstrated. Short GA and low birth weight, as well as morbidity and prenatal and postnatal treatments, have been associated with changes in enamel (52-56). Preterm children have high frequency of mineralization disorders (32-78%) observed in the primary and permanent dentition. In permanent dentition, up to 96% of premature babies were found with enamel defects (57).

Enamel is a unique hard tissue and it does not reshape, so structural changes resulting from lesions during its development are permanently recorded. Changes in tooth enamel are some of the most visible oral effects of premature birth and can

traditionally present as enamel hypoplasia, which is defined as a quantitative alteration of enamel, or as opacity of the enamel, which is defined as a qualitative change in enamel translucency (58). These defects are usually located on the primary teeth which are being mineralized around the period of premature birth – the primary incisors, canines and first molars, although the second primary molars may also be involved.

Microscopic studies of enamel from premature babies showed areas of increased porosity, hypoplasia and shallow pits (59). It is not known what are the effects of premature birth and associated morbidities, nor of the nutritional disorders and postnatal treatments upon the ameloblasts in different stages of evolution.

When changes of the porosity degree in the primary enamel are found in premature infants, chemical changes in the enamel and dentin may also be assumed. However, there are no studies on the chemical composition of enamel and primary dentin in the teeth of premature babies. Of particular interest is the incorporation of some of the major elements (P, Ca, C, O) during the mineralization of dental hard tissue. The physiology and pathology of calcium are regulated by magnesium, phosphorus, acid-base ratio, parathyroid hormone (PTH) and vitamin D. In the last trimester of pregnancy there is a major accumulation of calcium (100-150 mg/kg foetal weight) / day) (60).

Recent studies have shown that the effects of preterm birth can be extended to permanent dentition, although it was previously thought that enamel defects were limited to primary dentition (58).

CONCLUSIONS

The high survival rate of extreme preterm infants in particular contributes to the formation of a new group of children in society, with many risks and related chronic conditions. There is a general delay in the development of teeth in extremely premature babies and problems with the quality of the enamel have been observed. Malocclusions with cross bites, open bites and deep bites have also been reported. The dental clinician should consider that delayed dental development and eruption could have an impact on the timing of orthodontic diagnosis and the potential treatment for this category of children.

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