THE INCIDENCE OF ENAMEL HYPOPLASIA AND HYPOCALCIFICATION IN LOW BIRTH WEIGHT CHILDREN ACCORDING TO TEETH TYPE

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ABSTRACT
Objective: The aim of this study was to determine the type of deciduous teeth with hypoplasia and enamel hypocalcification in low birth weight children.

Material and Methods: Low birth weight baby is a neonate born with birth weight below 2500 grams. This was a clinical epidemiologically designed cohort study involving examination of hypoplasia and enamel hypocalcification in 147 low birth weight children in Child Health Department Hasan Sadikin Hospital Bandung, aged 9-48 months and 350 normal birth weight children as control group. Hypoplasia was diagnosed when there were pits and grooves on the surface of teeth, and hypocalcification was diagnosed when the teeth showed opacity. The examination was done prospectively for 6 months with 3 months intervals.

Results: The incidence of both enamel hypocalcification and hypoplasia in deciduous teeth is higher in low birth weight children (p<0.01).

Conclusion: The deciduous teeth of low birth weight children were more likely to have hypoplasia/hypocalcification, especially the first incisor tooth of the upper and lower jaws. Hypoplasia and hypocalcification occur during a critical phase within the intrauterine developmental period and affect different types of teeth differently, possibly due to their different time of development.

Keywords: Low birth weight, Enamel hypoplasia, Enamel hypocalcification

Introduction

The growth and development or odontogenesis of deciduous teeth starts at around 6-7 weeks of age of the embryo and progresses through several phases - initiation, proliferation, histodifferentiation, morphodifferentiation,  aposition, calcification, maturation, and exfoliation.²,³ The critical period of deciduous teeth growth and development occur very early prenatally, during the embryo and fetal periods 2-8 weeks age, while the critical period of the permanent teeth of course occurs postnataally.²,⁴

Teeth enamel forming/amelogenesis is a process of development of enamel matrix and then calcification.¹,²,³,⁴ This is a most sensitive period for disorders that might cause problems with teeth structure, which, occurring during enamel matrix formation, might cause enamel hypoplasia, a deficit or even no enamel being formed, and, deficit in the subsequent teeth calcification period causes hypocalcification – overall, tooth hypomaturation.⁴,⁵,⁶

Hypoplasia and hypocalcification can manifest as mild to severe - mild hypoplasia when there is a groove or small horizontal pit, and severe when there is a groove on the pit along the ‘crown’ or no enamel forming - mild hypocalcification when there is an area of opacity and severe when the teeth look very dark, yellowish or brownish.⁴,⁵,⁶,⁷

Low Birth Weight (LBW) of children, i.e. of less than 2500 grams, indicates Intra Uterine Growth Retardation (IUGR), caused by maternal, fetal, or placental factors. Maternal factors - mother with Cytomegalovirus, or TORCH infection, and severe infection during the first trimester of pregnancy, being more than 35 years old is a risk factor as is Diabetes Mellitus, hypertension/preeclampsia, and malnutrition. Fetal factors are generally chromosomal abnormalities. Placental factors – Placentitis, placental infarct. Amelogenesis is affected by genetic and environmental factors, prenatal systemic factors that might affect/disturb the growth and development of the organs, including deciduous teeth.⁵,¹²
About 70% of LBW children are Small for Gestational Age (SGA) and comprise Symmetrical and Asymmetrical types. In the Symmetrical type there is less growth in each of head circumference, body length and body weight, and this occurs at the beginning of pregnancy, whereas in disturbances of the Asymmetrical type the body weight is proportionally less than both the body length and head circumference, and it occurs later during the second or third trimester of pregnancy. Symmetrical SGA children therefore are expected to exhibit greater hypoplasia and enamel hypocalcification.

Since the different types of teeth develop at different stages, the aim of this study was to be more specific, to find out which of the teeth most often get hypoplasia or enamel hypocalcification in Low Birth Weight children.

Method and Aims

The research design was clinical epidemiology, a cohort study of given sample size and an age-matched control group. The subjects consisted of 147 Low Birth Weight (LBW) children aged 9-48 months, born at Hasan Sadikin Hospital Faculty of Medicine Padjadjaran University, and 350 Normal Birth Weight children as a control group. Variables are: outcomes - hypoplasia and hypocalcification; predictors - children with LBW; and discriminators - teeth type.

LBW - children with birth weight below 2500 gram. Hypoplasia - a pit, hole, and groove on the surface of the tooth (Variable: Hypoplasia). Hypocalcification - when the teeth show opaque/opacity, yellowish or brownish dark colour and non-transparency (Variable: Opacity).

With parental informed consent, intra oral examination of the children entailed using mirror and sonde to detect hypoplasia and hypocalcification. Tests were repeated at two month intervals for six months (i.e. progressively four times for each child) to examine if there was hypoplasia/hypocalcification in other teeth erupting later.

The aim of this study was to determine the type of deciduous teeth with hypoplasia and enamel hypocalcification in Low Birth Weight children.

Results

The results of hypoplasia and enamel hypocalcification of 147 LBW and 350 Normal Birth Weight children are shown in Table 1 and 2.

Table 1. The incidence of deciduous teeth hypoplasia and hypocalcification in Low Birth Weight children according to the type of teeth.

<table>
<thead>
<tr>
<th>Teeth</th>
<th>Hypocalcification (opacity)</th>
<th>Hypoplasia</th>
<th>Teeth</th>
<th>Hypocalcification (opacity)</th>
<th>Hypoplasia</th>
</tr>
</thead>
<tbody>
<tr>
<td>51</td>
<td>62.75</td>
<td>18.3</td>
<td>61</td>
<td>62.75</td>
<td>17.65</td>
</tr>
<tr>
<td>52</td>
<td>57.82</td>
<td>7.19</td>
<td>62</td>
<td>57.52</td>
<td>7.84</td>
</tr>
<tr>
<td>53</td>
<td>26.14</td>
<td>3.27</td>
<td>63</td>
<td>26.14</td>
<td>3.92</td>
</tr>
<tr>
<td>54</td>
<td>16.99</td>
<td>2.61</td>
<td>64</td>
<td>16.34</td>
<td>2.61</td>
</tr>
<tr>
<td>55</td>
<td>7.19</td>
<td>-</td>
<td>65</td>
<td>6.54</td>
<td>-</td>
</tr>
<tr>
<td>81</td>
<td>58.86</td>
<td>6.54</td>
<td>71</td>
<td>58.21</td>
<td>5.88</td>
</tr>
<tr>
<td>82</td>
<td>45.1</td>
<td>3.27</td>
<td>72</td>
<td>43.79</td>
<td>3.27</td>
</tr>
<tr>
<td>83</td>
<td>27.45</td>
<td>1.31</td>
<td>73</td>
<td>28.76</td>
<td>1.31</td>
</tr>
<tr>
<td>84</td>
<td>15.03</td>
<td>2.61</td>
<td>74</td>
<td>15.03</td>
<td>2.61</td>
</tr>
<tr>
<td>85</td>
<td>6.54</td>
<td>0.65</td>
<td>75</td>
<td>6.54</td>
<td>0.65</td>
</tr>
</tbody>
</table>

In this study 51, 61, 71 and 81 are the most often affected; and 52-55 and 62-65 and 72-75 and 82-85 are progressively less affected.
Diagram 1.  The incidence of hypoplasia and enamel hypocalcification of deciduous teeth in Low Birth Weight children according to the type of teeth.

(Note: Max – maxillary, upper teeth; Mand – mandible, lower teeth)

The diagram shows the incidence of the defect of deciduous teeth in LBW children according to the type of teeth. The highest percentage of enamel hypocalcification of deciduous teeth in LBW children is in the first and lateral upper incisors.

Table 2. The incidence of hypoplasia and enamel hypocalcification of deciduous teeth in Normal Birth Weight children according to the type of teeth.

<table>
<thead>
<tr>
<th>Teeth</th>
<th>Hypoplasia</th>
<th>Hypocalcification</th>
<th>Teeth</th>
<th>Hypoplasia</th>
<th>Hypocalcification</th>
</tr>
</thead>
<tbody>
<tr>
<td>51</td>
<td>-</td>
<td>3.80%</td>
<td>61</td>
<td>-</td>
<td>2.80%</td>
</tr>
<tr>
<td>52</td>
<td>-</td>
<td>5.30%</td>
<td>62</td>
<td>-</td>
<td>2.80%</td>
</tr>
<tr>
<td>53</td>
<td>-</td>
<td>0.70%</td>
<td>63</td>
<td>-</td>
<td>2.60%</td>
</tr>
<tr>
<td>54</td>
<td>-</td>
<td>0.50%</td>
<td>64</td>
<td>-</td>
<td>1.70%</td>
</tr>
<tr>
<td>55</td>
<td>-</td>
<td>0.20%</td>
<td>65</td>
<td>-</td>
<td>1.20%</td>
</tr>
<tr>
<td>81</td>
<td>-</td>
<td>0.70%</td>
<td>71</td>
<td>-</td>
<td>0.20%</td>
</tr>
<tr>
<td>82</td>
<td>-</td>
<td>1.50%</td>
<td>72</td>
<td>-</td>
<td>0.20%</td>
</tr>
<tr>
<td>83</td>
<td>-</td>
<td>0.70%</td>
<td>73</td>
<td>-</td>
<td>0.70%</td>
</tr>
<tr>
<td>84</td>
<td>-</td>
<td>0.20%</td>
<td>74</td>
<td>-</td>
<td>0.70%</td>
</tr>
<tr>
<td>85</td>
<td>-</td>
<td>-</td>
<td>75</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
No hypoplasia in Normal Birth Weight children; and hypocalcification less than in LBW children.

Statistical analysis Z test (p<0.01) shows that there were significant differences in incidence of hypocalcification in overall teeth. The incidence in LBW children is higher than Normal birth weight.

Generally there were significant difference in incidence of hypoplasia (p<0.05) in LBW children.

Diagram 2. The incidence of enamel hypoplasia and hypocalcification of deciduous teeth in Normal Birth Weight children according to the type of teeth.

There were significant difference of incidence in hypocalcification and hypoplasia in overall teeth, the incidence of hypocalcification and hypoplasia in LBW is significant higher than NBW children

These results reveal that the upper and lower incisive teeth suffer the most from hypocalcification in LBW children, while Normal Birth Weight children (Table 2) can get a little hypocalcification on 52 (5.3%) and 51 (3.8%). We found in this study that enamel hypocalcification in LBW children was mostly manifested in 51 and 61 (62.75%).

Discussion

This study revealed that the incidence of both enamel hypocalcification and hypoplasia in deciduous teeth is higher in LBW children (p<0.01). The manifestations were more severe in the 30% of those with the early Symmetric type.8,11 70% of the SGA children tested were of the later Asymmetric type with their growth disturbance during the second or third trimester (>16 weeks intrauterine) while in the Symmetric type the disturbance occurred in the beginning of pregnancy (<16 weeks intrauterine).

The highest incidence of hypocalcification was on the anterior teeth of the upper and lower jaws, especially on 51.61 (62.75%), which were formed early 6-7 weeks prenatally and the first calcification at 12 weeks.1,6,13

Hypocalcification manifests frequently as “opaque = not transmitting light”, opacity with white, yellow or brown colors in the form of small or large white dots and might be symmetrically bilateral on the left and right sides caused by systemic factors. It is caused by shortage of organic matrix absorption and restriction of matrix mineralization. It can also be caused by calcium deficiency in children with low blood calcium.4,14,15
Hypoplasia might also have a common cause with subsequent hypocalcification of deciduous teeth, depending on the time the latter occurs i.e. during the second or third trimester of pregnancy. The defects depend on the length and time of the form of the matrix as well as subsequent calcification. Enamel calcification and forming dentin is a sensitive long lasting process.2

Hypoplasia occurs in LBW children, clearly in this study, due to growth restriction in the beginning stages of pregnancy when the tooth is still in the amelogenesis phase (enamel matrix forming). Hypoplasia occurs if the matrix synthesis, of secretion of resorption processes, are restricted, that might disturb the next mineralization and enamel forming. When such disturbances occur for a long period, then the amount of enamel will decrease and be incomplete, or even not formed at all.1617 Such a condition might occur during <16 weeks prenatally, while hypocalcification means a restriction of the enamel caused by the decrease of calcification at the complete matrix, which subsequently starts at 16 weeks prenatally.

Dental hypoplasia and hypocalcification of LBW children was found/occur because general IUGR can cause restricted teeth development.17

Restricted matrix forming in hypoplasia might be caused by systemic disturbance during the secretion stage in the beginning of pregnancy - decreased matrix secretion by ameloblasts, or restricted matrix absorption, or a change in matrix composition that results in decreased matrix formation. Hypoplasia mostly occurs on the upper right and left of the insisive teeth (17.34%). Insisive teeth are the first formed deciduous teeth.

The Normal Birth Weight children in our study had also hypocalcification caused by local or traumatic factors such as the use of endotracheal intubation in neonates with asphyxia or respiratory distress. The enamel defect caused by local factors is unilaterally found.1317

A longitudinal study of very low birth weight children showed hypocalcification. - 50% of the second deciduous molar, 40% first deciduous molar and 30% deciduous caninus, due to prenatal conditions and birth trauma.18 Another study shows enamel defect higher in LBW children (in very low birth weight is higher than in low birth weight).19

The high incidence of hypocalcification in LBW children in this study revealed that there was only mild enamel defect of deciduous teeth, as also shown by the low incidence of hypoplasia which is a more severe defect than hypocalcification. This might be that the most LBW infants in this study were live births without severe complication. Low weight means that there is mal-development due to mal-nutrition in this period.

A study by Laskaris (2000) found that in LBW children the incidence of enamel defect of deciduous teeth was about 20-33% - it was 23.08% in our study; nearly the same as in other countries. Enamel defect, caused by systemic factors, usually manifests bilaterally and that caused by local factors manifests unilaterally. The enamel defect of deciduous teeth in LBW children could also be caused by such local factors as trauma, which can restrict/obstruct the development of ameloblasts and result in local defect.420 Systemic factors such as nutritional deficiency and prenatal or postnatal infection will cause more bilateral defects. Normal Birth Weight children get enamel defect less than LBW children.20 It is established that prenatal development depends equally on: the genetic condition of the child (50%), and the mother’s intrauterine and genetic condition (50%).21

Normal birth weight children have no hypoplasia, LBW children who had no defects might be due to that, for them, ‘growth restriction’ occurred when the calcification of anterior teeth was nearly completed.

Calcification of the anterior teeth is completed while the posterior teeth are still in the calcification process.16 So, by the last period of pregnancy, LBW and Asymmetric SGA might be seen to result in hypocalcification; while the restriction that occurs at the end of the third trimester (40 weeks) would not restrict the anterior teeth because the calcification is already complete, but it might affect the caninus and posterior teeth that are still in the calcification process.

Conclusion

All of the deciduous teeth in LBW children might have enamel hypoplasia or hypocalcification This is mostly on the first incisors of the upper and lower jaws.

REFERENCES

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