

Research Report

Enamel defect of deciduous teeth in small gestational age children

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ABSTRACT

Background: Enamel defect could be caused by genetic and environmental factors in prenatal period. Meanwhile, prenatal malnutrition could also cause small gestational age (SGA). Small Gestational Age is the term used for a neonatal baby with birth-weight below the -2SD normal value or 10th percentile on the intrauterine Lubchenco curve. This condition is due to intra-uterine growth restriction, and eventually ends up with several developmental defects of organs, including teeth. In fact, deciduous tooth development has a critical phase within this development period. **Purpose:** The aim of this study is not only to find out the incidence of enamel defect in SGA children, but also to know the percentage of SGA risk factor to develop enamel defect. **Method:** This was an epidemiology research with consecutive admission technique. It consisted of 153 SGA children aged 9–48 months. Next, the Ponderal index was used to assign SGA types, symmetrical or asymmetrical one-in this study 59 and 94 respectively. On the other hand, three hundred and ninety Appropriate for Gestational Age (AGA) children aged 4–48 months were also included in the study as a control group. Enamel defect then was determined by intraoral examination, classified into hypoplasia and hypocalcifications. Chi-square test was finally used to determine the relative risk ratio between the SGA and the control AGA children. **Result:** The result of this research showed that incidence of enamel defect in SGA children was 86.92%, meanwhile, that in AGA children was 23.08%, 66.00% of which were commonly suffered from hypocalcification. With $p < 0.05$ it is also known that SGA children has the risk of enamel defect with hypocalcification, about 79% higher than AGA children. **Conclusion:** It could be concluded that 79% of SGA children had the risk of deciduous tooth enamel defect with hypocalcification as the most.

Key words: Enamel defect, small gestational age, intrauterine growth restriction

ABSTRAK

Latar belakang: Defek email dapat terjadi karena faktor genetik dan lingkungan sistemik yang terjadi saat prenatal. Adanya malnutrisi prenatal dapat mengakibatkan kelahiran bayi dengan kecil masa kehamilan. Kecil masa kehamilan (KMK) adalah bayi dengan berat badan lahir di bawah -2SD nilai normal atau dibawah persentil 10 kurva pertumbuhan intra uterin Lubchenco. Kondisi ini terjadi sebagai akibat intra uterine growth restriction (IUGR) yang mengakibatkan defek perkembangan organ tubuh, termasuk gigi sulung karena masa kritis pertumbuhan dan perkembangan gigi sulung terjadi pada periode prenatal. **Tujuan:** Tujuan penelitian ini adalah untuk mengetahui insidensi defek email gigi sulung pada anak KMK dan mengetahui besarnya risiko KMK untuk terjadinya defek email pada gigi sulungnya. **Metode:** Disain penelitian adalah epidemiologi dengan consecutive admission. Sample terdiri dari 153 anak KMK berusia 9–48 bulan. Diperoleh 59 tipe simetri dan 94 tipe asimetri dengan indeks Ponderal. Sebagai kontrol diperiksa 390 anak Sesuai masa kehamilan (SMK) berusia 4–48 bulan. Pemeriksaan intra oral dilakukan untuk melihat ada tidaknya defek. Tipe defek adalah hipoplasia dan hipokalsifikasi. Uji Chi-kuadrat digunakan untuk menguji risiko relatif defek email pada anak KMK dan SMK. **Hasil:** Hasil penelitian menunjukkan insidensi defek email pada anak KMK sebesar 86,92% dan pada anak dengan sesuai masa kehamilan (SMK) sebesar 23,08%, dengan jumlah defek terbanyak adalah hipokalsifikasi sebanyak 66,60%. Dengan $p < 0,05$ anak KMK berisiko 79% untuk mengalami defek email gigi sulung. **Kesimpulan:** Dari penelitian ini disimpulkan bahwa anak KMK berisiko 79% mengalami defek email gigi sulung dengan tipe defek terbanyak adalah hipokalsifikasi.

Kata kunci: Defek email, kecil masa kehamilan, intra uterine growth restriction

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INTRODUCTION

Enamel is a structure that cannot be remodeled. It means that if there is any defect occurred on the enamel, it will be considered as a permanent. Genetic and environmental factors, either systemic or local, can actually cause enamel defect at the stages of histodifferentiation, morphodifferentiation, apposition, and classification during the first, second, or third trimester of prenatal period with hypoplasia or hypocalcification depended on the time the defect occurred.^{1–6} Enamel defect caused by systemic factor, usually effect entire teeth, meanwhile the one caused by local factor does not effect entire, but unilaterally. Meanwhile, environmental factor in prenatal period disrupting the growth and development of deciduous teeth is intra uterine growth restriction (IUGR) causing small for gestational age (SGA) baby. The defect then can easily cause caries because of the accumulation of plaque. If this defect occurred on deciduous teeth is not promptly treated, it might cause early teeth extracted soon. This condition then affects their chewing function and aesthetics, so later it can cause malnutrition that can disrupt not only the growth and development of jaws, but also the development of psychology and health in general, causing the disruption of later growth and development processes entirely.^{2,7}

SGA babies, furthermore, are those whose birth weight was below the -2 SD normal value or 10th percentile on the intra-uterine Lubchenco curve, and those whose birth are premature (<37 weeks of pregnancy), normal, or even mature (more than its appropriate months of pregnancy).^{8–11} SGA is actually caused by disturbances that occurred during the development of intrauterine growth restriction (IUGR), which is the restriction of prenatal development in wombs.^{12–14}

There are two types of SGA, symmetric type with disturbances occurred at the beginning of the first trimester of pregnancy and asymmetric type with problems occurred at the second or third trimester.^{8–12,15} The recorded incidence of SGA babies in Dr. Hasan Sadikin Hospital in 2005, were about 7.6–10%, meanwhile in USA it was known about 3–10% of all births.^{16,17}

SGA is actually caused by intrauterine growth restriction (IUGR) that can be caused by maternal factors during pregnancy, placenta.² Maternal factors involves the mothers' age above 35 years old or at teenage period; the physical appearance of mothers that were short and thin; the none or slow increasing of mother's weight during the third trimester can cause malnutrition. Other problems are vascular disease, severe infection during pregnancy, erythematous lupus syndrome, antiphospholipid syndrome, anemia, severity, lack of health service during pregnancy, nulliparity, smoking habit, alcohol consuming, cocaine consuming, living in plateaus, and low social-economy status. Furthermore, there are prenatal factors such as genetic abnormality and chromosome abnormality, abnormality of infant's placenta and tumor.^{8–12,15} Besides that, there are also defects in the development of many

organs, including the development of teeth that can be caused by IUGR since prenatal period is considered as critical period for the development of deciduous teeth.^{1–4}

In addition, previous studies relating enamel defect with low birth weight babies (LBWB) and premature birth show that children with premature birth and LBWB have higher risk of oral abnormality like enamel hypoplasia, hypocalcification, dental discoloration, abnormal dental structure, palatal groove, and delayed dental eruption.^{1,18–19} The prevalence of enamel defect in deciduous teeth, about 20–100%, even is suffered by children with the history of premature birth and LBWB.^{1–7,20–28}

Besides the retardation of deciduous tooth eruption, SGA children also suffer enamel defect.⁵ It indicates that the growth of deciduous teeth in SGA children are disrupted. Therefore, this study was aimed to analyze the incidence and risk of enamel defect in SGA children.

MATERIAL AND METHOD

The subject of this study were 9–48 months-old children born in Dr. Hasan Sadikin Hospital Bandung with the history of small for gestational age (SGA). On the other side, as control group were 4–48 months appropriate for gestational age (AGA) children, but not suffering caries. It means that the age range in SGA subjects is different from that in AGA subjects since unlike AGA children, SGA children suffer delayed eruption.¹⁵

The inclusion criteria, are the children must be 9–48 months old classified as SGA subjects, and must be 4–48 months old classified as AGA children; that the data of mothers and children must be completed; and that children must have abnormal genetics or syndrome as exclusion criterion. Enamel defect then is determined by whether there is hypoplasia or hypocalcification or not. Meanwhile, types of SGA were determined by measuring the Ponderal index with the following formulation:

$$\sqrt[3]{\frac{\text{Birth-weight} \times 100}{\text{Birth-height}}}$$

Intraoral examination then was conducted through several stages: at first, the inform consent was fulfilled; secondly, dental examination was conducted with enough lighting like lamp mirror, and then teeth were cleaned and dried with cotton. Hypoplasia actually can be determined if pit and fissure can be seen, and if there is also partially lost enamel. Meanwhile, hypocalcification actually can be determined by examining whether enamel can be penetrated by light or not, oral examinations is done three times with 3 month interval; thirdly, next structured interview is conducted by matching with the birth history; fourthly, the obtained data then was tabulated into dummy table. Chi-square test was finally used to analyze the difference of risk in SGA children and in AGA ones.

RESULT

The number of SGA children as patients of Dr. Hasan Sadikin Hospital in Bandung were about 184 children, 13 of whom were dead and 18 were not identified for the address, so only 153 of them were listed as subject of this study, 94 of whom were classified into asymmetric SGA, while 59 children were classified in symmetric SGA at the age 9–48 months. On the other side, 390 AGA children at the age of 4–48 months were listed as the control group.

The incidence of enamel defect in SGA children is about 86.92%, higher than that in AGA children (23.08%), meanwhile that of non enamel defect in SGA children is about 13.07%. Similarly, the incidence of enamel defect in SGA children (86.92%) is higher than that in AGA children (23.08%) since those SGA children have suffered intra-uterine growth restriction (IUGR) that eventually cause several developmental defects of teeth, like enamel defect (Figure 1).

Moreover, shows that the highest incidence of enamel defect types in SGA children is hypocalcification (66.60%), meanwhile hypoplasia is about 4.60%, and non defect is about 13.10%. On the other side, in AGA children hypocalcification is only about 23.10%, non defect is about 76.90%, and hypoplasia is 0% (Figure 2). Thus, it indicates that the hypocalcification incidence of enamel defect in AGA children (23.10%) is lower than that in SGA children since hypocalcification in AGA children is caused only by local factors. Meanwhile, that the higher incidence

of hypocalcification in SGA children (66.60%), and the lower of hypoplasia (4.60%) shows that the enamel defect suffered by those SGA children is not severe because of the lower incidence of hypoplasia, considered as the most severe type of enamel defect, only about 4.5%.

Relative risk ratio (RRR) in SGA children is about 3.79 indicating that the risk of enamel defect in SGA children is about 3.79 times high or about 79% (with the formulation $P = \frac{R}{1+R}$, so it becomes $P = \frac{3.79}{1+3.79} = 0.7912 = 79\%$). Then, using chi square test, it is found out that $p < 0.001$ indicating that SGA children has bigger risk of enamel defect.

In addition, That the incidence of symmetric SGA is about 100%, meanwhile that of asymmetric SGA is about 78.72%, and that of non enamel defect is 0%. The reason for all of those symmetric SGA subjects suffer enamel defect is because those symmetric subjects suffer the problem earlier, at the beginning of the first trimester, than those asymmetric ones at the second or third trimester (Figure 3). Similarly, it can be seen also at table 2 at which the risk of enamel defect in those symmetric subjects is higher than in those asymmetric ones.

The biggest incidence of enamel defect types in asymmetric SGA children is hypocalcification. The reason why hypocalcification occurs more commonly in asymmetric subjects is because the number of asymmetric subjects is higher than that of symmetric ones. Meanwhile, there are no asymmetric SGA children suffering hypoplasia.

Table 1. The relative risk of enamel defect in SGA Children compared with that in AGA children

Type of Subject	Enamel Defect				Total	
	Defect		Non Defect		n	%
	N	%	N	%		
SGA	133	86.93	20	13.07	153	100,00
AGA	90	23.08	300	76.92	390	100,00
Total	224		319		543	

$$RR: \frac{133}{153} + \frac{90}{390} = 0.87 + 0.23 = 3.79; \chi^2: 185.11; p < 0.001$$

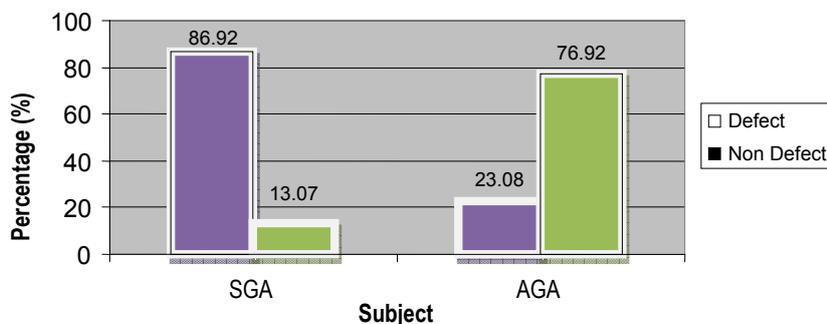


Figure 1. Incidence of enamel defect in SGA and AGA children.

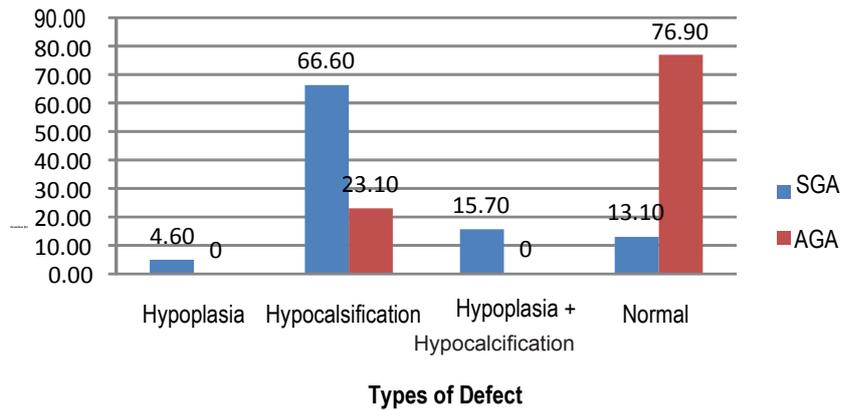


Figure 2. Incidence of enamel defect in SGA and AGA children based on types of defect (hypoplasia, hypocalcification).

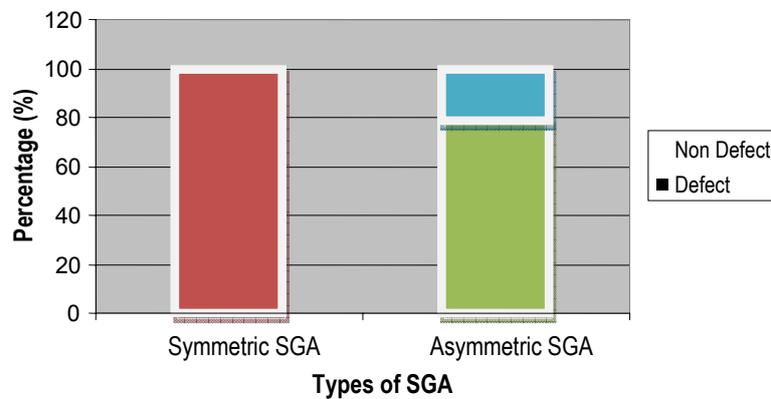


Figure 3. Incidence of enamel defect based on types of SGA.

Table 2. The relative risks of enamel defect in symmetric and asymmetric SGA children compared with those in AGA children

SGA Types	RR	P
Symmetric	4.35	<0.001
Asymmetric	3.39	<0.001

The risk of enamel defect in symmetric SGA children is about 4.35 times high or about 81% (4.35:5.35), meanwhile in asymmetric SGA children is about 3.39 times high or about 77% (3.39:4.39) (Table 2). It means that symmetric type has higher risk than asymmetric one, about 4.35 times as high as the other one.

DISCUSSION

The incidence of enamel defect in SGA children is higher than that in AGA children since SGA children have problems with the growth and development of their deciduous teeth, one of which is IUGR. Enamel defect actually consists of hypoplasia and hypocalcification. Nevertheless, hypocalcification is the most commonly suffered defect. The reason is because the majority of

children studied in this study are classified into asymmetric SGA (61.44%) without suffering severe complication. However, those SGA children can actually suffer enamel defect, which is usually local enamel defect caused by local factors like trauma. Trauma involves stressing of a small group of ameloblast, so the growth of teeth is disrupted, and furthers it causes local defects.¹⁰ Besides, trauma can also be caused by the use of endotracheal intubation tools in neonatal babies with asphyxia, which is distress of breathing, because of birth delivery factors. Nevertheless, manifestation of enamel defect caused by local factors does not affect the teeth entirely, only unilaterally. Unlike local factors, systemic factors, such as prenatal malnutrition, prenatal or postnatal infection, involve the higher number of ameloblast, so the defects do not affect the teeth locally, but entirely and bilaterally.^{21,29,30} Besides that, prenatal development is also depended on genetic materials of children (50%), and both intrauterine environment and genetics of mothers (50%). Thus, AGA children might still suffer/get enamel defects even in lower severity compared to those in SGA children.³¹

The relative risk ratio in SGA children is 3.79 times as high as that in AGA children. It indicates that the risk of enamel defect in SGA children is 3.79 times higher,

about 79%, than that in AGA children. It means that SGA children have problems with the development of their deciduous teeth more commonly.⁵ This condition is caused by intrauterine growth restriction (IUGR) causing SGA which can stimulate any defects in many organs, like teeth. This condition is also supported by the fact that the critical development of deciduous teeth occurs at some prenatal phases, like histodifferentiation (9–10 weeks), morphodifferentiation (11–12 weeks), apposition, and calcification (12–16 weeks). As a consequence, abnormal dental structure then might occur at the end of Bell stage (<16 weeks) which might later cause any disruption problems during the growth of dental enamel, such as hypoplasia. Besides that, if the disruption occurs at calcification phase (>16 weeks), hypocalcification will occur.²¹

Therefore, based on the fact that the incidence of enamel defect in SGA children with $p < 0.05$ is higher than that in AGA children (Figure 1), RR of SGA children is 3.79 times, and the score of DDE in SGA children > that in AGA children, it indicates that the risk of enamel defect in SGA children is higher than that in AGA children.

In addition, Figure 3 shows that the incidence of enamel defect in asymmetric SGA children is 78.70%, while that in symmetric SGA children is about 100%. The figure also shows that there are 21.28% of asymmetric SGA children who do not suffer enamel defect. It means that the percentage of enamel defect in symmetric SGA children is higher than that in asymmetric ones. The reason is those who were classified into symmetric type were all suffer problems at age above >8 weeks, whereas below 8 weeks it might cause major defect or even death. It can also be seen from the fact that there were no asymmetric SGA children suffering the defect. It might be caused by the fact that symmetric SGA children suffered the defect at the beginning of prenatal period (the first trimester), while asymmetric SGA children suffered it furtherly. Thus, the highest incidence of hypocalcification about 61.44% is in SGA children since it occurred in the middle of the second or third trimester at which the development of deciduous teeth is in the phase of calcification, so any problems at that period can cause hypocalcification.^{3,32,33}

Similarly, Figure 3 shows that the incidence of enamel defect in symmetric SGA children is higher than that in asymmetric SGA ones. Table 2 also indicates that RRR of symmetric SGA children is 4.35 times higher, meanwhile RRR of asymmetric SGA is only about 3.39. It means that symmetric SGA children have higher risk than the asymmetric SGA ones. The reason is because the problems during embryonic period (the first trimester) stimulate worse impacts than those during fetal period (the second and third trimesters).^{27,34} The reason is because during embryonic period (2–8 weeks) fetuses are more sensitive to problems. During this period the proliferation of cells is actually getting more active, indicating that the increasing number of cells is higher than those of the size of cells. But, the problems occurred during this embryonic period could

also decrease the number of cells. Unlike this embryonic period, during fetal period, at the age of 16 weeks until delivery (the second and third trimesters), the sensitivity of fetuses against the problems decreased.³¹ This condition is also supported by the opinions of some experts who stated that symmetric SGA children have more severe defect, than asymmetric SGA ones.⁸⁻¹¹

Moreover, it is also known that non enamel defect can be found only in asymmetric SGA children. This is because when the growth of intrauterine is disrupted at the third trimester, the process of calcification is almost finished, and consequently, enamel defect does not occur.^{12,29}

As a conclusion, firstly, it is found out that SGA children have higher risk of enamel defect, about 79%, than the AGA children. Secondly, it is also found out that symmetric type of SGA has higher risk than asymmetric one. Finally, it is found out that enamel hypocalcification is the most commonly found defect.

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