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Research Report

Enamel defect of primary dentition in SGA children in relation to onset time of intrauterine growth disturbance

Willyanti Soewondo Sjarif
Department of Pediatrics Dentistry
Faculty of Dentistry, Universitas Padjadjaran
Bandung - Indonesia

ABSTRACT

Background: *Prenatal disturbances disturb the development of organs resulting in small for gestational age (SGA) babies and also causes enamel defects in primary teeth. There are disturbances occur in the beginning of pregnancy causing symmetrical SGA, and asymmetrical type of SGA, where the disturbances occur late in pregnancy.* ***Purpose:*** *This research was to determined differences in severity of enamel defect of primary dentition in small for gestational age children based on the time of intrauterine growth restriction.* ***Methods:*** *This was a clinical epidemiological cohort study. The Ponderal index was used to determine SGA type. The subjects were 129 SGA children aged 9-42 months, 82 with asymmetrical SGA and 47 with symmetrical SGA. Two hundred normal birth weight children were the control group. Intra-oral examinations to determine enamel defect used the FDI modification of the Developmental Defect of Enamel score at 3 months intervals. Statistical t-tests were used to test the difference in severity of enamel defect, and chi-square to find out the difference of Relative Risk Ratio (RRR).* ***Results:*** *The results showed that the enamel defect scores of symmetrical SGA were significantly higher than those with asymmetrical SGA. RRR for severe defect was also significantly higher in symmetrical type for anterior and canines.* ***Conclusion:*** *The study suggested that the severity of enamel defect for infants with symmetrical SGA was higher than those with asymmetrical SGA, indicating that the severity of the defect occurs in the beginning of pregnancy is more severe than in the late pregnancy.*

Key words*: Enamel defect, small for gestational age, symmetrical, asymmetrical, intra-uterine growth restriction*

ABSTRAK

Latar belakang: *Adanya gangguan prenatal mengganggu perkembangan organ, mengakibatkan terjadinya bayi lahir dengan kecil masa kehamilan (KMK) dan defek email pada gigi sulung. Terdapat 2 tipe KMK yaitu tipe simetri; gangguan terjadi pada awal kehamilan; dimana lingkar kepala, berat dan panjang lahir lebih rendah dari normal. Tipe asimetri dimana gangguan terjadi saat kehamilan lanjut: panjang dan berat badan lahir lebih rendah dari normal.* ***Tujuan:*** *Penelitian ini bertujuan meneliti perbedaan keparahan defek email gigi sulung pada anak KMK berdasarkan saat terjadinya gangguan hambatan pertumbuhan intrauterin.* ***Metode:*** *Jenis penelitian adalah epidemiologi dengan studi Kohort. Ponderal indeks digunakan untuk menentukan tipe KMK subjek terdiri dari anak KMK usia 9-42 bulan, 82 tipe asimetri dan 47 tipe simetri, 200 anak dengan berat lahir normal sebagai kontrol. Pemeriksaan intra oral dilakukan untuk menentukan skor defek email yaitu dengan menggunakan skoring modifikasi DDE indek dari FDI. Subjek di teliti dengan interval 3 bulan, t-test digunakan untuk menentukan perbedaan defek email pada KMK simetri dan asimetri sedangkan chi square menentukan perbedaan RRR (Rasio Resiko Relatif).* ***Hasil:*** *Hasil menunjukan bahwa skor defek enamel pada KMK simeteri lebih tinggi secara signifikan dibanding pada KMK asimetri dan RRR pada defek berat lebih tinggi pada KMK simetri*

pada gigi anterior dan kaninus. ***Simpulan:*** *Penelitian ini menunjukkan bahwa defek email lebih parah bila terjadi pada awal masa kehamilan (tipe simetri) dibanding bila terjadi pada saat kehamilan lanjut (tipe asimetri).*

Kata kunci*: Defek email, kecil masa kehamilan, simetri, asimetri, hambatan pertumbuhan intrauterin*

Correspondence: Willyanti Soewondo Sjarif, c/o: Kedokteran Gigi Anak, Fakultas Kedokteran Gigi Universitas Padjadjaran. Jl. Sekeloa Selatan 1 Bandung 40132, Indonesia E-mail: willyantir@yahoo.com

INTRODUCTION

Small for gestational age (SGA) describes a newborn infant with birth weight less than normal for its gestational age, to the extent of being under the 10th percentile of the intrauterine growth curve. These kinds of deliveries might cause many problems in the future including morbidity and mortality. In general, SGA neonates are in poor condition and are at high risk for both their short term and long term health. Intra uterine growth restriction (IUGR) occurring in the prenatal period can have an effect on foetal development causing anomaly to several organs.[1-8] The incidence of SGA in the USA is about 3-10% of all deliveries, in Cipto Mangunkusumo Hospital Jakarta (1999) 4.42% and in Hasan Sadikin General Hospital Bandung it was 7.6-10% of all deliveries in 2005.[9-10] The prenatal period is a critical time especially in forming primary dentition.[11-18] Overall, the incidence of enamel defect in primary dentition is 22-33%.

There are two types of SGA, based on the onset of the restriction of inter-uterine growth i.e. symmetrical SGA: which occur in the first trimester of pregnancy (embryonal phase) and a symmetrical SGA: which occur in the second and third trimester of pregnancy (foetal period).[1-7] Symmetrical SGA infants have disturbances in both brain and physical growth as shown by delayed growth of head circumference, body length and body weight, but the whole body is in good proportion. Asymmetrical SGA infants also have smaller body weight and length but relatively normal head circumference.[1-6] Determination of SGA Type therefore requires an accurate record of gestational age, birth length and head circumference.

In general the clinical manifestation is worse in symmetrical SGA infants than in asymmetrical ones. Avery and Chicago reported that anomalies of dentition which occurred during the embryonal period (symmetrical SGA infants) is worse than in foetal period (asymmetrical SGA infants).[1-8] Overall, 71% of enamel defect in primary dentition are caused by prenatal systemic factors, the effect of developmental disturbances occurring at the beginning of dental forming and calcification during the first, second or third trimester of pregnancy, and manifesting as hypoplasia or hypocalcification. The enamel defect of primary dentition becomes a problem because it is unregenerate, so the effect is permanent.[11-18] Enamel defect might also worsen enamel quality and cause easier accumulation of plaque that trigger caries occurence. Untreated caries can then cause abscesses, resulting in premature loss of primary dentition. One of the predispositions to caries is the anomaly of enamel structure that is involved with prenatal developmental growth disturbances. The birth condition known as SGA is caused by intra uterine growth restriction (IUGR).[1-8] In Indonesia and other developing countries IUGR is still a main health problem,with the highest mortality rate

The cause of IUGR might be placental, foetal and maternal characteristics such as the age of mother older than 35 years old or young mother, short and thin stature, or low increase of body weight during the third trimester of pregnancy. It might also caused by vascular disease during pregnancy such as hypertension and preeclampsia, or caused by severe infectious disease, lupus erythematosus, antiphospholipid syndrome, anemia, malignancy, nuliparity, smoking, alcohol, cocaine, and low socioeconomic status.[1-5] Family with low socio-economic conditions can result in bad nutrition in pregnant mothers that affects inter-uterine health. Klaus and Fanaroff, found that many poor pregnant women had bad nutritional status and gave birth to babies with low birth weight (less than 2500 g), with more than 60% being SGA.[1-5] The other factors are foetal characteristics, such as type of pregnancy (single/ multiple pregnancy), congenital anomalies (genetic and chromosomal), and placental factors (placental anomaly, infark, tumor and placentitis).[1-8] Several studies on enamel defect suggest that genetic and environmental factors might be implicated.[11-17] Even though a genetic factor could be the cause in some cases of the enamel defect, prevention is not an option, but early detection and intervention in all cases might facilitate managing the anomaly and minimizing its severity and subsequent effects.[18-19]

The goal of the study was to determine the differences in severity of enamel defect in the primary dentition of SGA infants, based on the onset of intrauterine growth disturbance. It is important to predict the severity of the defect, to determine prognosis and treatment planning.

MATERIALS AND METHODS

The subjects was of this study were 129 SGA infants, the age range 9 to 42 months, born in Hasan Sadikin General Hospital, Universitas Padjadjaran Bandung Indonesia. As group consisted of 200 infants with normal birth weight (appropriate for gestational age- AGA) in range of age 4 to 42 months and caries free was used as control group.

The different of the SGA and AGA youngest subjects was in accordance with a study conducted by Willyanti that

SGA children had delayed eruption of teeth compared to AGA children.[20] Inclusion criteria also required complete data of birth; for mothers and children, and an exclusion criteria was infants with general anomalies (such as genetic anomaly). This was a clinical epidemiological ambispective cohort study, with given sample sizes. After obtaining informed parental consent, and completing physical examination of SGA (and AGA/control) patients, the enamel anomaly or enamel defect and presence of dental caries were determined.

Scoring for hypoplasia and hypocalcification using modified Developmental Defect of Enamel (DDE) of Federation Dental Internationale (FDI).[21] The subjects were examined three times, at one month intervals, to determine whether there were enamel defects on new teeth. The development of dentition was monitored in case there was any defect on the next erupted tooth. Subjects with only 1 or 2 teeth erupted were monitored, and examination on subyects with primary dentition fully erupted.

Small for gestational age (SGA) is defined if the baby a baby was born with birth weight under the 10th percentile of Lubchenco curve of intrauterine growth and development of weight for gestational age. Type of SGA was determined using the Ponderal index.

$$\text{Ponderal Index} = \frac{\sqrt{\text{Birth weight x 100}}}{\text{Birth length}}$$

Type of SGA was defined as symmetric if the Ponderal index scores was 20 to 25 and asymmetric if the Ponderal index scores was either less than 20 or more than 25. Severity of enamel defect of primary dentition was identified as extent of hypoplasia/hypocalcification. Hypoplasia was defined when there was pit, fissures, or cavity in the surface of the enamel while hypocalcification was defined when the teeth were unglistening and not transparent.[11-19] Dental examination was done using a mouth mirror, explorer, and probe with paper lighting. The teeth surface were cleaned, dried using a cotton role, then examined to record any defects on primary dentition.

Enamel hypoplasia/hypocalsification (EHP) score 1 (normal) was determined when the enamel transparent; score 2 (opacity) when the enamel opaque/ white, not transparent, or yellowish/ brownish; score 3 when there were pits and fissures on some of teeth surface; score 4 when there was un-neat vertical fissures; score 5 when there were exact horizontal fissures; and score 6 when most of the enamel missing or teeth were smaller. Scoring used the FDI modification of the Developmental Defect of Enamel (DDE) for enamel hypoplasia/ hypocalcification (EHP) and an index of enamel defect severity (EDS) was determined as follows;[21] Index enamel defect severity (EDS) was determined using the FDI modification of Developmental Defect of Enamel (DDE) as follows:

$$\text{Enamel Defect Score (EDS)} = \frac{\text{EHP x Total dentition with defect x 10}}{\text{Total teeth at risk}}$$

The degree of severity of enamel defects of primary dentition was then classified relative to a statistical cut-off point of a median score of 12 determined from a Kruskal Wallis test (Normal 0; mild/light 1-12; and severe >12). Difference of enamel defect severity of primary dentition based on onset of intrauterine developmental growth disturbance was compared using a t-test. Chi-square was used to determined differences in incidence of enamel defect based by type of SGA and to differentiate enamel defect risk rates based on stage of intrauterine developmental growth disturbance. Risk ratios of symmetric against asymmetric SGA based on severity of defect, on anterior, canine and posterior teeth were separately tested by t-test.

RESULTS

Small for gestational age (SGA) infants had more severe enamel defect of primary dentition (EDS mean: 12.27) than AGA control infants (EDS mean: 0.39) (Table 1). Type of SGA related to incidence of enamel defect. Enamel defect affects 100% of infants with symmetric SGA and in asymmetric SGA still high but less at 75.6% (Table 2a), while the mean score on symmetric SGA infants was significantly higher (15.29) than for asymmetric SGA infants (10.38). It indicated that the enamel defect of primary dentition is more severe in the symmetric SGA compared with the asymmetric SGA condition (Table 2a).

Symmetric SGA infants were at significantly higher risk of both light and severe enamel defects on their anterior teeth than asymmetric SGA infants -3.74 times at risk of light enamel defect and 7.11 times at risk of having severe defect (Table 3). Symmetric SGA infants were at significantly higher risk of both light and severe enamel defects on the caninus teeth than asymmetric SGA infants -2.19 times at risk to have light defects and 2.96 times at risk to have severe defects (Table 4). Symmetric SGA infants were at higher risk of both light and severe enamel defects on the posterior teeth than asymmetric SGA infants

Table 1. EDS Score means of SGA and AGA infants

Subject	EDS mean score	95%	Conf. Interval
SGA	12.27	10.89	13.72
AGA/control	0.39	0.295	0.475

t= 26.10, p < 0.001

Table 2a. Incidence of enamel defect based on type of SGA

Type of subject	Enamel defect		Total
	Defect	No Defect	
SGA Symmetric	47 (100%)	-	47 (100%)
SGA Asymmetric	62 (75.6%)	20 (24.4%)	82 (100%)
Total	109 (84.49%)	20 (15.51%)	129

(although this was less significantly so for light defects) -1.42 times at risk to have light defects and 1.93 times at risk to have severe enamel defects (Table 5).

DISCUSSION

The study showed that enamel defect score (EDS), based on the FDI modification of the Developmental Defect of Enamel (DDE) score for enamel hypoplasia/ hypocalcification (EHP), was higher in SGA compared to AGA infants, indicating that the enamel defect inprimary dentition is more severe for SGA than for normal AGA infants. This is because the IUGR causing SGA in infants occursin the early foetal prenatal period, a critical period of primary dentition development.[8] IUGR at that stage causes disturbances/anomalies of the organs, including primary dentition.[22,23] The results showed that enamel defect of primary dentition in SGA might affect several or even all types of teeth, bilaterally An interaction of genetic and environmental factors might effect the growth and development of dentition, and local environmental factors may have effect the growth of teeth environmental. Stewart and McDonald state that systemic factors might be the whole cause of enamel defect.[16,24]

Our study has also shown that the enamel defect anomalies were more severe in SGA infants. Also the results indicate variation in severity of enamel defect according to the type of SGA, in EDS/DDE score was higher (more severe defect) in the primary dentition of infants with symmetrical type of SGA. It seems that this is because for them the anomaly occurs earlier, i.e. during the first trimester or embryonic phase critical for dentition, and for the asymmetric type it occurs later at the end of the second or third trimester or foetal phase.

Table 2b. EDS mean score based on type of SGA

Type of subject	N	EDS mean	Std. Err.	Conf. Interval 95%	
Asymmetric SGA	82	10.38	0.79	8.86	11.98
Symmetric SGA	47	15.29	1.29	12.75	17.92
Total	129	12.32	0.72	10.90	13.73
Difference		-4.91	1.42	-7.73	-2.09

Note: Difference = mean (asymmetric) – mean (symmetric); t=3.4457; p<0.001

Table 3. Anterior teeth - relative risk ratio of symmetric against asymmetric SGA based on light and severe defect

	Type of SGA		Total	RRR	95% CI	X^2	p
	Exposed	Unexposed					
Light							
Symmetrical	30	59	89	3.74	0.98-14.35	5.53	0.019
Asymmetrical	2	17	19				
Total	32	76	108				
Severe							
Symmetrical	21	10	31	7.11	1.86-27.18	17.20	0.001
Asymmetrical	2	19	21				
Total	23	29	52				

Table 4. Caninus teeth - relative risk ratio of symmetric against asymmetric SGA based on lightand severe defect

	Type of SGA		Total	RRR	95% CI	X^2	p
	Exposed	Unexposed					
Light							
Symmetrical	29	18	47	2.19	1.21-3.99	8.60	0.003
Asymmetrical	9	23	32				
Total	38	41	79				
Severe							
Symmetrical	5	1	6	2.96	1.53-5.73	6.62	0.010
Asymmetrical	9	23	32				
Total	14	24	38				

Table 5. Posterior teeth - relative risk ratio of symmetric against asymmetric SGA based on the light and severe defect

	Type of SGA		Total	RRR	95% CI	X^2	P
	Exposed	Unexposed					
Light							
Symmetric	16	14	30	1.42	0.81 – 1,91	0.87	0.351
Asymmetric	28	37	65				
Total	44	51	95				
Severe							
Symmetric	5	1	6	1.93	1.23-3.05	3.58	0.058
Asymmetric	28	37	65				
Total	33	38	71				

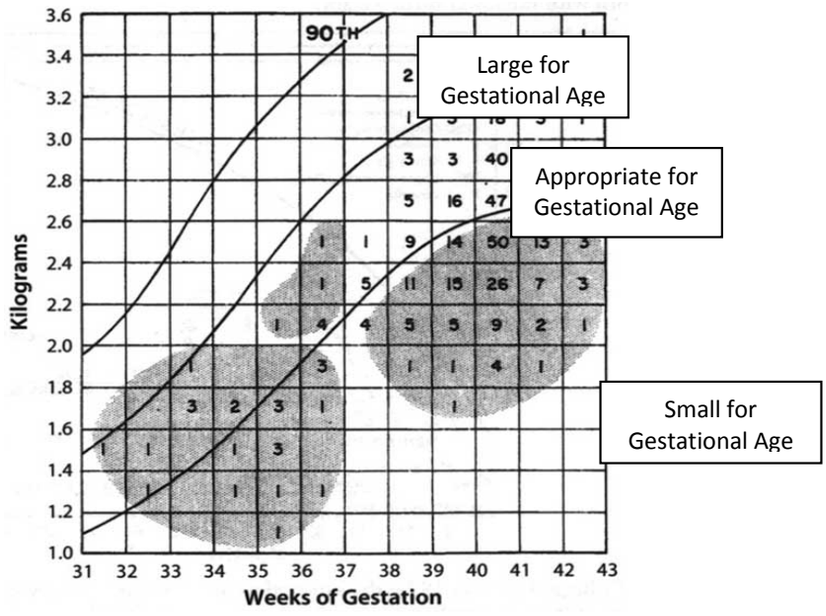

Figure 1. Intrauterine deivelopment curve.[1,5,6]

It was also seen in this study there was no hypoplasiain the asymmetrical type, while in the symmetrical type there were both hypoplasia and hypocalcification. Hypocalcification in the asymmetrical type is a milder anomaly of the structure compared with hypoplasia. It is a disturbance sof enamel matrix calcification that occurs after the 16th week of pregnancy. Hypoplasia is an anomaly caused by disturbance of matrix forming by ameloblast-disturbance of matrix synthesis or the resorbtion that causes disturbance on the next mineralization. Along lasting disturbance might result in incomplete enamel or no enamel forming at all.[25-26] Accordingly the asymmetrical type shows better clinical signs than symmetrical type.[1-4]

As a result of IUGR during embryonal phase or foetal phase that might disturb dentition development, SGA infants may have hypoplasia and hypocalcification. The manifestation of the anomaly is based on the time of disturbance and the organs' development activities.[11,14-17,24-26] In our study, the symmetrical type SGA had more severe enamel defect than asymmetrical type because the defect occured during the embryonic phasecritical phase for organogenesis.[29,30] Some of the SGA (24.4%) had no anomalies because the calcification process had completed when the disturbance/anomaly occured (Table 2a).

The results showed that the incidence of enamel defect of primary dentition in symmetrical SGA was higher than in asymmetrical SGA and the defect was more severe too. This is in accordance with statements in previous studies that anomalies which occur during the embryonic period (1st trimester) will be more severe than those occurring later during the foetal period (middle of 2nd and 3rd trimester) because the foetus is in highly sensitive condition-cell proliferation and highly active with the cell number increase more than the cell size. Disturbance during the embryonic period may therefore decrease the amount of cells. During the foetal period (2nd and 3rd trimester) there is a decrease of the sensitivity from IUGR disturbance – this is in accordance with Taeusch who reports that symmetrical SGA infants had more severe anomalies than infants with asymmetrical SGA.[27-29]

Enamel is the hardest tissue on which calcification might occur, consisting of crystals with 96% inorganic material, and only 4% water and organic material. Enamel is formed by an extracellular matrix from the synthetic and protein secretion by ameloblasts. Enamel, which is only formed once, is different to cartilage or bone and will not regenerate and resorb.[8,25,30] Enamel defect might occur in the amelogenesis period, i.e. matrix aposition process and mineralization since the beginning of the 4th month of pregnancy. Aposition is the end stage of morphodifferentiation. Matrix forming consists of secretion and maturation, enamel matrix starts as an occlusal part of dentition. The first calcification begins on the 4th month prenatal up to the antenatal period. During amelogenesis the ameloblast is highly sensitive to what might disturb its activities and any disturbance might result in enamel defect in the form of hypoplasia and also hypocalcification-hypoplasia being shortage of enamel matrix, and hypocalcification being when the enamel matrix is sufficient but there is shortage of calcification.

As mentioned, these disturbances might be caused by genetic or environmental factors during the perinatal or postnatal period.[11-17,24] Prenatal environmental factors that might be the cause of enamel defect of the teeth are maternal factors such as severe infectious disease at the beginning of pregnancy, chronic infection, long lasting malnutrition, premature and low birth weight. Mother's diseases during pregnancy such as maternal diabetes, hypertension, maternal alcoholism, torch, high fever at the beginning of pregnancy, might also cause hypoplasia,[16] and might also result in SGA.

The possibility (Relative Risk Ratio: RRR) of severe defect (>12) of the anterior and caninus teeth was significantly higher in symmetrical compared with asymmetrical SGA (Tables 3, 4, 5). The severe defect on posterior teeth was less significant in symmetrical SGA because the posterior teeth, especially second primary molar are the last teeth formed.

Previous studies had not looked at disturbances in prenatal growth.[12-3,18-9,21] It was examined in this study and concluded that the severity of enamel structure anomaly of primary dentition was higher in infants with SGA, and also, for those with symmetrical SGA it is more severe than for those with asymmetrical SGA. It means that the enamel defect/anomalies that occur in the beginning of pregnancy (embryonic phase) are more severe than those that occur later (during the foetal phase). This information will be important for assisting predictive prognosis and treatment planning. It was concluded that the severity of enamel defect which occurs in the beginning of pregnancy was more severe than in late pregnancy.

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