International Journal Dental and Medical Sciences Research (IJDMSR)
ISSN: 2393-073X Volume 3, Issue 1 (Jan- 2019), PP 31-35
www.ijdmsr.com

Frequency Distribution Of Enamel Hypoplasia Of Primary Teethon Down Syndrome Children At Potads Foundation

Pradita Annastia[1], Meirina Gartika[2], Willyanti Soewondo[3]
[1,2,3]*Department of Pediatric Dentistry, Faculty of Dentistry, Universitas Padjadjaran*

**Correspnding Author: Meirina Gartika,*
Phone: +062811204863, address: Jl. Sekeloa Selatan no. 1 Bandung 40132 Indonesia

ABSTRACT:- Enamel hypoplasia is one of the abnormalities of the enamel structure caused by disturbances during the formation of enamel matrix. Down Syndrome is a chromosomal disorder caused by the extrachromosome 21. The purpose of this study was to find out the frequency distribution of the primary/deciduous teeth enamel hypoplasia on Down Syndrome children at POTADS foundation. This was a descriptive survey technique study. The subject were 70 Down Syndrome children collected through total sampling. Enamel hypoplasia was determined by using mouth mirror and adequate lighting to examine/to look at the enamel surfaces of the teeth which show hole(s), pit(s), horizontal or vertical grooves until partial loss of the enamel. The results showed the frequency distribution of primary teeth enamel hypoplasia on Down Syndrome children at POTADS foundation were as follows: based on age were mostly on 7 years old children (5.71%); based on the type of teeth were mostly primary incisive lateral (12.85%), and based on sex/gender were mostly on females/girls (11.42%). It was concluded thar the frequency distribution of primary teeth enamel hypoplasia on Down Syndrome children at POTADS foundation was 21.42%.

Key words:Enamel hypoplasia, Down Syndrome

I. INTRODUCTION

Down Syndrome (DS) is the most frequently found chromosomal abnormalities all over the world. The estimated incidence was about 8 million peoples.A study by Mikyong Shin[1] revealed the prevalence of DS during the period 1979-2003 increased to 31.1%, for example 9.0% up to 11.8% of 10,000 neonates around 10 parts of the United States. In 2002, the prevalence on children and adolescents (0-19 years) were 10.3% of 10,000 live births, that made Down Syndrome the most chromosomal abnormality in people. According to the World Health Organization (WHO) the estimated incidence was 1 of 1,000 up to 1 of 1,100 births. At present, the total of DS patients are 8 millions worldwide. About 3000 to 5000 babies were born with DS every year. Data from RISKESDAS 2013 showed 300,000 children in Indonesia had Down Syndrome.[2]

Down Syndrome mightoccur on all typesof race. The growth of a child with DS might show abnormality, slow and barriers. Oral manifestation of a DS child are mostly smaller sized mouth, macroglossia, and teeth anomalies such as mirodontia, conical shape, hypodontia, taurodontia, and enamel defects such as enamel hypoplasia or enamel hypocalcification.[3]

Enamel hypoplasia is one of the abnormalities that is caused by disorders at the time of enamel matrix forming that makes imperfect surface of the enamel.[4] The abnormalities might be caused by genetical factors, systemic environment, and local factors. The systemic environment are trauma at birth, chemical, chromosomal abnormality, infection, malnutrition, metabolic diseases and neurologic disorders. Local factors are trauma or infection on the teeth, and radiation.[5]

Aminabadi[6] stated the distribution of enamel defects of 121 normal children aged 3-5 years were 55.37% had several enamel disorders and 22.31% had enamel hypoplasia. A study by Amalliah[7] at 16 subdistricts of Serpong showed at least 630 normal children in each group revealed the prevalence of enamel hypoplasia on 3-5 year-old children were 54%, and 26.2% on 11-13 year-old children.

II. METHODS

This was a survey technique study. The population/subjects were all DS children at POTADS foundation Bandung. The sampels/subjects were collected by total sampling.

III. RESULTS

The subjects were 70 children consisted of 39 males/boys and 31 females/girls. The distribution of the subjects based on age and sex/gender is shown in Diagram 1.

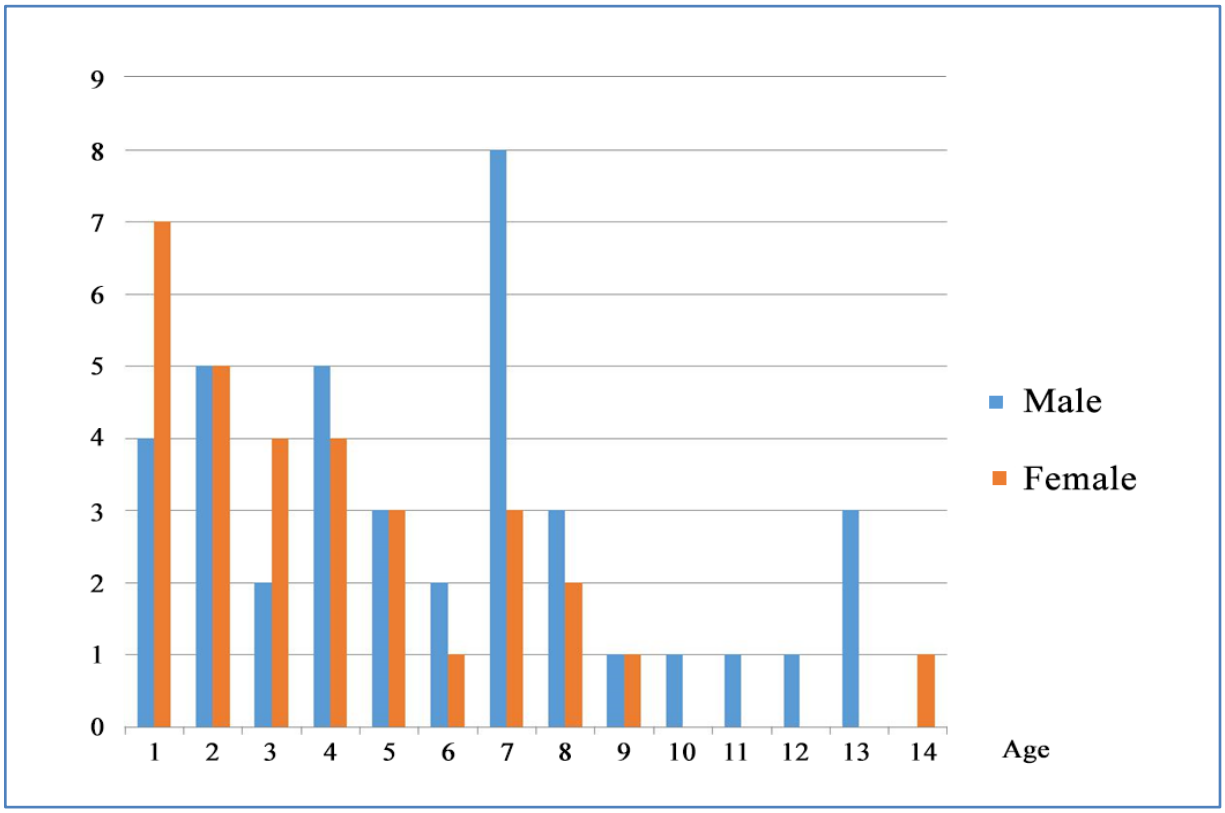

Diagram 1. Characteristics of DS children at POTADS foundation based on age and sex.

Diagram 1 shows that of 70 DS children at POTADS foundation, 55.71% were males/boys and 44.28% females/girls. Most of the children (11) were 7-year-old, 8 (11.42%) of which were males/boys. Clinical examination of the primary teeth enamel hypoplasia was done on DS children aged 1-14 years. Diagram 2 shows the total of children with enamel hypoplasia based on the age.

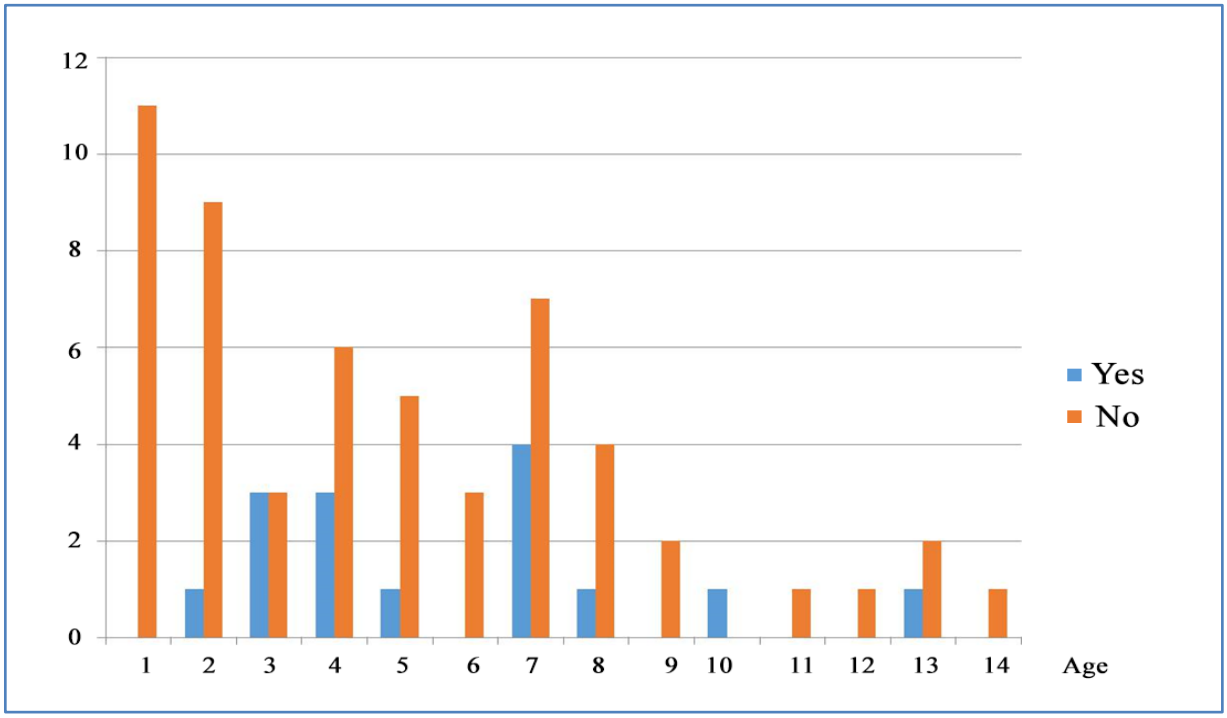

Diagram 2. Frequency distribution of enamel hypoplasia on DS children based on age

Diagram 2 shows that of the 70 subjects, 4 (5.71%) aged 7 years had enamel hypoplasia. Enamel hypoplasia was found on 15 of 70 DS children (21.42%). Diagram 3 shows the distribution of enamel hypoplasia of the primary teeth.

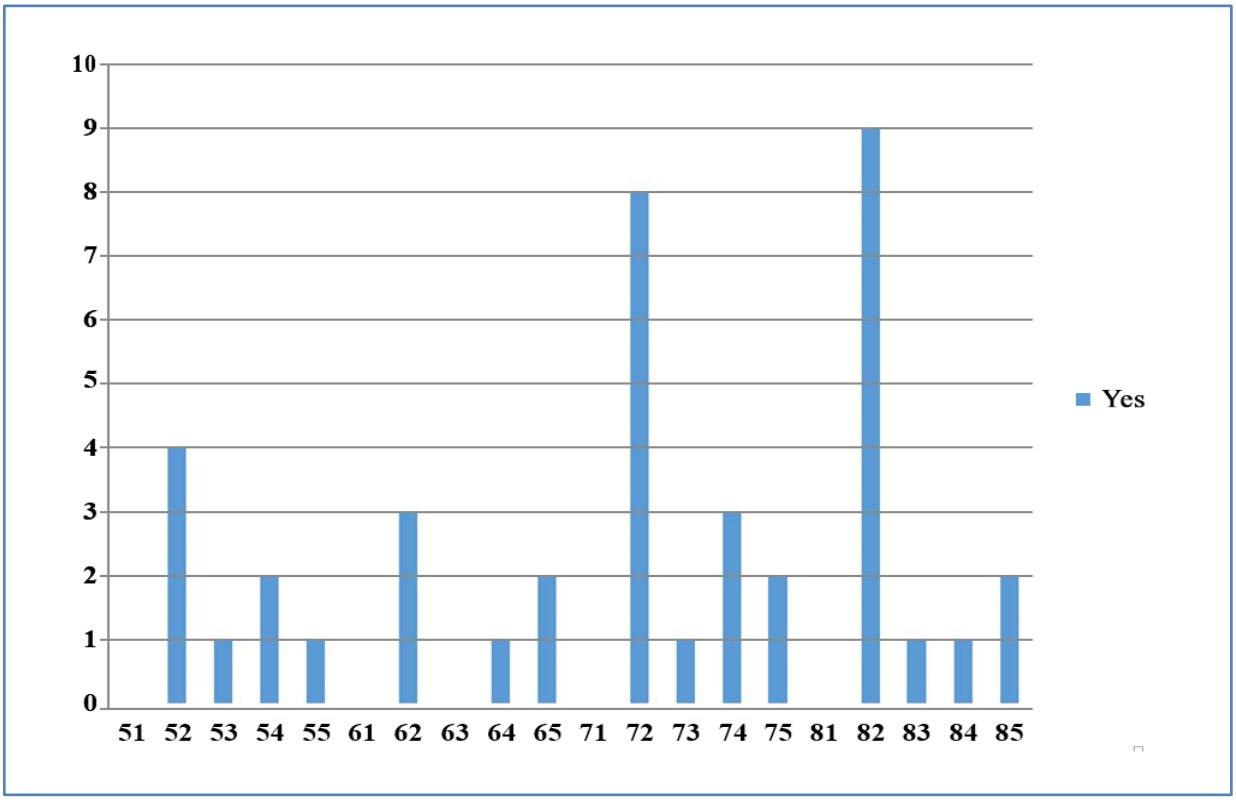

Diagram 3. The frequency distribution of enamel hypoplasia on DS childrenbased on the primary teeth.

Diagram 3 shows that 58.57% enamel hypoplasia occured on the primary teeth. Most of the enamel hypoplasia were found on tooth 82 (9 = 12.85%) and on tooth 72 (8 = 11.43%). Afterwards, all the DS children with enamel hypoplasia at POTADS foundation were grouped based on gender. Diagram 4 shows the frequency distribution of those with enamel hypoplasia based on gender.

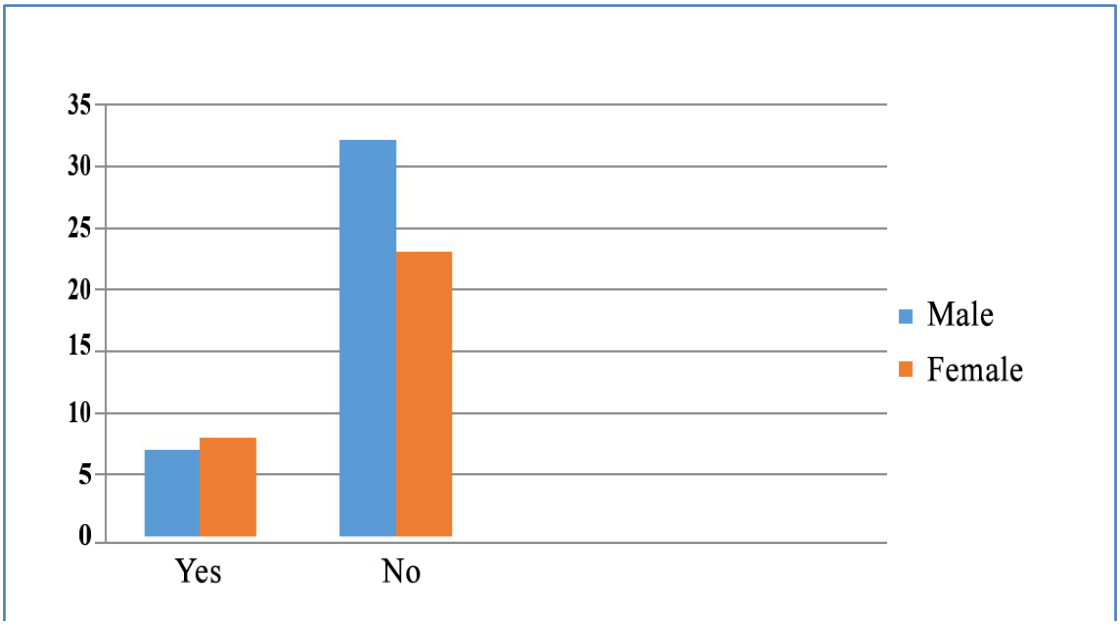

Diagram 4. Frequency distribution of enamel hypoplasia on DS children based on gender.

Diagram 4 shows that 7 male DS children had 10% enamel hypoplasia, and 8females had 11.42% enamel hypoplasia. It means that more female DS children had enamel hypoplasia compared to males.

IV. DISCUSSION

Diagram 1 shows of the 70 DS children, the total of males (55.71%) is higher than females (44.28%).This was similar with the study by Bertoli et al[8]that found of 518 DS children, 282 were males and 236 females; and Kovaleva et al[9] who found 649 males and 531 females. There was a high frequency of chromosomal abnormalities of the oosit and spermatozoa. Men/males might influence the non disjunction that caused aneuploid on the sperm. This might be found on carriers as well as normal chromosomal men/males.[10]The higher number of males with DS is caused by the meiosis disorders that influenced the chromosome 21 and chromosome Y at the time of spermatogenesis.[9] It is evidently found that non disjunction during meiosis could be influenced by the father's age, even with only a slight impact. The father's age is a risk factor in non disjunction occurence.[11]

As seen in Diagram 2, most of the subjects (11) were children aged 7 years. A study by Radhi[12] found that there was no significant difference based on age, but the enamel defects were found higher on DS children compared with general population of children. Aminabadi[6] stated the enamel defect distribution of 121 normal children aged 3-5 years, 22.31% were enamel hypoplasia. Slayton[13] found on 698 normal children aged 4-5 years, 44 (6%) had enamel hypoplasia; 3% had one tooth with enamel hypoplasia; 2% had two teeth with enamel hypoplasia, 1% had three teeth with hypoplasia, <1% had four teeth with hypoplasia, and only one child had more than four teeth with enamel hypoplasia. While Lunardelli[14] found on 431 normal children aged 3-5 years the prevalence of enamel hypoplasia were 11.1%.

Anomalies of teeth structure are oftenly occur on primary as well as permanent teeth, with 5 times higher incidence on DS children compared to general population of children.[12] Tooth forming is called odontogenesis, consists of several phases. The damage of teeth enamel occur during amelogenesis, i.e. at the time of matrix aposition and mineralization process. During this period (amelogenesis), the ameloblast is highly/very sensitive against any disorder that caused by systemic and local factors. This disorder might cause damage of the enamel that is called enamel hypoplasia.[15]

The forming of tooth morphology at early stage of tooth growth and development is influenced by genetical and environmental factors.[16] When the cause of enamel hypoplasia was a genetical factor, it might involve the primary as well as the permanent teeth.[17] An individual with a hereditary disorder such as Down Syndrome might be in a high risk to have tooth disorder. The enamel hypoplasia on DS children could be seen through decreased amount of the enamel matrix of the tooth.[16]

Diagram 3 shows that enamel hypoplasia occurs mostly on the lateral and central incisor teeth. This is in accordance with the study on low birth weight children by Franco et al[18] that revealed enamel hypoplasia on 23 (4.8%) of 480 incisor; 4 (1.1%) of 372 molar, and 2 (0.9%) of 217 caninus. Aine et al[19] in their study on prematurely born children found that enamel hypoplasia oftenly occur on the central and incisor teeth of the upper jaw. On the contrary, Lunardelli[14] stated that on 431 normal children the most often exposed enamel hypoplasia were the second molar of the upper jaw (26.8%) followed by the second molar of the lower jaw (17.6%), the first upper molar tooth (11.9%), the first lower molar tooth (11.6%), and lower caninus tooth (11.6%), upper caninus tooth (10.3%), and upper incissive tooth (9.2%). The last exposed was the lower incissive tooth (1%).

Enamel defect occurs more often on the surface of tooth buccal instead of other surfaces. The impact of growth and development disorders in children might be reflected on the teeth. Franco et al[18] explained the linkage between the frequency of enamel hypoplasia with chronological teeth calcification. The upper incisor is the first teeth to be calcificated at the third to fourth months prenatal. The amelogenesis of the primary incisor is almost complete at birth, but not the caninus and molar teeth. Theoretically, if the disorder occured at the 12th week prenatal, the hypoplasia lesion will appear on the central incisor of the upper and lower jaws.[20] Accordingly, if the disorder occured at the 16th week, enamel hypoplasia will appear on the posterior teeth, because that is the time of enamel matrix forming on the anterior and posterior primary teeth. While enamel hypoplasia on children with normal birth weights is caused by local or traumatic factors.[21]

Based on Diagram 4, enamel hypoplasia occurs more often on females, and the comparison of females : males was 11.42 : 10.00. This was in accordance with the study by Griffin and Donlon[22] that revealed the prevalence on enamel hypoplasia on females was 67.2%, and 32.7% on males. But it was different with the study by Alhammad[23] hat revealed higher prevalence of enamel hypoplasia on males compared with females (25.6 : 24.1). The difference between males and females was relatively low and not significant.

V. CONCLUSION

The frequency distribution of enamel hypoplasia on Down Syndrome children at POTADS foundation was 21.42%.

REFERENCES

[1]. Shin, M., Besser, L. M., Kucik, J. E., Lu, C., Siffel, C., & Correa, A. 2009. Prevalence of *Down Syndrome* Among Children and Adolescents in 10 Regions of the United States. *Pediatrics*. Vol. 124(6), 1565-1571.

[2]. Rampi, C. E., & Gunawan, P. 2017. Gambaran Kebersihan Gigi dan Mulut pada Anak Penderita *Down Syndrome* di SLB YPAC Manado. *JurnalKedokteran Klinik*. Vol. 1(3), 35-41.

[3]. Acerbi, A. G., Freitas, C. D., & Magalhaes, M. H. 2001. Prevalence of numeric anomalies in the permanent dentition of patients with Down syndrome. *Spec Care Dentistry*. Vol. 21(2), 75.

[4]. Finn, S. B. 2003. *Clinical Pedodontics 4th Edition.* W.B. Saunders.

[5]. Chowdhury, M.R. and Dubey, S., 2014. Role of Cytogenetics and Molecular Genetics in Human Health and Medicine. In A, S, Verna & A. Signh, eds. *Animal Biotechnology*. Elsevier, 451-472.

[6]. Aminabadi, N. A., Oskouei, S. G., Pouralibaba, F., Jamali, Z., & Pakdel, F. 2009. Enamel Defects of HumanPrimary Dentition as Virtual Memory of Early Developmental Events. *Journal of Dental Research, DentalClinics, Dental Prospects*. Vol. 3(4), 111-113.

[7]. Amalliah, I. 2002. Defek Email pada Gigi Anak Periode Usia Gigi Sulung dan Permanen. *Fakultas Kedokteran Gigi, Universitas Indonesia* , 9.

[8]. Bertoli, M., Biasini, G., Calignano, M. T., Celani, G., et al. 2011. Needs and Challenges of Daily Life For People With Down syndrome Residing in The City of Rome, Italy. *Journal of Intellectual Disability Research*. Vol. 55(8),801-820.

[9]. Kovaleva, N. V. , Butomo, I. V., Korblein, A. 2001. Sex ratio in Down syndrome. Studies in patients with confirmed trisomy 21. Article in Sitologia Genetika.

[10]. Martin, Renee, H. 2008. Meiotic errors in human oogenesis and spermatogenesis. Reproductive BioMedicine Online. Vol. 16(4), 523-531.

[11]. Sherman, S.L., Freeman, S. B. Allen, E. G. 2005. Risk factors for *nondisjunction* of trisomy 21. Cytogenetic and Genome Research. Vol. 111, 273-280.

[12]. Radhi, N. J., El-Samarrai, S. K., & Alkhafaji, J. T. 2015. Enamel Defect of Primary and Permanent Teeth in Relation To Nutrients Daily Intake Among Down's Syndrome Children. *Journal of Baghdad College ofDentistry*. Vol. 27(3), 152-158.

[13]. Slayton, R. L., Warren, J. J., Kanellis, M. J., Levy, S. M., & Islam, M. 2001. Prevalence of Enamel Hypoplasia and Isolated Opacities in The Primary Dentition. *Pediatric Dentistry*. Vol. 23(1), 32-36.

[14]. Lunardelli, S. E., & Peres, M. A. 2005. Prevalence and Distribution of Developmental Enamel Defects in The Primary Dentition of Pre-School Children. *Brazilian Oral Research*. Vol. 19(2), 144-149.

[15]. Balogh MB, Fehrenbach MJ. (2006). *Dental Embryology, Histology, andAnatomy 2nd Ed*. St. Louis: Elsevier Saunders.

[16]. Dritsa, V., et al. 2015. *Enamel Structure on Children with Down Syndrome - AnFT-IR Spectrosocpic Study.* Intech.

[17]. Carvalho,L., Bernardon,J., Bruzi,G., Andrada,M, & Vieira,L.2003. Hypoplastic Enamel Treatment in Permanent Anterior Teeth of a Child. Operative Dentistry, Vol.38(4), 363-268.

[18]. Franco, K. M., Line, S. R., & Moura-Ribeiro, M. V. 2007. Prenatal and Neonatal Variables Associated with Enamel Hypoplasia in Deciduous Teeth in Low Birth Weight Preterm Infants. *Journal of Applied Oral Science*. Vol. 15(6), 518-523.

[19]. Aine, L. et al. (2000). Enamel Defects in Primary and Permanent Teeth of Children Born Prematurely. *Journal Oral Pathology Medicine*. Vol. 29, 403-409.

[20]. Idiculla, J. J., Brave, V., Puranik, R., & Vanaki, S. 2011. Enamel Hypoplasia and its Correlation with Dental Caries in School Children of Bagalkot, Karnataka. *Journal of Oral Health and Community Dentistry*. Vol.5(1), 31-36.

[21]. Willyanti. 2013. *Enamel Defect of Primary Dentition in SGA Children inRelation to Onset Time of Intrauterine Growth Disturbance.* DentalJournal. Vol. 46(2), 55-60.

[22]. Griffin, R. C., & Donlon, D. 2009. Patterns in Dental Enamel Hypoplasia by Sex and Age at Death in Two Archaeological Populations. *Archives OralBiology* , 93-100.

[23]. Alhammad, Nouf, S., 2011. Tooth Wear, Enaml Hypoplasia and Traumatic Dental Injuries Among Cerebral Palsy Children of Riyadh City. *King SaudUniveristy Journal of Dental Sciences*. Vol. 2, 1-5.

Correspnding Author: Meirina Gartika,
Department of Pediatric Dentistry, Faculty of Dentistry, Universitas Padjadjaran