MEDWELL PUBLISHING
Research Journal of Biological Sciences 4 (4): 537-541, 2009
ISSN: 1815-8846

An Evaluation of the Rate of Dental Caries among Hypoplastic and Normal Teeth: A Case Control Study

M. Vahid Golpaygani, K. Mehrdad, A. Mehrdad and G. Ansari
Department of Paedodontics, Dental School, Shahid Beheshti Medical University, Evin, Tehran, Iran

Abstract: Structurally defected teeth are not only weak but also provide favorable areas for colonization of bacteria. This subsequently will increase the incidence of dental caries in such dentition. Enamel hypoplaisa is considered one of many caries inducing factors. The aim of this investigation was to evaluate the frequency of dental caries among hypoplastic teeth in a group of 6-9 years old Iranian children. One hunder and one children of 6-9 years of age from Paedodontic Department of Shahid Beheshit Dental School were randomly selected. All the primary and permanent teeth of these children (2266 teeth) were examined for the Presence of enamel hypoplasia and dental caries. Overall, 1671 of teeth (73.9%) were primary and 589 (26.1%) were permanent. Of the permanent teeth, 315 were first permanent molars. Pearson, Mantel Haenszel and Fisher Exact test were used to analyze the data. A significant relationship was found between enamel hypoplasia and dental caries both in permanent ($p<0.0001$) and primary teeth ($p = 0.038$). The odds ratio was 7.362 for all the permanents, 4.47 for the first permanent molar and 1.887 for the primary teeth. Hypoplastic teeth are more likely to develop caries than non-hypoplastic teeth. Hypoplastic primary teeth are 2 times and the 1st permanent molars are 4 times more susceptible to dental caries than non-hypoplastic ones.

Key words: Hypoplasia, enamel, first molar, permanent, caries, restoration, permanent teeth

INTRODUCTION

Enamel hypoplasia is the result of disruption in the process of enamel matrix formation, which in turn causes defect in quality and thickness of enamel. These defects vary in nature such as dots, grooves and/or variation in color. Depending on the length and severity of disruption in ameloblastic activity, limited or broader areas of enamel could become involved (Prime *et al*., 1984).

Those factors that affect earlier amelogenesis can clearly produce more severe results. Prenatal disruption, even mild ones, can produce linear defects called prenatal lines. Both dentitions could be affected by enamel hypoplasia, however, the incidence is more severe in permanent dentition (Prime *et al*., 1984; Limeback *et al*., 1992).

Local or systemic factors interfering with natural process of amelogenesis cause what is called hypoplasia of enamel. While, the factors affecting the calcification or maturation of enamel is named enamel hypoclacification. Clinically, hypoplasitc enamel shows a rough and pitted surface and can easily absorb dies. A number of studies have demonstrated the relationship of childhood diseases and enamel hypoplasia (Fraser and Nikiforuk, 1982; Monteron *et al*., 2003).

Recent investigations indicate that 3-15% of children show signs of enamel hypoplasia in their permanent dentition (Fraser and Nikiforuk, 1982; Monteron *et al*., 2003; Jälevik *et al*., 2005). The incidence of these defects has increased in individuals with vitamin D deficiencies, hyper-para thyroidism, congenital diseases and those with maternal rubella infection (Pindborg, 1982). Linear hypoplaisa can frequently be detected in primary teeth of children from 3rd world countries (Duncan *et al*., 1988; Silberman *et al*., 1989; Matee *et al*., 1994). Both systemic and local factors are responsible for the occurrence of this phenomenon. Such systemic factors include genetic and chromosomal abnormalities, congenital, metabolic and CNS defects, infectious and endocrine diseases, nutritional disorders, intoxication, kidney, liver and intestine diseases. Local factors include trauma, electrical burns, X-ray exposure and local infection. Many researches have indicated that there is a possible correlation between enamel hypoplasia and dental caries (Silberman *et al*., 1991; Pascoe and Seow, 1994).

Most of the previous studies have mainly been conducted on primary dentition. Consequently, the risk factors causing caries were not minimized in primary dentition. Direct clinical examination of newly erupted teeth would provide a more reliable result. Interestingly, restored teeth were not included in any of the previously

Corresponding Author: G. Ansari, Department of Paedodontics, Dental School, Shahid Beheshti Medical University, Evin, Tehran, Iran

reported studies, which could have been hypoplastic prior to the restoration. The aim of this investigation was, therefore, to evaluate the frequency of dental caries among hypoplastic teeth in a group of 6-9 years old Iranian children.

MATERIALS AND METHODS

This analytical case-control study was performed on a group of 101 children, between 6-9 years of age. Patients were selected from those referred to pedodontic Dept at the Dental school, Shaheed Beheshti Medical University, Tehran. Only cases were included who represented with erupted first permanent molars. Hypoplastic teeth were used as cases and healthy teeth were used as controls. General information was obtained through observation, interviewing the parents and a questioner. Examination of teeth was started from the right maxillary first permanent molar to the left maxilla, followed by left then right mandibular first permanent molars. WHO chart was used to record the type of caries. The types of enamel hypoplasia were categorized in teeth based on Silberman *et al*. (1990).

Type I hypoplasia: Enamel discoloration due to hypoplasia (Fig. 1).

Type II hypoplasia: Abnormal coalescence due to hypoplasia (Fig. 2).

Type III hypoplasia: Missing some parts of enamel due to hypoplasia (Fig. 3).

Type IV hypoplasia: A combination of previous three types of hypoplasia (Fig. 4).

In cases of simultaneous presence of both the primary tooth and its corresponding permanent tooth, data was gathered on the permanent tooth only. A total of 2260 teeth were the final number of teeth examined, from which 315 were first permanent molars. Non-probability approach was used as the sampling method. The statistical analysis was carried out using SPSS 10 software and the test employed were fisher exact and

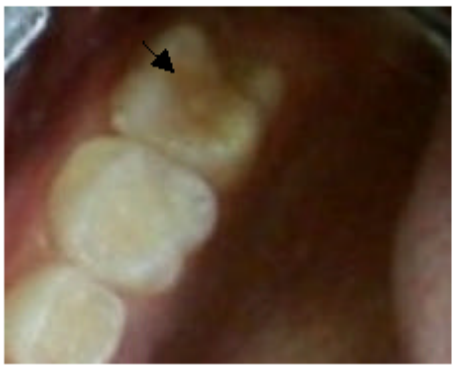

Fig. 1: Enamel hypoplasia type I, arrow shows enamel discoloration

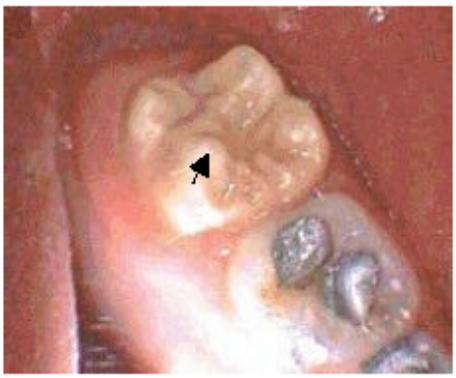

Fig. 2: Enamel Hypoplasia type II. Arrow shows abnormal coalescence

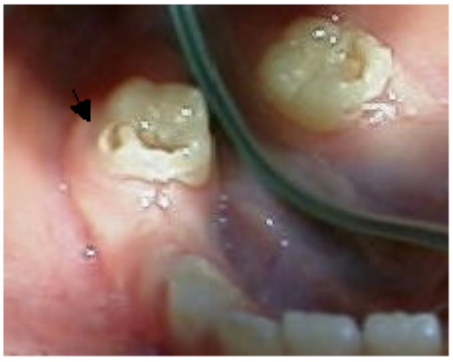

Fig. 3: Enamel hypoplasia type III. Arrow shows enamel destruction due to the defect

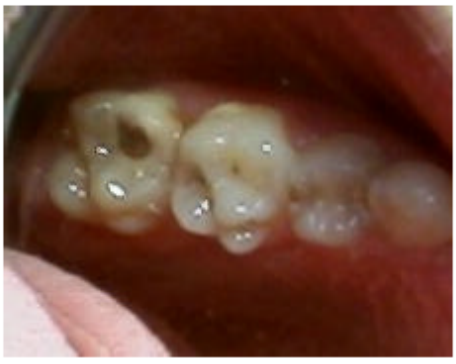

Fig. 4: Enamel hypoplasia type IV, A combination of types I, II and III

pearson and manteal-haenszel tests. The odds ratio was also calculated for further interpretation of the collected data.

RESULTS

From 101 children who were examined in this study, 27 (26.7%) had at least one tooth with enamel hypoplasia and the total number of hypoplastic teeth were standing at 80. Out of 1606 primary teeth, however, 43 teeth were affected by enamel hypoplasia in which 20 of them were carious or had restorations. The differences were statistically significant between the prevalence of dental caries in normal and hypoplastic primary teeth (Pearson chi-square (χ^2) = 4.314, df = 1, p = 0.038). The odds ratio (the chance of occurrence of dental caries in hypoplastic primary teeth was 1.887 with the following calculation:

$$\text{Odds ratio} = \left(\frac{1070\times20}{23\times493}\right) = 1.887$$

The confidence limit was between 1.27 and 3.469. Similar results were also obtained and confirmed by Mantel Haenszel test, p = 0.04 (Table 1).

Out of 589 permanent teeth of this study 37 were affected by enamel hypoplasia in which 11 were either carious or had a type of restoration (Table 2). The prevalence of dental caries in hypoplastic permanent teeth were found to be more than those without enamel hypoplasia. This was statistically significant when Pearson and chi-square (χ^2) tests were used to assess the data (χ^2 = 31.603 df = 1, p<0.0001).

The odds ratio was 7.362 and the confidence limits were between 3.324 and 16.306. This result was confirmed by Mantel Haenszel test too (p<0.0001).

Assessing the first permanent molars indicated that 34.4% of those with enamel hypoplasia had either restorations or caries while for non hypoplastic teeth the number was 10.6%. This difference was found to be statistically significant (Pearson chi-square (χ^2) = 14.353, df = 1, p<0.0001).

The odds ratio was 4.417 and the confidence limits was between 1.942 and 10.048. The above data was confirmed by Mantel-Haenszel test (p<0.001) (Table 3).

To indicate, which type of enamel hypoplasia is more responsible for dental caries, the number of carious teeth was evaluated based on their types. Results indicated that only in type 4 the incidence of dental caries as being increased significantly (Table 4) (Fisher exact test p = 0.008, Mantel-Hanszeal test p = 0.004).

Table 1: The number of primary teeth in relation with enamel hypoplasia, dental caries and fillings

Primary teeth	Without caries or filling	With caries or filling	Total
Without enamel hypoplasia	1070	493	1563
With enamel hypoplasia	23	20	43
Total	1093	513	1606

Table 2: The number of permanent teeth with enamel hypoplasia, dental caries or fillings

Permanent teeth	Without caries or filling	With caries or filling	Total
Without enamel hypoplasia	522	30	552
With enamel hypoplasia	26	11	37
Total	548	41	589

Table 3: The number and percentage of first permanent molars with enamel hypoplasia, dental caries and fillings

1st permanent molar	Without caries or filling (%)	With caries or filling (%)	Total (%)
Without enamel hypoplasia	253 (89.3)	30 (10.6)	283 (100)
With enamel hypoplasia	21 (65.3)	11 (34.4)	32 (100)
Total (%)	274 (100.0)	41 (100.0)	315

Table 4: The number of first permanent molars with different types of enamel hypoplasia in relation to caries and fillings

1st permanent molar	Without caries or fillings	With caries or fillings	p-value
Wthout enamel Hypoplasia	253	30	-
Type I eamel hypoplasia	9	3	0.139
Type II eamel hypoplasia	2	2	0.063
Type III Eamel hypoplasia	6	2	0.216
Type IV Enamel hypoplasia	4	4	0.008

DISCUSSION

The prevalence of dental caries in hypoplastic first permanent molars has indicated that enamel hypoplasia could contribute to the risks of dental caries. The minor variations among the earlier reported studies seems to mainly be due to differences in the method of study and children's age (Nikiforuk and Fraser, 1979; Limeback *et al*., 1992; Duncan *et al*., 1994). The age range of 6-9 years was considered for this investigation in order to minimize the risk of any interfering factor causing enamel caries in this age group of children.

The odd ratio (the chance of occurring dental caries in hypoplastic primary teeth) was found to be at 1.887 (Table 1). This indicates that the hypopastic primary teeth are almost 2 times more prone to dental caries than normal ones (p = 0.038). These differences has also been reported by earlier studies (Duncan *et al*., 1994; Li *et al*., 1996; Lai *et al*., 1997).

Hypoplastic permanent teeth were more sensitive to dental caries process when compared with Primary teeth as seen in the result of current investigation. The odds ratio of dental caries in relation to hypoplastic permanent teeth was 7.362. In other word, the hypoplastic permanent teeth are 7 times more sensitive to carious attack compare to those without hypoplasia (p<0.0001).

The majority of earlier investigations have been performed on primary dentition and there is a lacking in regards to the information on hypoplastic permanent molars. This evaluation indicates that the hypoplastic permanent dentition suffer more from caries attack than the primary teeth.

Restored primary or permanent teeth are also another subject that has not received enough attention in the earlier reports, while restored teeth may have had hypoplastic enamel which quite easily lead to caries. Hypoplasia in first permanent molars was also found to have a significant rise in the chance of dental caries development based on the current results ($p<0.0001$).

The odds ratio was 4.417 for relationship between enamel hypoplasia and dental caries in first Permanent molars which means first permanent molars with enamel hypoplasia are 4 times more prone to enamel caries. Children within 6-9 years age range were included in this investigation as the first permanent molars are present and still not affected by other causes of dental caries. Increased incidence of caries in the first permanent molars indicates that structural defects in enamel make it more soluble resulting in a faster break down pattern of enamel rods. This in turn, facilitates the presence of dental caries even before other risk factors could even gain enough time for enamel destruction. Among different types of enamel hypoplasia, which were classified earlier in this article, type 4 had significantly increased the risk of dental caries ($p<0.008$).

Although, all types of enamel hypoplasia is a predisposing factor for dental caries but as type 4 of the defects is very severe and destruction of enamel rods is so dramatic resultant dental caries is inevitable. Based on these findings, specially on first permanent molars, the enamel hypoplasia should be detected and properly managed on the earliest possible stage in order to enable an efficient prevention from further enamel destruction. Due to child's lack of awareness of good oral hygiene, parental intervention is of extreme importance, especially for those between 6-9 years of age.

CONCLUSION

- A significant relationship was seen between enamel hypoplasia and the prevalence of dental caries in primary and permanent dentition
- First permanent molars with hypoplasia are 4 times (4.417) more prone to dental caries than non-hypoplastic ones (confidence limits =1.942-10.048)
- According to the result of this investigation type IV hypoplasia (combination of other 3 types) demonstrated the strongest association with dental caries.

ACKNOWLEDGEMENT

Authors would like to thank Dr. Arash Vahid for his efforts in the preparation process of this manuscript.

REFERENCES

Duncan, W.K., S.L. Silberman and A. Trubman, 1988. Labial hypoplasia of primary canines in black Head Start children. J. Dent. Child, 55 (6): 423-426. PMID: 3198832.

Duncan, W.K., S.L. Silberman, A. Trubman and E.F. Meydrech, 1994. Prevalence and racial distribution of primary canine hypoplasia of the maxillary canine. Pediatr. Dent., 16 (5): 365-367. PMID: 7831143.

Fraser, D. and G. Nikiforuk, 1982. The etiology of enamel hypoplasia in children-a unifying concept. J. Int. Assoc. Dent. Child., 13 (1): 1-11. PMID: 6958762.

Jälevik, B., W. Dietz and J.G. Norén, 2005. Scanning electron microscopic analysis of hypomineralized enamel in permanent 1st molars. Int. J. Paediatr. Dent., 15 (4): 233-240. PMID: 16011781.

Li, Y., J.M. Navia and J.Y. Bian, 1996. Caries experience in deciduous dentition of rural Chinese 3-5 years old children in relation to the presence or absence of enamel hypoplasia. Caries Res., 30 (1): 8-15. PMID: 8850577.

Lai, P.Y., W.K. Seow, D.I. Tudehope and Y. Rogers, 1997. Enamel hypoplasia and dental caries in very low birth weight children. A case controlled, Longitudinal study. Pediatr. Dent., 19 (1): 42-49. PMID: 9048413.

Limeback, H., C. Schlumbohm, A. Sen and G. Nikiforuk, 1992. The effects of hypocalcemia/hypophosphatemia on porcine bone and dental hard tissues in an inherited form of type 1 pseudo-vitamin D deficiency rickets. J. Dent. Res., 71 (2): 346-352. PMID: 1556293.

Matee, M., van't M. Hof, S. Maselle, F. Mikx and van W.P. Helderman, 1994. Nursing caries, linear hypoplasia and nursing and weaning habits in Tanzanian infants. Commun. Dent. Oral. Epidemiol., 22 (5 Pt 1): 289-293. PMID: 7813178.

Monteron, M.J., J.M. Douglass and G.M. Mathieu, 2003. Prevalence of dental caries and enamel defects in connecticut head start children. Pediatr. Dent., 25 (3): 253-259. PMID: 12889699.

Nikiforuk, G. and D. Fraser, 1979. Chemical determinants of enamel hypoplasia in children with disorders of calcium and phospha February 20, 2009 homeostasis. J. Dent. Res., 58 (B): 1014-1015. PMID: 216712.

Pascoe, L. and W.K. Seow, 1994. Enamel hypoplasia and dental caries in Australian Aboriginal children: Prevalence and correlation between the 2 diseases. Pediatric. Dent., 16 (3): 193-199. PMID: 8058543.

Pindborg, J.J., 1982. Etiology of developmental enamel defects not related to fluorosis. Int. Dent. J., 32 (2): 123-133. PMID: 6749692.

Prime, S.S., D.G. MacDonald, H.W. Noble and J.S. Rennie, 1984. Effect of prolonged iron deficiency on enamel pigmentation and tooth structure in rat incisors. Arch. Oral. Biol., 29 (11): 905-909. PMID: 6596037.

Silberman, S.L., W.K. Duncan, A. Trubman and E.F. Mydrech, 1989. Primary canine hypoplasia in head start children. J. Pub. Health Dent., 49 (1): 15-18. PMID: 2783460.

Silberman, S.L., A. Trubman, W.K. Duncan and E.F. Meydrech, 1990. A simplified hypoplasia index. J. Pub. Health Dent., 50 (4): 282-284. PMID: 2391677.

Silberman, S.L., A. Trubman, W.K. Duncan and E.F. Meydrech, 1991. Prevalence of primary canine hypoplasia of the mandibular teeth. Pediatr. Dent., 13 (6): 356-360. PMID: 1843992.