

Enamel hypoplasia of primary canine: Its prevalence and degree of expression

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Abstract

Background: Enamel hypoplasia is a unique lesion showing wide variations in prevalence among populations. The present study aimed to evaluate frequency and degree of expression of enamel hypoplasia of primary canine in populations living in the eastern part of India. **Materials and Methods:** A prospective cross sectional study was performed in randomly selected 3539 school children. Labial surfaces of all primary canines were examined. The frequency and degree of expression of enamel hypoplasia between different ethnic groups were recorded. Based on the location of the defect on the tooth surfaces, the lesions were divided as prenatal, perinatal, and postnatal according to stage of mineralization. Statistical analysis was carried out using Chi square test. **Results:** The overall prevalence of primary canine hypoplasia was 10.3%. The mandibular primary canines were significantly ($P = 0.03$) more frequently affected than maxillary canines among different ethnic groups. The side difference among populations was not significant ($P = 0.96$) statistically. In 8.5% Bengali population, 7% Rajbangsi population, and 9.4% in other group of population, the defect occurred prenatally. Most of the defects, 64.7% in Bengali, 66.1% in Rajbangsi, and 58.8% in others occurred during perinatal stage of mineralization. The defect occurred postnatally in 26.8%, 26.7%, and 31.8% in Bengali, Rajbangsi, and in other group of population respectively. **Conclusion:** The overall prevalence of primary canine hypoplasia was 10.3%. The mandibular primary canines were more frequently affected ($P = 0.03$) than their maxillary counterparts among populations. Most of the defects correspond to perinatal stage of mineralization.

Key words: Enamel hypoplasia, populations, primary canine, stage of mineralization

INTRODUCTION

Localized enamel hypoplasia of primary canine has been reported in both prehistoric and modern populations with a prevalence varying from 0% to 89%.^[1] The lesion has been described as a roughly circular area of 1-2 mm diameter with a flat or concave configuration.^[2,3] The defect was first reported by Jorgenson (1956) who observed it in 21% of modern and 28% of medieval Danish population.^[4]

The etiology of the lesion is not clearly understood. Genetic and environmental factors interfering with enamel matrix formation are believed to be responsible for the development of the lesion. Skinner suggested that minor trauma to the developing canine tooth bud might be an important etiological factor.^[5]

The defect is seen more frequently in the mandibular canines than in the maxillary canines. According to Silberman *et al.*, the lesion may appear as minimal hypoplasia which is white, brown, or yellow in color with smooth surface texture, or sometimes obvious hypoplasia characterized by a combination of pitting, invagination, missing enamel, or a rough surface.^[6] Various investigators have found a significant association between dental caries and developmental enamel defects of primary canines.^[7,8]

The initiation of calcification of primary canine starts at 5 months in utero, beginning at the cusp tip and

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mineralization of crowns of all primary canines is not complete until about 9 months after birth. At birth, the incisal one third of the crown is formed. Thus, the location of the defect may provide valuable clues about the onset of the lesion.

The purpose of this article was to report the prevalence of labially occurring enamel hypoplasia of primary canine in a group of healthy children living in the eastern part of India. These children belong to relatively homogeneous socioeconomic level. The frequency and degree of expression of the developmental enamel defect between different ethnic groups and the location of the defect in relation to the stage of mineralization were studied.

MATERIALS AND METHODS

A prospective cross-sectional study was carried out using a two-stage random sampling technique among 5- to 7-year-old primary school children. A total of 32 primary schools located in the two districts of West Bengal were randomly selected. Ethical committee clearance was obtained from the concerned authority. A signed informed consent was received from each child's parent prior to the study. Selection criteria of the samples include children with negative history of extraction, caries, or restoration on any of the primary canines. Children with known systemic diseases or syndromic background were also excluded from the study. After elimination, 3539 school children were randomly selected. Among them, 1857 were boys and 1682 were girls. The field work was performed from January 2009 to December 2011. The dental examination was carried out by one examiner using the daylight. The labial surfaces of all primary canines were examined closely for the presence of a lesion. The presence or absence of labial enamel hypoplasia was recorded separately for each tooth. The enamel hypoplasia was recorded when there was a lack of continuity of the surface enamel.^[9] Brown, yellow, or opaque white discoloration with an intact surface was also recorded as enamel hypoplasia. Only obvious, not borderline cases were recorded. Gender and ethnicity of children were determined by their surnames. Based on the position of the defect on the tooth surface, the lesions were then divided as prenatal, perinatal, and postnatal according

to the stage of mineralization.^[9] The Chi-square test was used for statistical analysis of the data with 0.05 level of significance was set.

RESULTS

No differences in the occurrence of enamel hypoplasia between boys and girls were observed, thus their results were united. Table 1 shows the prevalence of defect in different populations studied. The overall prevalence of enamel hypoplasia was 10.3% of the 3539 children studied. The prevalence of the developmental defect in the upper arch was 2.3% in Bengali population, and 3.2% each in Rajbangshi and in the other group of population. In the lower arch, the prevalence was much higher, 11.9% in Bengali children, 8.5% in Rajbangshi population, and 10.2% in other group of population. The difference in the occurrence of at least one lesion between different ethnic groups was not significant ($P = 0.64$) [Table 1].

The expression of enamel defect by arch and by side among different populations is shown in Table 2. The left primary canines were more frequently affected than the right primary canines. However, the differences in the occurrence of hypoplastic lesions between the two sides were not statistically significant ($P = 0.96$) between different population groups. The location of the defect in the upper and the lower arches differed significantly among the populations ($P = 0.03$).

The majority of lesions occurred at a level corresponding to the perinatal stage of enamel mineralization in all populations studied here. This was followed by post natal and prenatal stages of mineralization [Table 3].

DISCUSSION

In this study, no statistically significant difference in the occurrence of the lesion was observed between boys and girls. This is consistent with the findings of Badger (1985), Silberman *et al.* (1989), and Lukacs (1991), but is in contrast with the other studies which showed that boys were more frequently affected than girls.^[8,10-12]

Table 1: Enamel hypoplasia of primary canine in relation to population groups

Children with	Bengali		Rajbangshi		Others	
	No	Percent	No	Percent	No	Percent
1 tooth involved	113	7.9	103	7.8	69	8.8
2 teeth involved	29	2.0	22	1.7	13	1.7
3 teeth involved	11	0.8	0	0	3	0
All canine involved	0	0	2	0.1	0	0
Children with no lesion	1280	89.3	1200	90.4	694	89.1

The difference in the occurrence of atleast one lesion between different population groups was not significant ($X^2=0.874$, $df=2$, $P=0.64$)

Table 2: The occurrence of hypoplastic lesion in the upper and lower arches

Arch	Bengali		Rajbangshi		Others	
	No	Percent	No	Percent	No	Percent
Maxilla						
Right	12	0.8	20	1.5	–	1.2
Left	21	1.5	22	1.7	9	2
Mandible						
Right	76	5.3	48	3.6	37	4.7
Left	95	6.7	65	4.9	41	5.2

Significant difference ($\chi^2=6.73$, $df=2$, $P=0.03$) was observed between upper and lower arches, but not between right and left sides ($\chi^2=0.06$, $df=2$, $P=0.96$) among populations

Table 3: Primary canine hypoplasia in relation to stages of mineralization

Population	Prenatal		Perinatal		Postnatal	
	No	Percent	No	Percent	No	Percent
Bengali	13	8.5	99	64.7	41	26.8
Rajbangshi	9	7	84	66.1	34	26.7
Others	8	9.4	50	58.8	27	31.8

Various investigators have studied the prevalence of enamel hypoplasia in the primary dentition.^[15-17] Relatively few studies are available on the developmental enamel defects in the primary canine. In the present study, prevalence of labially occurring enamel hypoplasia of primary canines was 10.3%. Badger (1985) in his survey of 220 US children observed 45% of primary canines as hypoplastic.^[10] Duncan *et al.* (1988) reported a prevalence of 37% in a group of 334 African American children.^[18] Silberman *et al.* (1991) found primary canine hypoplasia in 17% American and 33% African American children.^[8] Skinner and Hung (1986) observed a very low prevalence of 0.5% in a group of 2380 children.^[3] The prevalence observed in this study falls within the lower end of the range reported in the dental literatures. The considerable variations in prevalence may be due to differences in sample size, population group studied, and the criteria used for diagnosis of the lesion.

The results of this study showed that hypoplastic defect occurs significantly more frequently in the mandibular canines than in the maxillary canines ($P = 0.03$). Taji and others, in an Australian study, also reported an increased frequency of enamel hypoplasia in the mandibular canines than in the maxillary canines.^[2] Duncan and others (1994) observed primary canine hypoplasia three times more prevalent in the mandibular arch than in the maxillary arch.^[19] However, Badger and Needleman (1985) in their study found an almost equal distribution of the defect between maxillary and mandibular canines.^[10,15] Thus, the results of this study support the findings of most other studies. This may be partly explained by the fact that lower canines are more vulnerable to trauma or thick cheek muscles providing some protections to maxillary canines.

The etiology of the lesion is not exactly known. Jorgenson (1956) related it to a genetic origin.^[4] Taji (2000) concluded that none of environmental, genetic, or systemic factors can be ruled out.^[2] One possible explanation according to Nation *et al.* is that during birth, trauma to the developing canine is induced by compression of the lower buccal alveolar wall by pressure from the maxilla.^[9] Skinner (1986) also observed in some children that alveolar fenestration present over the developing primary canine tooth buds is very thin, exposing the developing tooth crown to physical trauma.^[5] Investigators have reported a relatively high prevalence of the defect on the mesial side of the primary canines.^[2,19] This probably indicates traumatic etiology of the lesion. Malnutrition resulting in calcium and phosphorus deficiency has also been implicated in producing primary canine hypoplasia.^[1,3,8]

The initiation of mineralization of all primary canines begins at cusp tip and progresses in the cervical direction. Because enamel cannot recover once it is damaged, enamel defects might provide repository of information on the timing of insult.^[17] In our study, most of the defects occur at mid crown level which corresponds to the perinatal stage of tooth mineralization. This is in agreement with the findings of other investigators.^[2,9] One possible explanation of enamel defects occurring at the middle third area of tooth crown is trauma to the developing tooth bud during child birth. The incisal third area of tooth crown is least affected by the developmental defect. This part of tooth crown is formed during intrauterine life. The developing tooth bud is relatively well protected in intrauterine life. Nation *et al.* suggested that nutritional deficiency or diseases during pregnancies might be responsible for the prenatal enamel defect.^[9]

Various investigators have found a significant association between ethnicity and primary canine hypoplasia.^[18,19] Duncan *et al.* observed that the lesion affects African Americans two times more frequently than the Americans.^[18] The relatively protruded position of primary canines, according to them, might be responsible for it. In our study, no significant difference in prevalence was observed among the populations.

CONCLUSION

- The overall prevalence of localized enamel hypoplasia of primary canine was 10.3%
- The mandibular primary canines were significantly ($P = 0.03$) more frequently affected than maxillary canines among populations in this study
- The left primary canines were more commonly associated with this developmental defect than

the right primary canines in both the arches among all populations studied here. However, this difference among populations was not statistically significant ($P = 0.96$)

- Most of the defect corresponds to the perinatal stage of tooth development.

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