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AGENESIS OF MANDIBULAR SECOND PREMOLAR

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ABSTRACT

Tooth agenesis is the most common of all dental developmental disorders. The mandibular second premolars are the teeth most commonly found missing (2.2 to 4.1%) after the third molars. There are more studies on tooth agenesis than the other malformations but yet they are limited. This article reviews the studies related to agenesis of mandibular second premolars and the underlying mechanisms behind the occurrence of this anomaly.

KEYWORDS: Mandibular second premolar; Agenesis; Genetics

INTRODUCTION

Numerous dental anomalies have been identified in the human dentition. Research is underway to unearth any correlation between the occurrences of seemingly varied anomalies. It is found that there are more studies on tooth agenesis than the other malformations.^[1] Absence of mandibular second premolars in studies done on the general population is found to be 2.5 to 5% in USA and Europe. This includes both unilateral and bilateral absence, the latter accounting for 60% of cases. The mandibular second premolars are the teeth most commonly found missing (2.2 to 4.1%) after the third molars.^[2, 3]

DISCUSSION

The most common of all dental developmental disorders is tooth agenesis and is found in one fourth of the total population (4.3 to 7.8% when third molars are excluded). Agenesis is more prevalent in females.^[3] According to a study on permanent teeth in Caucasians agenesis is evenly distributed in the maxilla and mandible. It is also

more common to find unilateral absence of a permanent tooth than bilateral.^[4] However the mandible is more affected in a sample of Japanese patients with 76.3% of patients exhibiting agenesis of multiple teeth. A study on 176 White European patients by Stritzel, Symons and Gage is the only one which was done exclusively to evaluate the agenesis of second premolars with regard to distribution, number and sites involved in either gender.^[3] The results of their study showed that agenesis of second premolars occurs more in the mandible, but when it affects the maxilla the agenesis occurs more symmetrically. Also in three fourths of the cases the agenesis occurred in one or two second premolars. All parameters were found to be the same for both sexes.^[5] Agenesis or delayed maturity of mandibular second premolars is more marked when other permanent teeth are also absent in the same case. This was revealed in a study on Brazilian patients where the agenesis of other permanent teeth was studied in a sample of mandibular second premolar agenesis. The mandibular second premolar initially mineralizes at three years of age ranging between two years and three months to three years and seven months. The eruption may occur 'upto 5 years, after 9 years or even at 13 years of age'.^[3] It was found by Bjerklin et al. that submerged deciduous molars caused aplasia of the premolars and vice versa.^[6] Also the presence of the primary molars at 20 years of age indicates a better chance for their retention.^[7] The development of tooth takes place in various stages. In each stage the tooth germ has to attain a threshold size, failing which it will no longer progress in its development. The tooth germ in such instances will undergo regression leading to hypodontia. Hypodontia and hypoplasia are essentially the same anomaly

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same anomaly manifested differently. There are many published pedigrees which establish this fact. An association is evident between congenital absence of a tooth and a decrease in its size as shown by the simultaneous occurrence of hypodontia of one and hypoplasia of the other incisor.[8] maxillary lateral Agenesis of mandibular second premolar results in delayed maturity of canines in both boys and girls but to a greater extent in girls (p=0.009). It is interesting to note that second molars are delayed in girls (p=0.04) but not in boys (p=0.98).^[9]



Fig 1: Agenesis of mandibular second premolars with retained deciduous second molars left in situ in a 38 year old female

GENETICS

The role of genetics in the occurrence of agenesis and other anomalies is irrefutable. A mutation in gene MSX1 on chromosome 4p has been detected by Vastardis. This was the result of a study conducted on a large family with agenesis of all second premolars and third molars.^[10] Many dental developmental anomalies have been hypothesized to be factors of the same genetic mutation. Hence they can be expected to be seen in the same subject or among relatives. A Japanese study on patients with agenesis of both mandibular second premolars has revealed "significantly high risk of agenesis of other types of permanent teeth, symmetrical tooth agenesis and oligodontia".^[11] The anomalies implicated by Hoffmeister and Pfeiffer include hypodontia, supernumerary teeth, peg-shaped lateral incisors, and ectopic eruption of maxillary first permanent molars. These are all due to alterations in the odontogenic apparatus. The incidence of agenesis of mandibular second premolars was found to be increased sixfold in cases where palatally displaced canines (PDCs) or maxillary caninefirst premolar transposition were present.^[12] Another study that supports this hypothesis is by

Garib et al. where only subjects with second mandibular premolar agenesis were studied. They found that agenesis of other permanent teeth, microdontia, deciduous molar infraocclusion, and certain dental ectopias such as palatally displaced canines and distoangulation of mandibular second premolars were found to be present concomitantly.^[13] The association between morphologically different anomalies has been well demonstrated in the study by Baccetti. Seven anomalies, namely aplasia of second premolars, small maxillary lateral incisors, infraocclusion of primary molars, enamel hypoplasia, ectopic eruption of the first molar, supernumerary teeth, and palatal displacement of maxillary canines were included in the study and it has been found that infraocclusion of primary molars, small maxillary lateral incisors, enamel hypoplasia, and palatal displacement of the maxillary canines were significantly associated with hypoplasia of second premolar.^[14] The fact that genetic mechanisms are responsible for the concurrent occurrence of different dental anomalies is further exemplified by the finding of 'exaggerated distoangular malposition of the unerupted mandibular second premolars in children with agenesis of the contralateral mandibular second premolars'.^[15] MSX1and MSX2 genes have been identified as the ones determining the morphology of teeth. These genes were previously the homeostatic Hox 7 and Hox 8 genes. The absence of the third molars, second premolars and lateral incisors in dentitions is the result of an attempt to reducetooth volume. This in turn is in response to the change from raw unprocessed diet to refined and prefixed foods. There is a play of both evolutionary and environmental factors in the occurrence of hypodontiaof these teeth.[8]

CLINICAL SIGNIFICANCE

Though it is accepted that root resorption increases with age, it is advisable to leave the deciduous second molar in its place if it is found retained (Fig. 1). Nordqvist *et al.* reported that the deciduous second molars have a good prognosis but also stated that there is a direct correlation between root resorption and age in these teeth. In a study conducted on a restricted sample the second primary molars showed no evidence of exfoliation till the age of 20 years though resorption was evident initially.^[16] In another

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study Ith-Hansen and Kjaer observed that primary molars remain in situ without any degenerative changes for 15 years after the age for exfoliation. They have not however discussed the status of these teeth beyond this period.^[17] Rune and Sarnäs observed that root resorption remained unaltered in about 50% of retained deciduous molars and that submergence of roots is not dependent on the stage of root resorption. Hence in cases of agenesis of the second premolar, they recommended surgical or prosthetic replacement when growth of dentition has been completed or considering retaining the residual space as an alternative.^[18]

CONCLUSION

Agenesis of mandibular second premolars is found to occur second only to the third molars. This anomaly is caused by both genetic and environmental factors. It is seen that certain other developmental disturbances are also determined by the same genes that cause the absence of mandibular second premolars and may be manifested concurrently. Further research is required to unravel the genetic mechanisms involved so that occurrence of such other anomalies too may be anticipated when mandibular second premolars are missing. Necessary steps may be taken for comprehensive management.

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