Original Article

Malposition of Unerupted Mandibular Second Premolar in Children with Palatally Displaced Canines

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ABSTRACT

Objective: To test the hypotheses that (1) the distal angulation of unerupted mandibular premolar (MnP2) is significantly greater in children with palatally displaced canines (PDC) than in those in a control sample; and (2) delayed tooth formation is significantly more frequent in children with both malposed MnP2 and PDC than in children with PDC only.

Materials and Methods: We examined retrospectively panoramic radiographs from 43 patients with PDC who had no previous orthodontics. A control sample consisted of age- and sex-matched patients. The distal angle formed between the long axis of MnP2 and the tangent to the inferior border was measured. Dental age was evaluated using the Koch classification.

Results: A significant difference was observed between the mean inclination of the right side MnP2 in the PDC group (75.4 degrees) and that of the control group (85.8 degrees). This difference was highly statistically significant (P < .0001). The same evaluation was carried out for the left side, with similar results. The average dental age was found to be delayed in patients who showed both abnormalities (malposed MnP2 and PDC) compared with patients who showed the PDC anomaly only.

Conclusion: Both hypotheses are retained. Statistically, PDC and MnP2 malposition are significantly associated suggesting a common genetic etiology, despite taking place on opposite jaws. While the presence of PDC or MnP2 anomaly has been associated with a delay in tooth formation, we find the presence of *both* anomalies to show a more profound delay. Our findings suggest a delay in tooth formation as a possible common genetic mechanism for these 2 malposition anomalies. (*Angle Orthod.* 2009;79:796–799.)

KEY WORDS: Malposition; Dental anomalies; Mandibular second premolar; PDC, Genetic etiology; Late development

INTRODUCTION

Orthodontists treat malposed teeth. As orthodontists, we are interested in knowing what causes teeth to assume abnormal positions during their development. To gain this knowledge, we study malposition anomalies. In a recent such study, it was discovered that exaggerated distoangular malposition of the unerupted mandibular second premolar (MnP2) was associated with agenesis of its antimere.¹ This finding relates the MnP2 malposition to a group of tooth development abnormalities of possible common genetic origin, including agenesis (hypodontia), peg-shaped maxillary lateral incisors, small maxillary lateral incisors, palatally displaced canines (PDC), infraocclusion of primary molars, and transpositions of various teeth.^{2–8} Accumulated evidence on associations among this group go well beyond coincidence,⁸ suggesting that they are part of a broader genetically related pattern of dental anomalies.

A genetic basis of PDC is widely acknowledged.^{4,9,10} Interestingly, delayed tooth formation was reported both in children with PDC^{11,12} and in children with malposition of MnP2.¹³ This study was undertaken to test the hypotheses that (1) the distal angulation of the MnP2 is significantly greater in children with PDC than in age- and sex-matched controls, and (2) delayed tooth formation is a significantly more frequent finding

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in children with both malposed MnP2 and PDC than in children with PDC but with a normal inclination of MnP2.

MATERIALS AND METHODS

Two samples, an experimental group and a control group, were selected from the pretreatment records of patients. The experimental sample consisted of 43 patients (15 males and 28 females). Age ranged from 8 to 14 years (mean, 11.5; standard deviation [SD], 1.4). Criteria for inclusion in this sample included (1) unilateral or bilateral PDC, (2) no previous orthodontic treatment, (3) mandibular deciduous second molars present, and (4) development of the MnP2 tooth bud in stages D to G of tooth formation, according to the classification of Koch et al.¹⁴ Unerupted stage D is defined with crown formation completed down to the cementoenamel junction, unerupted stage E is defined with root length smaller than crown length, unerupted stage F is defined with root length equal or larger than crown length, and unerupted stage G is defined with walls of the root canal parallel and the root apex still partially open. Of 43 patients with PDC, 25 patients had bilateral PDC, 10 had PDC on the right side only, and 8 had PDC on the left side only.

The control sample consisted of 43 patients with normally erupting canines and was collected from the same orthodontic patient pool to match age (rounded to half year) and sex in the study group.

We developed a unique method by which to measure the inclination of the MnP2.1 In both samples, panoramic radiographs were used to trace each MnP2, along with the neighboring mandibular first molar, the deciduous second molar, and a tangent to the inferior border of the mandibular body on that side. The long axis of the MnP2 was determined as the line connecting the uppermost point of the pulp with the point bisecting the distance between the mesial and distal points of the apex. A protractor was used to measure the distal angle formed between the long axis of the MnP2 and the line drawn tangent to the inferior border of the mandible. Figure 1 shows a typical drawing with assigned lines and the resulting angle. All tracings were made independently by a single examiner using 0.003 inch frosted acetate paper and a 0.5 mm pencil.

Descriptive statistics, including mean, standard deviation, and range, were calculated for the unerupted MnP2 angles measured in the experimental and the control groups. The significance of the differences between compared means was evaluated using the Student *t*-test for paired samples. The significance level was set at P < .05. Some patients had unerupted MnP2 on both sides. However, because one may not



Mandibular Plane

Figure 1. The distal angle between the long axis of the mandibular second premolar and the tangent to the lower border of the mandible defined on a typical drawing of the relevant part of an orthopanto-mogram.

include more than 1 data point per patient in the same statistics, the question of which side to choose arises, as well as whether this arbitrary choice influences the result and how. To avoid any possible inconsistency while showing all the data, we collected 2 independent sets of data: one for all the right sides of the sample group, and another for the left sides of the same group, each matched with the same side in the control sample.

To quantify the error of the method, a second set of data was traced and measured 1 month later by the same examiner. Standard deviations calculated for 2 repeated measurements of 2 tracings of 6 different panoramic roentgenograms were used as intraexaminer error. This procedural error was found to be 1.0 degree, within reasonable limits in the context of this study.

To test the second hypothesis, we had to define the developmental stage of MnP2 and single out patients with malposed MnP2. The developmental stage of MnP2 was evaluated using the Koch classification.¹³ MnP2 malposition was defined as the distal angle (between the long axis of MnP2 and the tangent to the

Table 1. Interpatient Test: Sample vs. Control-Right Sidea,b

Group	Ν	Range	Mean	SD	t-Test Value	P Value
Right side MnP2 in the experimental sample (PDC) Right side MnP2 in the control sample (reference)	40 40	51.0–108.0 71.0–98.0	75.4 85.8	10.2 6.2	-5.53	<.0001

^a Comparison of distoangular malposition of the MnP2 (ie, distal angle formed at intersection of long axis of unerupted MnP2 and tangent to inferior border of the mandible) in degrees, between the right side MnP2 in the PDC sample and that in the control sample.

^b MnP2 indicates unerupted mandibular second premolar tooth; PDC, palatally displaced canine.

Table 2.	Interpatient	Test: Sample	vs. Control-	-Left Sidea,b
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Group	Ν	Range	Mean	SD	t-Test Value	P Value
Left side MnP2 in the experimental sample (PDC) Left side MnP2 in the control sample (reference)	37 37	61.0–97.0 76.0–97.0	77.9 85.1	8.5 4.5	-4.54	<.0001

^a Comparison of distoangular malposition of the MnP2 (ie, the distal angle formed at the intersection of the long axis of unerupted MnP2 and tangent to the inferior border of the mandible) in degrees, between the left-side MnP2 in the PDC sample and the control sample. ^b MnP2 indicates unerupted mandibular second premolar tooth; PDC, palatally displaced canine.

lower border of the mandible) when it was smaller than 75 degrees. This value is about the mean angle of malposed MnP2, as observed in previous studies.^{1,15}

RESULTS

Table 1 shows descriptive statistics of the right-side MnP2 in the experimental group and the same side in the age- and sex-matched paired control group. Of 43 patients with PDC, 40 had an unerupted MnP2 on the right side. The mean distal inclination of the MnP2 in the right side of the experimental sample was 75.4 degrees, compared with a mean of 85.8 degrees obtained for the same side in the matched control group. The mean increase of 10.4 degrees in the distoangular malposition of the developing MnP2 in PDC patients was highly statistically significant (P < .0001).

Table 2 shows the descriptive statistics of the leftside MnP2 in the experimental group and the same side in the age- and sex-matched paired control group. Of 43 patients with PDC, 37 had an unerupted MnP2 on the left side. The mean distal inclination of the MnP2 in the left side of the experimental sample was 77.9 degrees, compared with a mean of 85.1 degrees obtained for the same side in the matched control group. The mean increase of 7.2 degrees in the distoangular malposition of the developing MnP2 in PDC patients was highly statistically significant (P < .0001).

Thus, the first hypothesis, that the distal angulation of the MnP2 is significantly greater in children with PDC than in age- and sex-matched controls, is retained. The difference between sides within the PDC sample calls for an intrapatient comparison to test whether this difference is of significance. Using a paired *t*-test, we found no significant difference between the right side and the left side. Pearson correlation was found to be 0.57, significant at the 0.01 level (P = .0004).

The distribution of the MnP2 dental developmental

stage in the PDC sample according to Koch classification was as follows: Of 23 patients showing MnP2 malposition, 61% were at stage E and 39% at stage F, while out of 20 patients showing normal inclination of the MnP2, 25% were at stage E, and 75% were at stage F. These results show that the average dental age is delayed in patients who show both abnormalities (malposed MnP2 and PDC) compared with patients who show PDC anomaly but with normal inclination of MnP2. Our second hypothesis that delayed tooth formation is a significantly more frequent finding in children with both malposed MnP2 and PDC is thus retained.

DISCUSSION

This study was designed to test the null hypothesis that angular malposition of unerupted MnP2 is not directly associated with PDC. The results suggest a statistically significant association between these 2 conditions.

The palatally displaced canine is a maxillary dental anomaly, whereas MnP2 is a mandibular anomaly. Hence common mechanical cause is unlikely. The absence of a shared mechanical cause suggests association through a common genetic disorder. Peck et al¹⁶ have already suggested that the homeobox gene MSX1 may be involved in the genetic control of PDC. The association of MSX1 with agenesis¹⁷ and with clefting¹⁸ has been established in genetic linkage analyses. Both agenesis and clefting have been shown to be associated with the MnP2 malposition anomaly.^{1,15} Results of this work, along with results from Shalish et al,^{1,15} associate the MnP2 angulation anomaly with PDC, agenesis, and clefting, suggesting the MnP2 anomaly is a variable in a genetically related group of dental anomalies likely to be associated with MSX1 mutations. It is likely that the MnP2 anomaly may appear in combination with any other of these inter-associated anomalies (eg, infraocclusion, mesially ectopic maxillary first molar, tooth transposition, tooth rotation, tooth size reduction, peg-shaped maxillary lateral incisor), perhaps because all of these anomalies are caused by the same mechanism. What could be this mechanism?

Delayed tooth formation was reported in children with clefting,¹⁹ with PDC,^{11,12} and with malposition of MnP2.¹³ It therefore seems possible that the common mechanism is a delay in tooth formation. If this is correct, one should expect a longer delay to increase the likelihood of anomalies and thereby the likelihood that more than 1 anomaly will be observed in the same patient. This means that children who show more than 1 anomaly should also show a greater delay in tooth formation. Indeed, an average greater delay was confirmed in this study in children showing both PDC and MnP2 anomalies, compared with children showing PDC but with a normal inclination of MnP2.

CONCLUSION

- PDC and MnP2 malposition anomalies are significantly statistically associated, despite their taking place on opposite jaws, suggesting a common genetic etiology.
- Although the presence of PDC or MnP2 anomaly has been associated with a delay in tooth formation, we find the presence of both anomalies to show a more profound delay.
- These findings suggest a delay in tooth formation as a possible common genetic mechanism for these 2 malposition anomalies.

REFERENCES

- Shalish M, Peck S, Wasserstein A, Peck L. Malposition of unerupted mandibular second premolar associated with agenesis of its antimere. *Am J Orthod Dentofacial Orthop.* 2001;121:53–55.
- Alvesalo L, Portin P. The inheritance pattern of missing, peg-shaped and strongly mesio-distally reduced upper lateral incisors. *Acta Odontol Scand.* 1969;27:563–573.
- 3. Garn SM, Lewis AB. The gradient and the pattern of crown-

size reduction in simple hypodontia. *Angle Orthod.* 1970;40: 51–58.

- Peck S, Peck L, Kataja M. The palatally displaced canine as a dental anomaly of genetic origin. *Angle Orthod.* 1994; 64:249–256.
- 5. Peck S, Peck L, Kataja M. Prevalence of tooth agenesis and peg-shaped maxillary lateral incisor associated with palatally displaced canine (PDC) anomaly. *Am J Orthod Dentofacial Orthop.* 1996;110:441–443.
- Peck L, Peck S, Attia Y. Maxillary canine–first premolar transposition, associated dental anomalies and genetic basis. *Angle Orthod.* 1993;63:99–109.
- 7. Symons AL, Stritzel F, Stamation J. Anomalies associated with hypodontia of the permanent lateral incisor and second premolar. *J Clin Ped Dent.* 1993;17:109–111.
- 8. Baccetti T. A controlled study of associated dental anomalies. *Angle Orthod.* 1998;68:267–274.
- 9. Zilberman Y, Cohen B, Becker A. Familial trends in palatal canines, anomalous lateral incisors, and related phenomena. *Eur J Orthod.* 1990;12:135–139.
- Pirinen S, Arte S, Apajalahti S. Palatal displacement of canine is genetic and related to congenital absence of teeth. *J Dent Res.* 1996;75:1742–1746.
- Becker A, Chaushu S. Dental age in maxillary canine ectopia. Am J Orthod Dentofacial Orthop. 2000;117:657–662.
- Chaushu S, Sharabi S, Becker A. Dental morphologic characteristics of normal versus delayed dentitions with palatally displaced canines. *Am J Orthod Dentofacial Orthop.* 2002; 121:339–346.
- Wasserstein A, Brezniak N, Shalish M, Heller M, Rakocz M. Angular changes and their rates in concurrence to developmental stages of the mandibular second premolar. *Angle Orthod.* 2004;74:332–336.
- 14. Koch G, Modeer T, Poulsen S, Rasmussen P. *Pedodontics: A Clinical Approach.* Copenhagen: Munksgaard; 1991:60.
- Shalish M, Will LA, Shusterman S. Malposition of unerupted mandibular second premolar in children with cleft lip and palate. *Angle Orthod.* 2007;77:1062–1066.
- Peck S, Peck L, Kataja M. Concomitant occurrence of canine malposition and tooth agenesis: evidence of orofacial genetic fields. *Am J Orthod Dentofacial Orthop.* 2002;122: 657–660.
- Vastardis H, Karimbux N, Guthua SW, Seidman JG, Seidman CE. A human *MSX1* homeodomain missense mutation causes selective tooth agenesis. *Nat Genet.* 1996;13:417– 421.
- Van den Boogaard MJ, Dorland M, Beemer FA, van Amstel HK. MSX1 mutation is associated with orofacial clefting and tooth agenesis in humans. Nat Genet. 2000;24:342–343.
- Ranta R. A review of tooth formation in children with cleft lip/plate. Am J Orthod Dentofacial Orthop. 1986;90:11–18.