Original Article

Dentoskeletal Features Associated with Unilateral or Bilateral Palatal Displacement of Maxillary Canines

Raffaele Sacerdoti, DDS, PhD[a]; Tiziano Baccetti, DDS, PhD[b]

Abstract: The aim of the present study was to analyze the prevalence and distribution of palatally displaced maxillary canines (PDC) in a large orthodontic population, and to investigate the associations between PDC, craniofacial features, and other dental anomalies such as aplasia or small-sized upper lateral incisors. An initial sample of 5000 subjects was evaluated. The reference values were calculated in a control group of 1000 subjects that was extracted from the initial sample. Chi-squared tests were used for statistical comparisons. The prevalence rate of PDC was 2.4%, with a male-to-female ratio of 1:3. PDC subjects with low angle vertical relationships showed a significantly high prevalence rate (60.2%). Unilateral PDC was significantly associated with aplasia of upper lateral incisors, whereas bilateral PDC was associated with aplasia of third molars. PDC showed reciprocal significant associations with bilateral small-sized upper lateral incisors. None of the three hypotheses offered in support of the ''guidance theory'' in the etiology of PDC were corroborated by the findings of the present study. The occurrence of other dental anomalies concurrent with PDC, sex differences, and the bilateral expression of PDC, all confirm the genetic component in the etiology of this tooth disturbance. (*Angle Orthod* 2004;74:725–732.)

Key Words: Palatally displaced canine, Impacted canine, Dental anomalies, Aplasia of upper lateral incisors, Small-sized upper lateral incisors, Hypodivergency

INTRODUCTION

The palatal eruption or the impaction of the maxillary permanent canine is an important chapter of oral pathology and represents frequently faced problems in clinical orthodontics. Two major theories have been proposed to explain the occurrence of palatally displaced maxillary canines (PDC), ie, the ''guidance'' theory and the ''genetic'' theory. According to the guidance theory, local conditions are responsible for the displacement of the canine.[1–6] While erupting, the canine lacks the guide that, in normal conditions, would be provided by the root of the lateral incisor because of hypoplasia or aplasia of this tooth. The genetic theory assigns the eruption anomaly of the upper permanent canine to a multifactorial complex that controls the expression of other, possibly concurrent, tooth anomalies.[7] Peck et al[7] have also indicated multiple evidential categories for the genetic origin of PDC, ie, familial occurrence,[8] bilateral occurrence (17–45%), sex differences (indicating involvement of the sexual chromosomes), differences in prevalence rates among different populations, and increased occurrence of other concomitant dental anomalies.

The search for associated dental anomalies is one of the most relevant methods to investigate into the genetic determinants of PDC.[7,9] The spectrum of possible associations among tooth anomalies had been studied by Hoffmeister between 1975 and 1985.[10] The following manifestations were found over three generations of a family: multiple missing teeth (aplasia of upper lateral incisors included), peg-shaped incisors, ectopic eruption of maxillary first permanent molars, and intraosseous displacement of maxillary canines.

In 1992 Bjerklin et al[11] investigated the associations among four tooth and eruption disturbances (ectopic eruption of first molars and of maxillary canines, infraocclusion of primary molars, and aplasia of premolars). The findings indicated the presence of significant reciprocal associations. These results were interpreted supporting the hypothesis of a common, presumably hereditary, etiology for the studied tooth disturbances, each disturbance having incomplete penetrance. A very high prevalence of associated tooth anomalies (70%) was calculated by Baccetti in 1993 in a sample of 169 inherited syndromes presenting with tooth disturbances, strongly suggesting the possibility of genetic

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relationships between tooth number, size, shape, and structure characteristics.[12] These relationships have been confirmed further in the studies by Baccetti[13] in 1998 and by Leifert and Jonas[14] in 2003. The existence of associations between different tooth anomalies is not only important from an etiologic point of view but also relevant clinically because the early diagnosis of one anomaly may indicate an increased risk for later appearance of others. However, information is not definitive about the differential role played by aplasia or small-sized upper lateral incisors (SSI) in subjects with PDC, especially with reference to a possible evaluation of the guidance theory.

Canine malpositions and agenesis of at least one tooth are abnormalities known to occur together frequently. Peck et al[15,16] evaluated the site specificity of tooth agenesis associated with the occurrence of different categories of canine malpositions. PDC appeared to be associated with a significantly increased prevalence for aplasia of at least one third molar. This type of dental anomaly could be ascribed to the so-called posterior orofacial field, ie, a condition of increased susceptibility to developmental defects in the distal elements of a dental series.[17,18]

The correlations between PDC and dentoskeletal characteristics in the sagittal plane (molar relationships and sagittal maxillomandibular discrepancy) have been described in the past.[19–21] No significant associations with any class I, II, or III craniofacial patterns have been reported. The literature does not provide information regarding the vertical skeletal relationships in association with PDC. However, an increased prevalence of an occlusal deep bite characteristic has been described in PDC subjects.[14]

The aim of the present study was to analyze the prevalence and the distribution of palatal displacement of the maxillary canine in a large orthodontic population to provide evidence concerning the existence of significant reciprocal associations between PDC, skeletal features, and other dental anomalies such as aplasia or small-sized upper lateral incisors and to indicate the etiological and clinical relevance of such associations.

The specific objectives of the present study were to:

- determine the prevalence and the sex distribution of PDC in a large orthodontic population;
- analyze site-specificity of tooth aplasia concurrent with PDC, as well as the craniofacial skeletal characteristics associated with PDC;
- evaluate the guidance theory in the etiology of PDC;
- assess the significance and reciprocity of the associations between PDC and other types of tooth disturbances involving the upper lateral incisor primarily.

MATERIALS AND METHODS

An initial sample of 5000 subjects (2347 males and 2653 females), 7 to 17 years old, from the files of the Department of Orthodontics of the University of Florence was examined

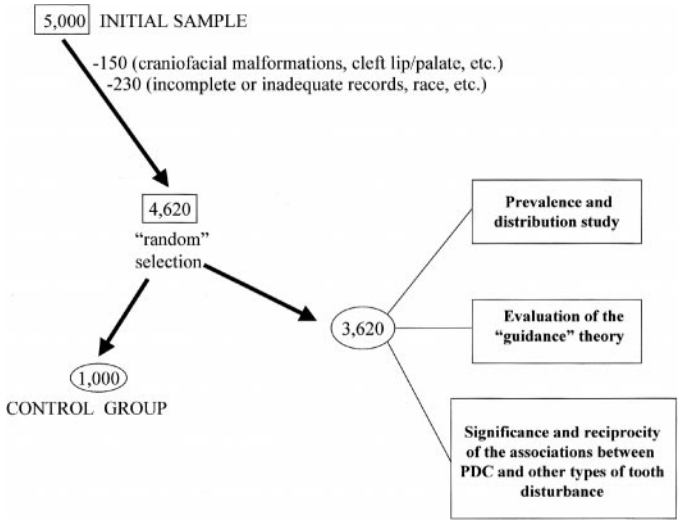

FIGURE 1. Description of study groups (numbers refer to subjects).

(Figure 1). All subjects were observed before any orthodontic treatment. Dental casts, intraoral photographs, and radiographic material (panoramic X-ray, lateral cephalograms) of all subjects were examined. A group of 150 subjects was excluded from the initial sample because of the presence of complex craniofacial malformations, cleft lip or palate (or both), sequelae of traumatic injuries to the teeth, odontomas, or cysts. Another 230 subjects were excluded because of incomplete or inadequate records, racial diversity (only Caucasian subjects were included in the study), familial relationships with other examined subjects, and severe tooth crowding within the dental arches.

The remaining sample of 4620 subjects was divided randomly into two groups. The first group of 1000 subjects contained 468 males and 532 females and was used as a control group. The ''reference'' prevalence rates for the examined parameters were calculated for this group. The remaining 3620 subjects comprised the sample from which the experimental groups were derived.

In addition to sex distribution and age, the following dental and craniofacial parameters were examined. Palatal displacement of maxillary canines (Figure 2): the intraosseous palatal position of the upper permanent canines, unilaterally or bilaterally, was evaluated on the basis of panoramic and periapical radiographs. Small-sized maxillary lateral incisors: unilaterally or bilaterally (Figure 2), defined as a severe crown-size reduction of the lateral incisors, in some cases associated with a certain degree of narrowing in diameter from the cervix to the incisal edge (peg-shaped lateral incisors).[22] A milder version of the latter form was considered as well in those cases showing a slight tapering of the crown from gingival to incisal (''screwdriver-head'' crown of the maxillary lateral incisor). Aplasia of maxillary lateral incisors, second premolars, and third molars (Figure 2): the diagnosis, unilaterally or bilaterally, was made on dental casts and panoramic radiographs. Craniofacial skel-

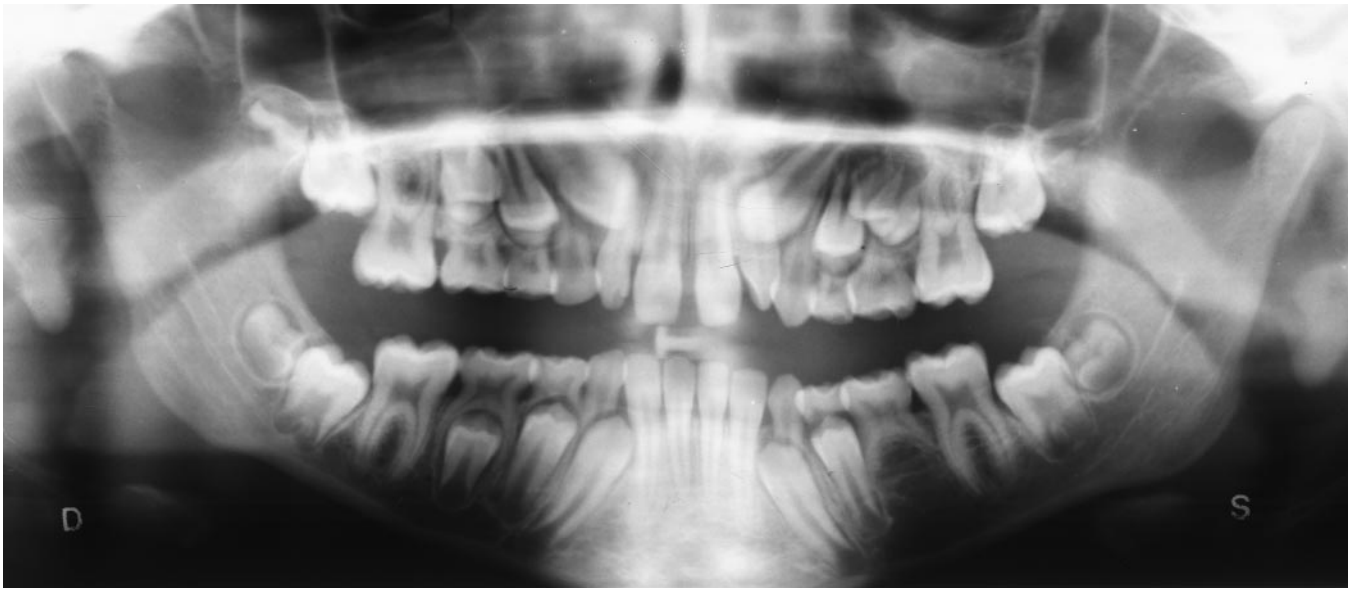

FIGURE 2. Bilateral palatally displaced maxillary canines, bilateral small-sized upper lateral incisors, and aplasia of lower left second premolar in a 9-year-old female subject.

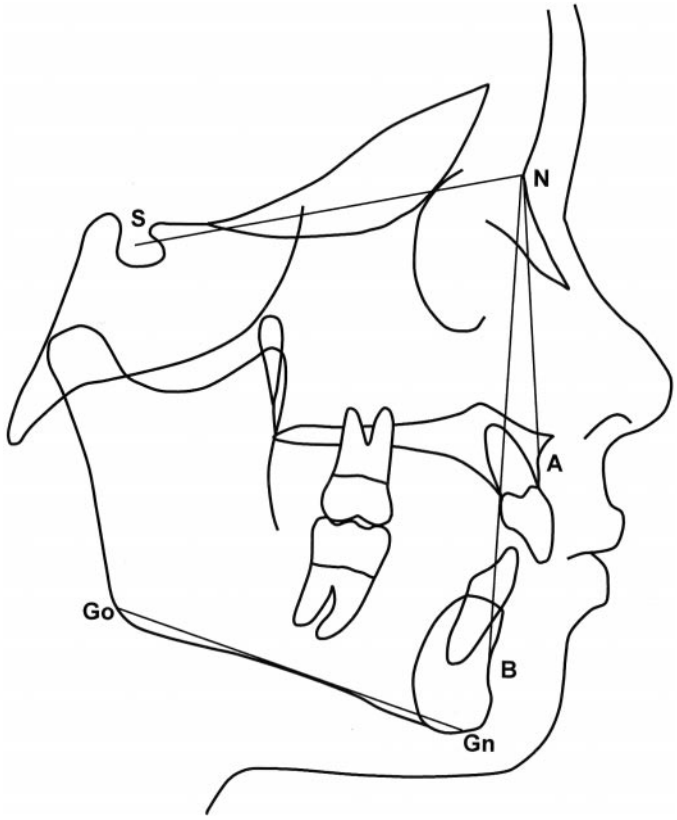

FIGURE 3. Cephalometric measurements for sagittal and vertical skeletal relationships.

etal relationships (Figure 3): the evaluation of the skeletal relationships was performed on the lateral cephalograms using the angular measure A-N-B for the sagittal relationships and the angular measure S-N/Go-Gn for the vertical relationships.

Reproducibility of the diagnosis was assessed by reexamining the records of 100 subjects one month after the first examination by one single operator (Dr Sacerdoti). Reproducibility was 100% for all dental anomalies except for small-sized maxillary lateral incisors (94%). Accuracy of cephalometric measurements was tested by means of Kappa test, and it was 0.96. Measurement error for the cephalometric angles was smaller than 1°.

The study was divided into the following three sections with respect to the three specific aims of the research design.

Prevalence and distribution study

The objective of this section of the study was the assessment of: prevalence rate of PDC; unilateral-bilateral ratio of PDC; male-female ratio of PDC; sagittal skeletal relationships in PDC subjects, defined as skeletal class I when A-N-B values ranged between 0° and 4°, skeletal class II when they were greater than 4°, and skeletal class III when they were smaller than 0°; vertical skeletal relationships in PDC subjects, defined as normodivergent when S-N/Go-Gn values ranged between 27° and 37°, hypodivergent when they were smaller than 27°, hyperdivergent when they were greater than 37°; prevalence rates of aplasia of third molars, second premolars, and upper lateral incisors in PDC subjects. This ratio was calculated by taking into consideration first, the total number of the PDC cases, then the unilateral ones and, finally, the bilateral ones with the goal of testing how PDC associates with the specific expression of ''posterior-field hypodontia.''[16]

These prevalence and distribution rates were compared statistically with those assessed in the control group.

Evaluation of the guidance theory

This section was aimed at testing the guidance etiologic theory proposed by Becker and coworkers[2–6] by means of a study of the associations between PDC and upper lateral incisor features. From the experimental sample of 3620 subjects, a group of 138 subjects was selected who presented at least one or more of the following anomalies: PDC, aplasia of the upper lateral incisors (AI), and small size of upper lateral incisors (SSI). A clinical substantiation of the guidance theory would have fulfilled the following requirements: prevalence rate for homolateral unilateral small-sized lateral incisors (SSIu) significantly higher than the prevalence rate for bilateral small-sized lateral incisors (SSIb) in subjects who presented with unilateral PDC (PDCu); prevalence rate for bilateral AI (AIb) significantly higher in subjects with bilateral PDC (PDCb) than PDCu; and prevalence rate of homolateral unilateral AI (AIu) significantly higher than contralateral unilateral aplasia in subjects with PDCu.

Significance and reciprocity of the associations between PDC and other developmental disturbances of the upper lateral incisors

The aim of the third section of the study was to evaluate the existence of significant reciprocal associations between PDC and different types of dental anomalies, with special emphasis on the features of the upper lateral incisors, and to side specificity of the anomaly.

Six types of dental anomalies were considered: PDCu; PDCb; AIu; AIb; SSI—unilateral (SSIu); and SSI—bilateral (SSIb).

Six groups of 20 subjects with one primarily diagnosed type of tooth anomaly each were extracted from the experimental group (3620 subjects). The individuals belonging to one of the six groups were not concomitantly present in any of the other five groups. The prevalence rates of the five other types of dental anomalies in association with the primarily diagnosed dental anomaly were calculated for each of the six groups (according to the method of Bjerklin et al[10] and Baccetti[11]). The prevalence rates of dental anomalies associated with the primarily diagnosed anomaly in the six groups of 20 subjects each were compared statistically with the prevalence rates in the control group of 1000 subjects. Associations between anomalies that were reciprocally exclusive were not considered (eg, bilateral SSIu in the group with AIb).

To investigate further into the specific role played by aplasia or SSIu in subjects with PDC, other associations and prevalences were calculated: reciprocal associations between PDC, SSI, and AI, regardless of the unilateral-bilateral expression of the anomaly; and reciprocal associations between PDC and AIu, AIb, SSIu, SSIb.

TABLE 1. Prevalence Rates of Examined Tooth Anomalies and Other Features in the Control Group (n = 1000)

Aplasia of lateral incisors	4.4%
Aplasia of lateral incisors (unilateral)	2.4%
Aplasia of lateral incisors (bilateral)	2.0%
Aplasia of second premolars	3.6%
Aplasia of third molars	20.7%
Small size of lateral incisors	3.8%
Small size of lateral incisors (unilateral)	2.4%
Small size of lateral incisors (bilateral)	1.4%
Palatally displaced maxillary canines	2.4%
Palatally displaced maxillary canines (unilateral)	1.8%
Palatally displaced maxillary canines (bilateral)	0.6%
Males	46.8%
Females	53.2%
Hypodivergent	33.0%
Hyperdivergent	21.0%
Normodivergent	39.0%
Skeletal class I	48.0%
Skeletal class II	32.0%
Skeletal class III	20.0%

Statistical analysis

All comparisons of prevalence rates were carried out by means of chi-squared tests (SAS 6.12, Statistic Analysis System Institute, Cary, NC). Yates' correction was applied when appropriate. Level of significance was set at $P < .05$.

RESULTS

The prevalence rates of tooth anomalies and other features of the control group are shown in Table 1.

Prevalence and distribution study

The prevalence of PDC in the experimental group was 88 subjects (2.43%). Unilateral-bilateral ratio of PDC was 58:30 subjects. Therefore, the prevalence rate for PDCb was 34%. The M-F ratio in PDC subjects was 23:65, which approximates a M-F ratio of 1:3.

The prevalence rates for sagittal skeletal relationships in PDC subjects were 15 class III subjects (17%), 27 class II subjects (31%), and 46 class I subjects (52%) (Table 2). These data reproduce closely the standard prevalence rates for the three sagittal skeletal classes in orthodontic populations, as shown by the rates in the control group (Table 1).

The prevalence rates for vertical skeletal relationships in PDC subjects were 53 hypodivergent subjects (60.2%), 13 hyperdivergent subjects (14.8%), and 22 normodivergent subjects (25%) (Table 2). The prevalence rate for hypodivergent subjects in the control group was significantly smaller (33%) (Table 1).

The prevalence rate for aplasia of third molars was significantly greater in PDCb subjects than in the control

TABLE 2. Prevalence and Distribution Study (n = 88). Statistical Analysis

Primarily Diagnosed Dental Anomaly	Associated Dental Anomaly/Feature	Prevalence (%)	Prevalence in the Control Group (%)	χ^2
PDC[a]	Third molars aplasia	21.5	20.7	0.03
PDC	Second premolars aplasia	7.9	3.6	2.97
PDC	Upper lateral incisor aplasia	11.9	4.4	2.75
PDCb	Third molars aplasia	36.6	20.7	4.4*
PDCb	Second premolars aplasia	10	3.6	1.75
PDCb	Upper lateral incisor aplasia	6.6	4.4	0.02
PDCu	Third molars aplasia	13.7	20.7	1.61
PDCu	Second premolars aplasia	6.8	3.6	0.86
PDCu	Upper lateral incisor aplasia	18.9	4.4	20.7*
PDC	Female sex	73.8	53.2	13.1*
PDC	Hypodivergence	60.2	33	26.2*

[a] PDC indicates palatal displacement of maxillary canines; PDCb, PDC bilateral; and PDCu, PDC unilateral.
* Significant comparisons with control group ($P < .05$).

group (Tables 1 and 2). The prevalence rate for AI was significantly greater in PDCu subjects than in the control group (Tables 1 and 2). The occurrence of aplasia of second premolars was similar in PDCu and PDCb subjects and in the control group (Tables 1 and 2).

Evaluation of the guidance theory

The prevalence rate for homolateral unilateral small-sized upper lateral incisors was not significantly higher than the prevalence rate for bilateral small-sized upper lateral incisors in subjects with PDCu. On the contrary, the bilateral presence of small-sized upper lateral incisors in PDCu cases was significantly more prevalent than the unilateral presence of the tooth size anomaly. A total of 35 subjects showed the association between PDCu and unilateral or bilateral small-sized maxillary lateral incisors. Of these, six subjects (17%) presented with homolateral unilateral small-sized upper lateral incisors, 26 subjects (74%) with bilateral small-sized upper lateral incisors, and three subjects (9%) with contralateral unilateral small-sized upper lateral incisors.

The prevalence rate for AIb was not higher in PDCb as compared with PDCu subjects. The prevalence rate for the two groups was identical with 2 of 30 (6.6%) PDCb subjects showing bilateral aplasia of lateral incisors and 2 of 30 (6.6%) PDCb subjects showing unilateral aplasia of lateral incisors.

The prevalence rate for homolateral AIu was not higher than contralateral unilateral aplasia in PDCu subjects. Once again, the prevalence rate for the two groups was identical: 5 of 58 (8.6%) PDCu subjects showing homolateral unilateral aplasia of upper lateral incisors, 5 of 58 (8.6%) PDCu subjects showing contralateral unilateral aplasia of upper lateral incisors. None of the three proposed hypotheses in support of the guidance theory was corroborated by the findings of the present study.

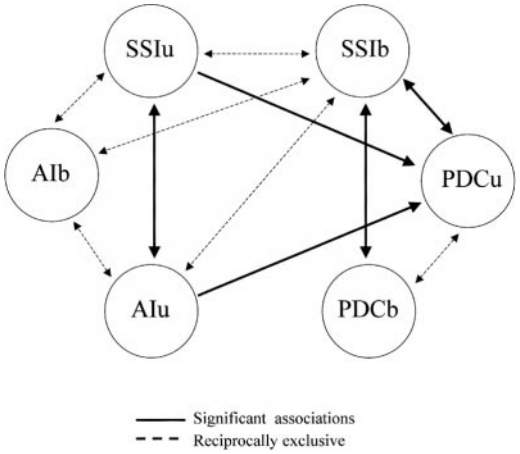

FIGURE 4. Graphical representation of the associations between unilateral and bilateral forms of examined dental anomalies.

Significance and reciprocity of the associations between PDC and other developmental disturbances of the upper lateral incisor

The results of the comparisons between the prevalence rates for the dental anomalies associated with the primarily diagnosed anomaly in the six subgroups of 20 subjects each and the prevalence rate of dental anomalies in the control group are shown in Figure 4 and Table 3.

The groups with PDCu and PDCb showed significant reciprocal associations with bilateral small-sized lateral incisors (SSIb). On the other hand, no significant association with unilateral small-sized upper lateral incisors (SSIu) was found with the exception of a significantly higher prevalence rate of PDCu in subjects with SSIu. The groups with PDCu and PDCb did not show any significant reciprocal associations with lateral incisors aplasia with the exception of a significantly higher prevalence rate of PDCu in subjects with AIu. AIu on one side of the upper arch presented

TABLE 3. Prevalence Rate and χ^2 Test for The Analysis of Associations Between Tooth Anomalies[a]

Primarily Diagnosed Dental Anomaly	PDCu		PDCb		AIu		AIb		SSIu		SSIb	
	Prev.	χ^2	Prev.	χ^2	Prev.	χ^2	Prev.	χ^2	Prev.	χ^2	Prev.	χ^2
PDCu			—	—[b]	2/20	2.01	0/20	0.49	0/20	0.49	8/20	79.3*
PDCb	—	—			1/20	0.98	0/20	0.12	0/20	0.12	12/20	365.5*
AIu	6/20	43.10*	0/20	0.00			—	—	6/20	43.10*	—	—
AIb	0/20	0.40	2/20	2.75	—	—			—	—	—	—
SSIu	5/20	28.5*	1/20	0.55	18/20	381.1*	—	—			—	—
SSIb	12/20	271.1*	8/20	120.8*	—	—	—	—	—	—		

[a] PDCu indicates palatally displaced maxillary canines, unilateral; PDCb, PDC bilateral; AIU, aplasia of upper lateral incisors, unilateral; AIb, AI bilateral; SSIu, small-sized upper lateral incisor, unilateral; and SSIu, SSI, bilateral.

[b] — indicates reciprocally exclusive.

* Significant comparisons with control group ($P < .05$).

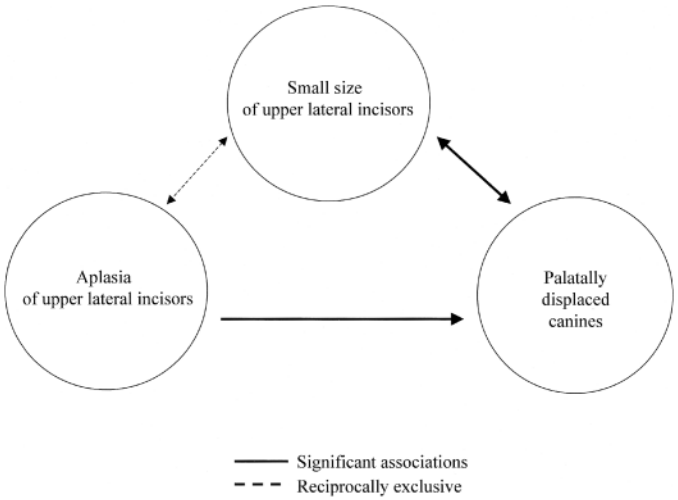

FIGURE 5. Graphical representation of the associations between palatally displaced maxillary canines, aplasia of upper lateral incisors and small-sized upper lateral incisors.

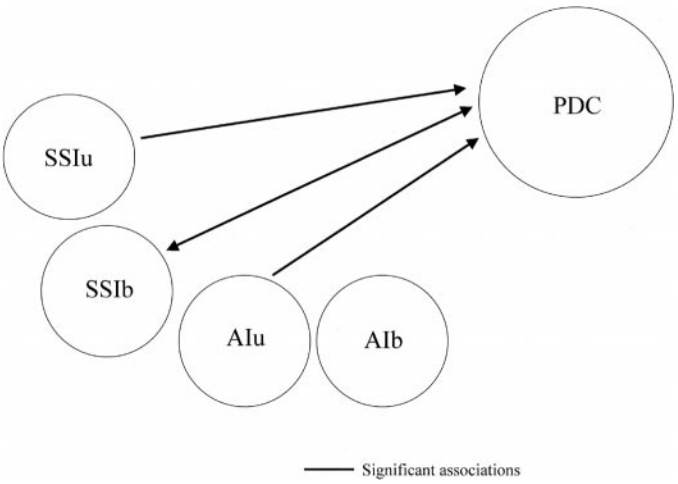

FIGURE 6. Graphical representation of the associations between palatally displaced maxillary canines and unilateral or bilateral forms of aplasia of upper lateral incisors and small-sized upper lateral incisors.

TABLE 4. Prevalence Rate and χ^2 Test for the Analysis of Associations Between PDC, AI, SSI Groups[a]

Primarily Diagnosed Dental Anomaly	PDC		AI		SSI	
	Prev.	χ^2	Prev.	χ^2	Prev.	χ^2
PDC			3/40	2.2	20/40	203.5*
AI	9/40	22.4*			—	—[b]
SSI	26/40	238.9*	—	—		

[a] PDC indicates palatally displaced maxillary canine; AI, aplasia of upper lateral incisors; and SSI, small-sized upper lateral incisors.

[b] — Reciprocally exclusive.

* Significant comparisons with control group ($P < .05$).

with a significant reciprocal association with small-sized upper lateral incisor (SSIu) on the other side.

Considering the groups regardless of the unilateral or bilateral expression of the anomaly, significant reciprocal associations between canine displacement and the size anomaly of the upper lateral incisor were found (Figure 5; Table 4). The study of the associations between PDC and AIu, AIb, SSIu, SSIb revealed statistically significant reciprocal associations between PDC and bilateral size anomaly of the upper lateral incisor, in the absence of any association with the bilateral aplasia of the same tooth (Figure 6; Table 5).

DISCUSSION

The aim of the present study was to analyze the prevalence and distribution of PDC in an orthodontic sample. PDC was examined also with regard to possible associations with other dental and craniofacial features. In particular, the associations between PDC and third molar aplasia, second premolar aplasia, upper lateral incisor aplasia, and small size of upper lateral incisors were studied. The prevalence rate of PDC was 2.4%, in agreement with previous studies on orthodontic populations.[7,8,13] However, the prevalence rates of dental anomalies in the present study do not necessarily reflect the prevalence rate of these anomalies in the general population because of the fact that the examined subjects had been referred to an orthodontic department.

PDC was bilateral in more than one-third of the sample, and the M-F ratio was 1:3. These data confirm a genetic

TABLE 5. Prevalence Rate and χ^2 Test for the Analysis of Associations Between PDC, AIu, AIb, SSIu, SSIb Groups[a]

Primarily Diagnosed Dental Anomaly	PDC		AIu		AIb		SSIu		SSIb	
	Prev.	χ^2	Prev.	χ^2	Prev.	χ^2	Prev.	χ^2	Prev.	χ^2
PDC			3/40	1.85	0/40	0.22	0/40	0.22	20/40	203.4*
AIu	6/20	43.10*								
AIb	2/20	2.32								
SSIu	6/20	43.11*								
SSIb	20/20	561.4*								

[a] PDC indicates palatally displaced maxillary canines; AIu, aplasia of upper lateral incisors, unilateral; AIb, bilateral; SSIu, small-sized upper lateral incisor, unilateral; and SSIb, SSI bilateral.

* Significant comparisons with control group ($P < .05$).

component in the etiology of this tooth malposition with a possible involvement of the sexual chromosomes.[7]

A section of the present investigation was dedicated to the analysis of the prevalence rates for tooth agenesis in PDC subjects. Peck et al[15,16] had suggested site specificity of tooth agenesis in PDC subjects, with the maxillary canine malposition associated with third molar agenesis in the absence of an increased prevalence rate for aplasia of upper lateral incisors. The present study analyzed these issues in a more detailed manner and revealed the existence of the association indicated by Peck and coworkers in subjects with PDCb, whereas PDCu was associated with AI. Different orofacial genetic fields appear to be linked to the PDCu or PDCb. The genetic mechanisms underlying PDC deserve to be elucidated further regarding the role of site specificity of associated dental anomalies vs the PDCu or PDCb phenotype of PDC.

One of the aims of the present study was to verify clinically the possible role of the size anomaly or aplasia of the upper lateral incisors as a local factor in the etiopathogenesis of PDC according to the so-called guidance theory.[2–6] The investigation model of the study of associated dental anomalies allowed the testing of three hypotheses offered to support the theory. According to the present findings, none of the three hypotheses was corroborated. PDCu was associated with unilateral small-sized lateral incisors on the PDC side of the dental arch in a very limited percentage of the cases (17%). In the vast majority of the cases (about three-quarters), PDCu was associated with bilateral small-sized lateral incisors. In 9% of the cases, unilateral small-sized lateral incisors were found opposite to the PDCu side of the dental arch. The prevalence rate for homolateral AIu was not significantly greater than the prevalence for contralateral unilateral aplasia in PDCu subjects. The percentage of subjects with AIu on the PDC side of the dental arch was only 8.6%.

Reciprocal associations between PDC and aplasia or SSI were investigated according to the methodology proposed by Bjerklin et al[11] and by Baccetti[12] for the identification of a shared genetic component in the etiology of these tooth disturbances. Specific attention was devoted to the unilateral or bilateral expression of the dental anomaly.

Both PDCu and PDCb demonstrated significant reciprocal associations with bilateral small-sized upper lateral incisors. On the contrary, no significant association was found between PDCu and PDCb and aplasia of the lateral incisors, with the exception of a significantly greater prevalence rate for PDCu in subjects with unilateral aplasia. When the three dental anomalies were taken into account regardless of unilateral or bilateral expression, both the presence of a significant reciprocal association between PDC and small-sized upper lateral incisors and the absence of a significant reciprocal association between PDC and aplasia of upper lateral incisors were confirmed. Finally, the existence of a significant reciprocal association between aplasia of the lateral incisor on one side of the upper dental arch and small-sized lateral incisor on the opposite side of the arch in the same subjects was corroborated.[12,23–25]

The relationship between PDC and craniofacial skeletal characteristics in the anteroposterior plane (sagittal maxillomandibular discrepancy) has been investigated in the past, with no significant associations with any specific craniofacial pattern (class I, II, or III) being described.[19–21] The results of the present study confirm that the distribution of categories of sagittal skeletal relationship in subjects with intraosseous malposition of the maxillary canines is very similar to standard orthodontic populations. Evidence has been gathered here, for the first time, that reveals a significant association between vertical craniofacial features and PDC. The prevalence rate for hypodivergent cases in the PDC subjects was three times greater than in control subjects.

To summarize, three of the five categories of clinical evidence proposed by Peck and coworkers[7] in support of a genetic component in the etiology of PDC have been confirmed by the results of the present study. In particular, the canine malposition is significantly associated with a size defect of the upper lateral incisor, especially in bilateral forms. Bilateral expression of PDC occurs frequently, and the prevalence of PDC is significantly greater in females than in males. According to the parameters investigated here, a triad of signs appear to be linked with PDC expression in growing subjects. More than 25% of PDC subjects examined in this study presented with: (1) female sex,

(2) hypodivergent vertical skeletal relationships, and (3) SSIb. The early recognition of the concurrence of these three characteristics may aid in the identification of those subjects who will develop a palatal displacement of the maxillary canine.

CONCLUSIONS

The PDC has shown a significant reciprocal association with SSI. Both PDCb and PDCu are significantly associated with bilateral small-sized upper lateral incisors.

PDCu exhibited a significant association with AI. PDCb was significantly associated with aplasia of third molars.

The concurrence of other dental anomalies with PDC, significant differences in sex distribution, and the high prevalence rate for PDCb confirm the genetic component in the etiology for this tooth disturbance, at least for its bilateral form.

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