Original Article

Prevalence of Tooth Transposition, Third Molar Agenesis, and Maxillary Canine Impaction in Individuals with Down Syndrome

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Abstract: Alterations in the size, morphology and number of teeth are among the many inherited disorders that have been reported in individuals with Down syndrome. By contrast, third molar agenesis and tooth transposition have been largely ignored and the prevalence of canine impaction has not been reported. The intention of this study was to quantify the occurrence of these anomalies in a group of individuals with Down syndrome, using standardized records, which included a clinical examination, dental casts, and a panoramic radiograph. The results show a notably high prevalence of third molar agenesis (74% of individuals older than 14 years), canine impaction (15%), and maxillary canine/first premolar transposition (15%), compared to published figures from random population samples. These anomalies should not be seen as separate, independent entities, but as associated phenomena. The slow rate of cell growth and a consequent reduced cell number that characterize this syndrome may be responsible for the underdevelopment of the upper jaw, the delayed dental development, the reduction in teeth number and size, and the altered path of canine eruption. No explanation, other than genetics, is immediately available to explain why the maxillary canine/first premolar transposition should represent another phenotypic expression of this trisomy. (*Angle Orthod* 2000;70:290–296.)

Key Words: Down syndrome; Third molar agenesis; Canine impaction; Transposition

INTRODUCTION

Many dental anomalies commonly seen in patients with Down syndrome have been reported in the literature over the past few years. Abnormalities in the number (fewer), size (smaller) and morphology (peg-shaped and other morphological deficiencies),[1–6] and the timing of their development (late dentition)[7,8] are constant features of this syndrome.

Among completely normal individuals, but seen elsewhere in the dental literature, these same anomalies have been shown to be linked with (a) congenital absence of third molars,[9–11] (b) palatal displacement of maxillary canines,[12–16] and (c) tooth transposition (ie, a severe disturbance of tooth order and eruptive position) that may occur at any of several specific sites in the mouth.[17]

Although the prevalence of oligodontia among patients with Down syndrome has been investigated extensively,[1,3,7,18] only one study has specifically evaluated the prevalence of congenitally missing third molars.[19] This is surprising since the absence of third molars is the most common expression of congenital absence of permanent teeth in healthy individuals and is not considered to be an isolated dental abnormality.[11] In the study concerned, the experimental sample was comprised of individuals ranging from 3 to 41 years of age in whom almost half of the third molars were described as missing. This was true even though a minimum age limit for determining congenital absence was not defined and, in the absence of information to the contrary, the sample included those individuals in the lower end of the age scale.[19]

The prevalence of maxillary canine impaction in a normal population is 1% to 3%, depending on the population studied.[20–23] To our knowledge, there has been no investigation of the prevalence of canine impaction among individuals with Down syndrome. This is also particularly surprising, since the dental anomalies so frequently seen in

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Down syndrome patients are the same anomalies known to be associated with palatal canine displacement.

Similarly, despite a wide search of the published research in this area, the present authors have not come across published reports of tooth transposition in association with Down syndrome with the single exception of an isolated, single case report in the Spanish literature.[24]

It is therefore the intention of this study to determine the prevalence of third molar agenesis, maxillary canine impaction, and tooth transposition in a group of individuals with Down syndrome.

MATERIALS AND METHODS

The sample consisted of 34 individuals with Down syndrome, 15 female patients and 19 male patients ranging in age from 11 to 24 years (mean age 18 ± 3.9 years, median age 18.5 years). One of the individuals lived in an institution for the mentally retarded, 17 lived in 3 protected hostel-like residences within the community, and 16 lived at home with their parents. A standardized set of records was obtained from each individual, including a detailed clinical examination, dental casts, and a panoramic radiograph.

All patients in the sample benefited from continuing dental care in the dental clinic at the Jerusalem Elwyn Institute for patients with disabilities and at the unit for treatment of patients with disabilities in the Department of Pediatric Dentistry and at the Center for Craniofacial Disorders in Handicapped Children, in the Department of Orthodontics, the Hebrew University-Hadassah School of Dental Medicine, in Jerusalem.

The dental records for each patient were carefully studied to exclude trauma or extraction as the cause of tooth absence among the patients in the sample. For the section of the study dealing with congenital absence of third molars, the experimental group was reduced to include only the 27 patients who were aged 14 years and over, in accordance with studies on critical age for third molar agenesis.[11]

For the purpose of this study, the terms peg-shaped and small lateral incisors, impacted tooth and palatally impacted maxillary canines followed the definitions used in our earlier work,[25,26] while maxillary canine/first premolar transposition was determined in accordance with the work of Peck et al.[17] Determination of palatal impaction of the maxillary canines was made using the method described by Chaushu et al.[27]

RESULTS

In Table 1, the 34 patients are listed by age and sex and identifies congenitally missing teeth, maxillary canine impaction, tooth transposition, and anomalous lateral incisors. Only 7 (26%) of the 27 patients over 14 years of age had all 4 third molars. Varying degrees of third molar agenesis were noted in the other 20 (74%) patients. Of these, 1 (5%) presented with a single missing third molar, 7 (35%) of the patients were missing 2 third molars, 1 (5%) was missing 3 third molars and 11 (55%) showed total absence of all 4 third molars. There was no significant difference in agenesis between the right and left sides and no sexual predilection was noted. However, more third molars were missing from the maxilla than the mandible (13:5). It is pertinent to note that in 12 (60%) of the 20 patients with missing third molars, at least 1 other tooth was absent and, in 5 (25%) of these, small or peg-shaped laterals were present. Excluding third molars, missing teeth were found in 20 patients (59% of the total sample). More teeth were missing in the maxilla than in the mandible (27:17) and the most prevalent missing tooth was the upper lateral incisor (19 out of 44 missing teeth), followed by lower incisors (10 out of 44), and the upper and lower second premolars (9 out of 44).

Impacted permanent canines were observed in 5 patients (15% of the sample), 3 of whom displayed 1 maxillary impacted canine and 2 with a single impacted canine in each jaw. In all, 5 palatally impacted canines were found in the maxilla and 2 in the mandible (2.5:1). A similar ratio of 2.5:1 indicated a female to male preference. In 3 of the patients with impacted maxillary canines, anomalous or missing laterals were also observed.

Maxillary canine/first premolar (Mx.C.P1) transposition was found in 5 patients (3 male patients and 2 female patients, 15%). Two of these were bilaterally affected, making a total of 7 transpositions. All patients with unilateral transposition exhibited an affected left side. Four of the patients showed at least 1 congenitally missing tooth, with a missing maxillary lateral incisor evident in 3. Two of the 4 patients who had a missing tooth and were over 14 years of age also revealed complete absence of the third molars. Only 1 patient with Mx.C.P1 had no further teeth missing, but displayed 2 peg-shaped laterals. Unilateral Mx.C.P1 transposition was not found with contralateral canine impaction in any of the patients.

Figures 1 and 2 are panoramic radiographs and Figure 3 shows the maxillary dental cast of 3 different patients showing a varying expression of congenital absence of third molars and lateral incisors, maxillary palatal canine impaction and Mx.C.P1 transposition.

DISCUSSION

In the present study almost 74% of the Down syndrome patients older than 14 years expressed varying degrees of third molar agenesis, compared to a 16.4% prevalence among healthy Americans.[11] In an earlier study,[19] the diagnosis of congenitally missing third molars among 129 Caucasian patients with Down syndrome, revealed a prevalence of 48%. This is a notably lower figure than in the present sample, even though their sample included about 50% of youngsters below 14 years of age, who could have

TABLE 1. Dental Anomalies in the Experimental Group of Down Syndrome

Age	Sex	Missing Third Molars	Other Missing Teeth	Impacted Canines	Mx.C.P1 Transposition	Peg-Shaped Laterals	Small Laterals
11	M	x	22		23,24		
11	M	x	17				
11	F	x	15,25,35,45,12,22,41,42	13			
12	M	x					
12	M	x			23,24/13,14	12,22	
13	M	x					12,22
13	F	x	22		23,24/13,14		
14	F	18,28					
15	M	18,28,48,38	41		23,24		
17	F	18,28,38,48	25	13			
17	F	18,38	42,32				
17	M	18,28,38,48	25			22	
17	M	18,28	22				
17	F	18,28,38,48	12,22,42,35		23,24		
17	F	18,28	12,22				
18	M	28,48,38					
18	M	18,28,48,38	15,45,41,31				12,22
19	F						
19	F	18,48,38,28	22			12	
19	M	18,28,38,48					
20	M	18,28	12,22	13,43			
20	F		12,22				
21	M		13,22,31			12	
21	M	18,28,38,48				31	
21	M	18,28,38,48					
21	M	18,38					
21	M	18,28,38,48	42				
22	F						
22	F						
22	F		12,22,42				
22	M	18,28,38,48	12,22				
22	M		13,43,33				22
22	F	18,28		13		22	12
23	F						
24	F	48		23,33			

developed third molars later, thereby reducing the number of patients with congenital absence of third molars.

Garn et al[11] postulated that third molar agenesis is not an isolated phenomenon, but rather a polymorphism related to the prevalence of other missing teeth, to the timing of tooth calcification, and to the order of tooth eruption. A delay in tooth development would lead to a crown size reduction and, if serious enough, would cause agenesis.[9–11,28]

Of the 20 individuals having at least 1 missing third molar, 12 (60%) presented with at least 1 other tooth missing and 5 (25%) with small or peg-shaped lateral incisors.

These figures are very similar to those of Garn et al,[10] who reported a 53% prevalence of ''associated'' missing other teeth among 100 healthy individuals with third molar agenesis. The present findings represent a 13-fold increase compared to the control group of patients who possessed all 4 third molars. By contrast, the present group of Down syndrome patients shows a high prevalence of missing teeth, even when third molars are present (4 out of 7 cases). Thus the frequency of missing upper laterals is elevated whether or not there is absence of other teeth.

The dental reductions seen in relation to size, shape, and number could be the expression of a known decrease in number (rather than size) of cells in many body organs due to the slower intermitotic period in trisomic cells.[29,30] This phenomenon has been held responsible for the general growth retardation in Down syndrome.[31]

Impacted teeth are those that remain unerupted in the jaw beyond the time at which they should normally be erupted,[32] and are not expected to erupt in a reasonable time. In the present study, 5 (15%) patients with Down syndrome presented with a total of 7 impacted canines.

The prevalence of maxillary canine impaction in a random population varies from less than 1% to 3%, depending on the population studied.[20–22] The prevalence in an Israeli population was found to be 1.53%.[23] Using this as a baseline means that there is approximately a 10-fold increased occurrence of impacted maxillary canines among patients

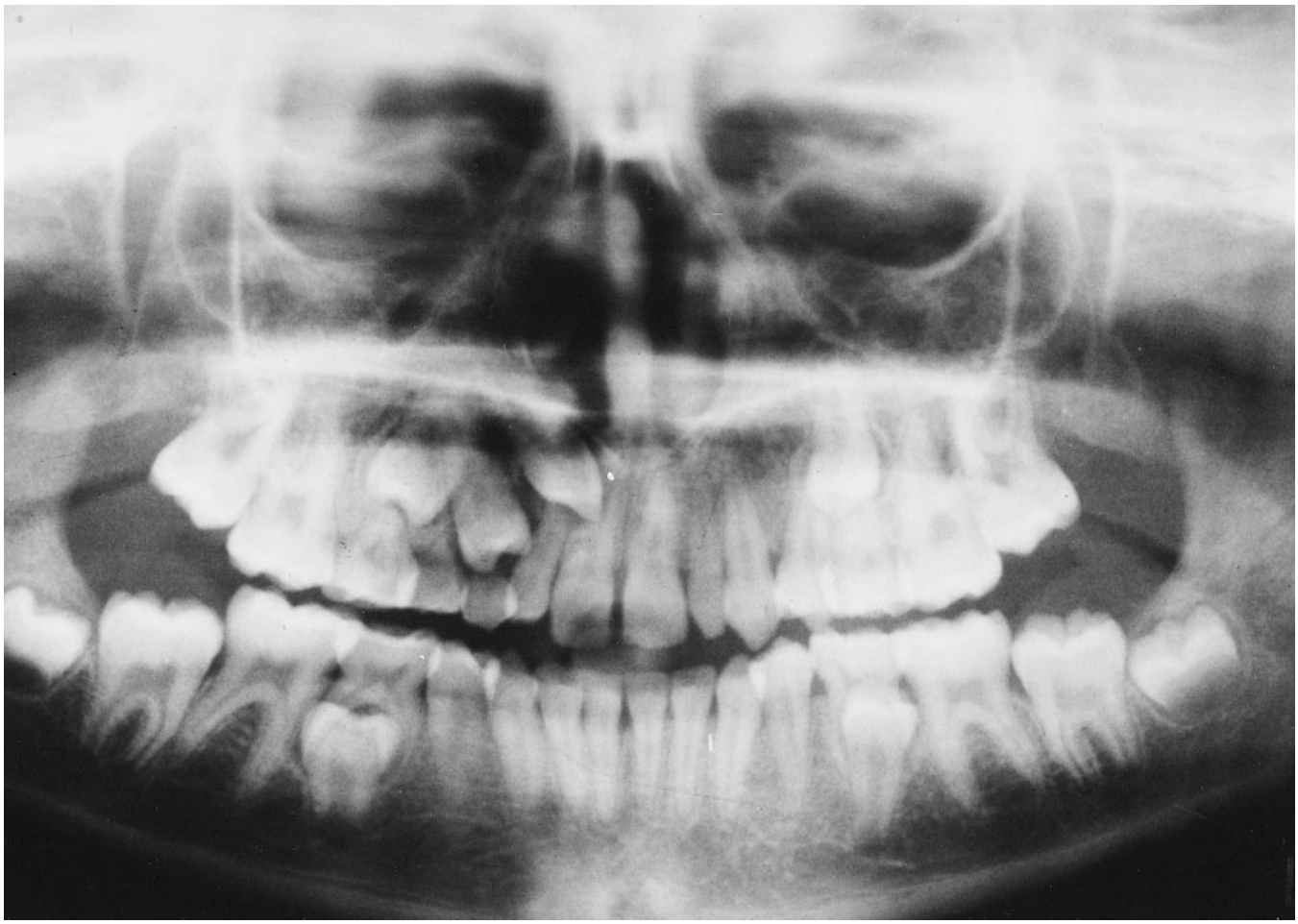

FIGURE 1. The panoramic view of a 22-year-old patient illustrating palatal impaction of the right maxillary canine, absence of maxillary third molars, small right and peg-shaped left maxillary lateral incisors.

with Down syndrome, compared to the healthy Israeli population. This elevated figure can be attributed to any or all of the following: an underdeveloped maxilla,[33] delayed dental development,[7,8,12,13] and the presence of small or missing lateral incisors.[25,33–35] Canine impaction was more prevalent among female patients, which is consistent with the findings of other studies performed among healthy individuals in the US[22] and Europe[36]. In healthy children, the permanent canines erupt considerably earlier in girls than in boys. In children with Down syndrome, this tendency occurs only in the mandible.[8] In the maxilla the canine eruption was considerably delayed in female patients, increasing the likelihood of impaction.

Canine/premolar transposition is rare in material from prehistoric, historic, and present day Homo sapiens, with a prevalence of less than 0.1% in modern populations.[32,37,38] The substantially increased rate of occurrence among certain specific, in-bred, populations (more than 8% prevalence among a prehistoric small island community) suggests a genetic etiology.[37] A recent, comprehensive, multicenter study found that Mx.C.P1 transposition was associated with increased frequency of other dental anomalies, such as tooth agenesis and peg-shaped lateral incisors, with a female predilection, a significant Caucasian ethnic preference, and a high bilateral occurrence. This further supports a genetic etiology.[38]

Our results show a remarkably high prevalence (15%) of Mx.C.P1 transpositions among patients with Down syndrome. In 4 out of the 5 transposition cases, this dental anomaly was present in dentitions that also exhibited either congenitally missing or peg-shaped maxillary laterals. These findings support the existence of a hereditary primary displacement of the tooth germ[26] in the Down syndrome population in addition to the other well-documented dental anomalies. Left-side preference for transposition is a finding supported elsewhere in the literature,[39] and is reminiscent of other oral anomalies such as cleft lip and palate. It is pertinent to note that the prevalence of cleft lip and palate has been shown to be 3- to 5-times higher in Down syndrome than in the general population.[40]

Others have speculated that blood circulation is impaired

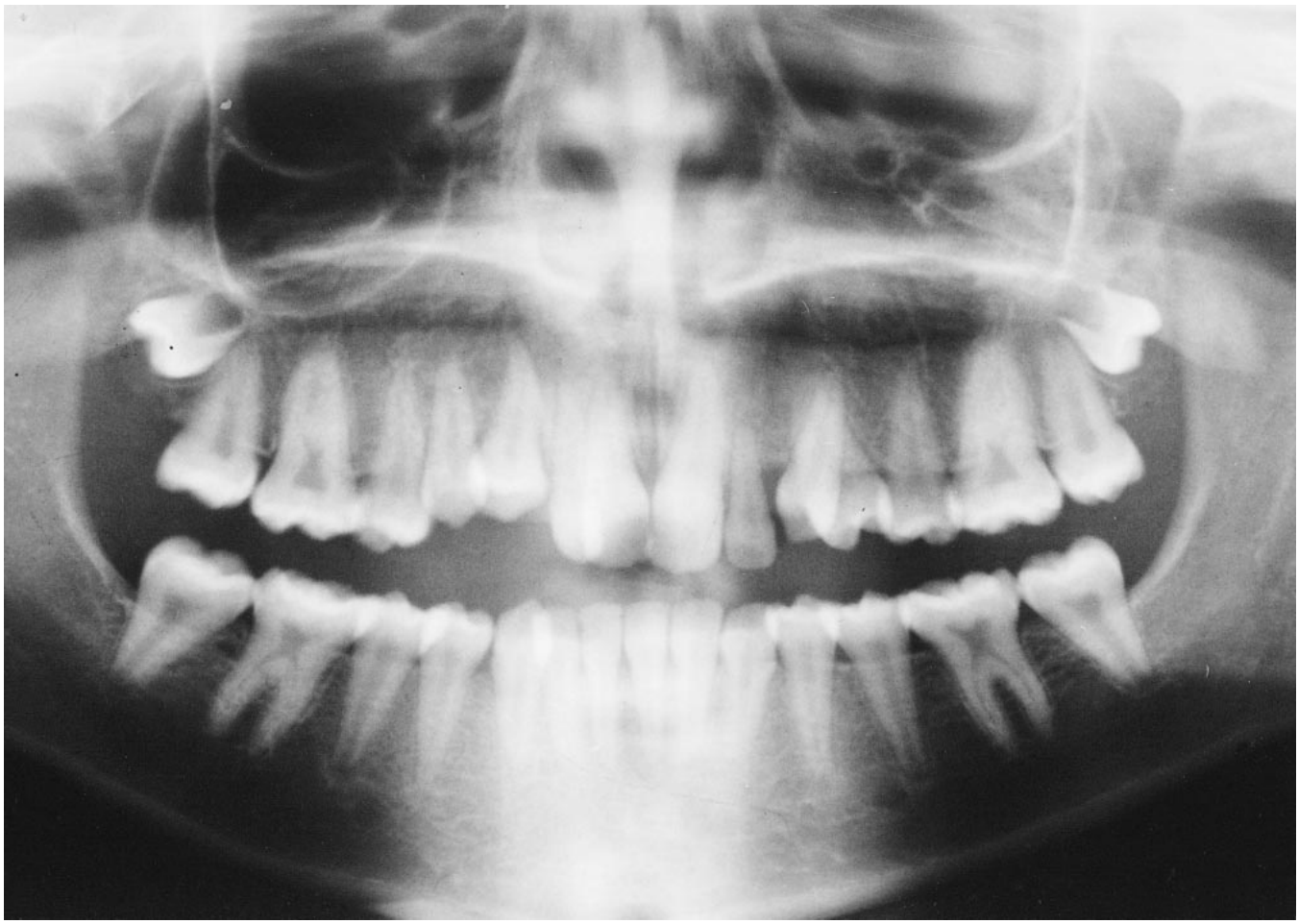

FIGURE 2. The panoramic view of a 13-year-old patient showing absence of maxillary left lateral incisor and bilateral Mx.C.P1 transposition.

in Down syndrome[41] and that an inadequate blood supply to the upper jaw could hamper its growth and cause degeneration of the odontoblasts, leading to small or missing teeth.[19] Perhaps it is no coincidence that there are several phenomena that occur frequently together and appear to be concentrated in the anterior maxilla, namely missing, small or peg-shaped lateral incisors, and canine impactions. However, this does not provide a plausible explanation for the elevated prevalence of transposition.

On the other hand, other investigators have made an association between missing teeth and prenatal peripheral nerve tissue development.[42] They have shown that missing teeth occur 20 times more frequently in Down syndrome, but that it pursues the same agenesis pattern of missing teeth as the general population, presumably indicating a more severe neurological disturbance in the Down patients.[43]

The results of the present study suggest a concentration or ''honing'' of a specific group of well-defined and linked dental anomalies in Down syndrome, in which their occurrence is many times that seen in control groups of normal individuals.

CONCLUSION

This study reveals a high prevalence of agenesis of third molars, impacted maxillary canines, and maxillary canine-premolar transposition in patients with Down syndrome. These anomalies should not be seen as separate, independent entities, but as intimately associated phenomena: the underdevelopment of the upper jaw, delayed dental development, reduction in teeth number and size, and the associated altered path of canine eruption. No explanation other than genetics is immediately available to explain why the maxillary canine/first premolar transposition should represent another phenotypic expression of this trisomy.

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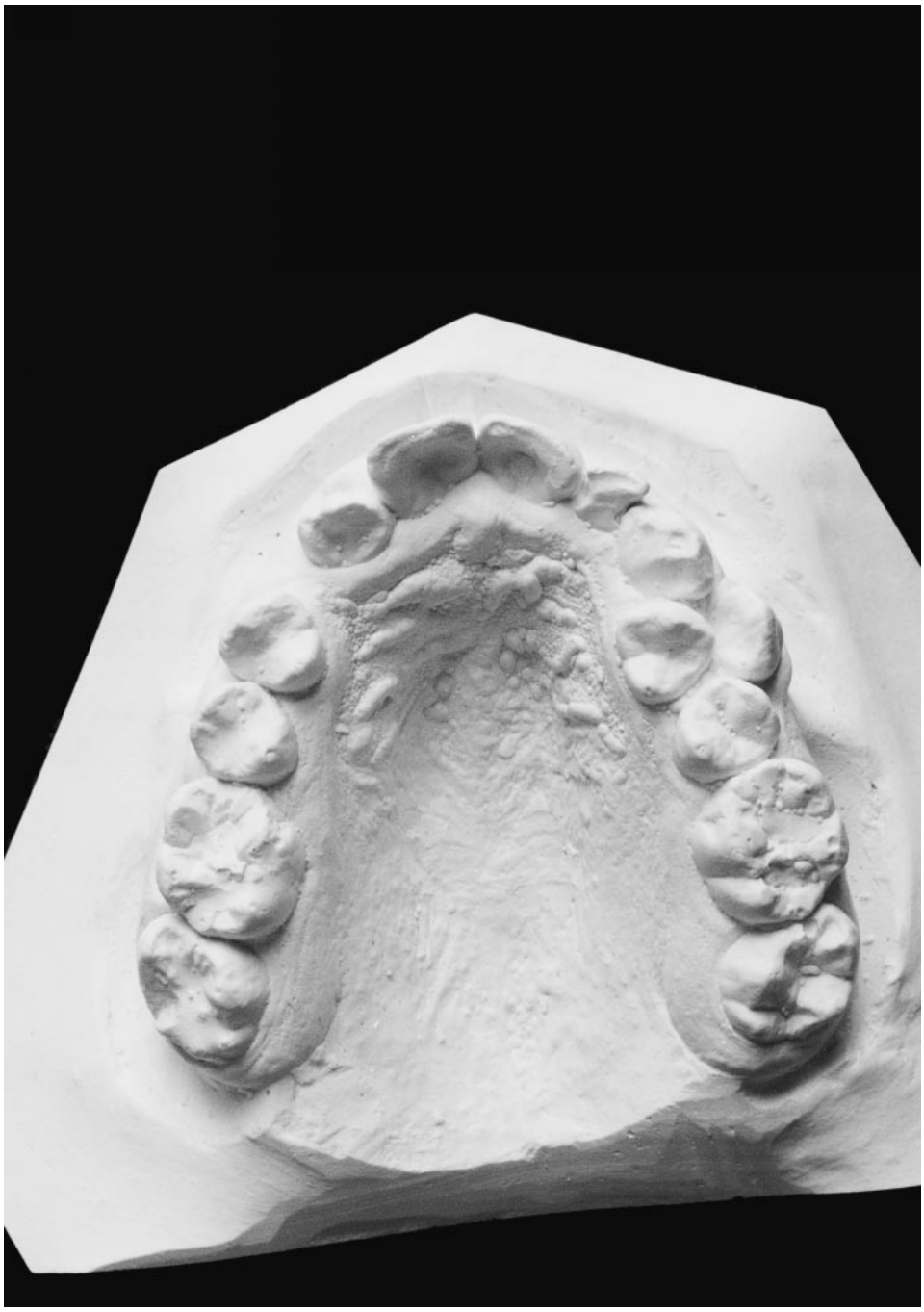

FIGURE 3. Occlusal view of maxillary dental cast of a 17-year-old patient showing left Mx.C.P1 transposition, missing lateral incisors, over-retained deciduous left canine and left lateral incisor. The right permanent canine has erupted into the place of the absent lateral incisor.

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