# Esthetic and Functional Rehabilitation for Oligodontia in the Mixed Dentition: Case Report

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## ABSTRACT

Tooth agenesis can be considered the most common anomaly of craniofacial development in humans, with an estimated prevalence reaching up to 20% worldwide. Treatments for tooth agenesis include orthodontic space closure, use of fixed or removable dentures, and placement of dental implants. Some of these options may not be applicable to children in mixed dentition because they can prevent the normal development of facial bones. A multidisciplinary, staged approach considering the number, size, and development of the remaining teeth in the long term would be the most suitable treatment. The purpose of this paper is to describe a 5-year clinical follow-up of a case of oligodontia in a young female patient in mixed dentition. (J Dent Child 2012;79(3):193-6)
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Tooth agenesis means lack of formation of certain teeth. It is classified, according to the number of missing teeth, as hypodontia (absence of fewer than 6 teeth), oligodontia (absence of 6 or more teeth), and anodontia (absence of all teeth).[1,2] Its etiology is related to different factors, including rupture of the tooth germ, hereditary factors, syndromes (particularly ectodermal dysplasia), viral diseases (especially rubella) and certain endocrinal disorders.[1,3,4] Although tooth agenesis is associated with at least 49 syndromes,[5] the prevalence of cases not associated with syndromes, systemic diseases, or trauma can reach 20%, making it the most common anomaly of craniofacial development in humans.[2,6] It may be linked to genetic factors, such as MSX1, PAX9, and AXIN2 mutations as well as modification of proteins and extracellular matrix molecules, producing changes in odontogenesis.[1,7]

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The permanent dentition is more often affected by agenesis than the primary dentition, with a prevalence ranging from approximately 1% to 10% in the general population, excluding the third molars; in the primary dentition, the prevalence ranges from approximately 0.5% to 1%.[8] Severe agenesis (absence of 4 or more teeth other than the third molars) has an estimated prevalence of 0.25%.[9-11] Agenesis of the permanent mandibular incisors is quite uncommon, with a reported prevalence of approximately 4% in a Finnish population.[12] Nevertheless, a high frequency of congenitally missing mandibular incisors (19%) has been described in individuals of Asian ethnicity.[13]

Females present a higher incidence of tooth agenesis than males (3:2).[14] Early diagnosis and effective clinical management of tooth agenesis are important, as this condition can lead to esthetic, psychological, and functional problems, such as malocclusion, periodontal damage, and inhibition of alveolar growth.[15] Its management typically involves a multidisciplinary approach, and the number, size, and development of the remaining teeth are significant factors in the long-term treatment plan.[16,17]

The purpose of this paper is to describe a 5-year clinical follow-up of a case of oligodontia in a young patient in mixed dentition and the proposed treatment plan for esthetic and functional rehabilitation in each developmental stage.

## CASE REPORT

An 8-year, 7-month-old Caucasian girl, accompanied by her parents, presented at the Children's Clinic of the Lutheran University of Brazil, Canoas, Brazil, in March of 2012. Her chief complaint was space between her permanent maxillary teeth and missing teeth. Similar cases were reported in previous generations of the family, which indicated hereditary causes. She had a concave facial profile, obtuse nasolabial angle, and marked labiomental fold (Figure 1A and B). Intraoral clinical examination revealed a large maxillary interincisal diastema and absence of mandibular incisors (Figure 1C and D). The patient was in the mixed dentition stage and had a good ratio of transverse arches, Class II Division 1 malocclusion, deep bite, and proclination of the permanent maxillary central incisors (Figure 2).Panoramic radiography indicated the absence of tooth germs of the permanent maxillary lateral incisors, permanent mandibular central and lateral incisors, and all the third molars. The remaining permanent teeth were developing normally (Figure 2D).

The following problems were identified: (1) large maxillary interincisal diastema caused by agenesis of the permanent maxillary lateral incisors, (2) proclination of the permanent maxillary central incisors, (3) mesioversion of the permanent maxillary left central incisor, (4) deep bite and (5) collapsed lower lip due to vertical bone loss caused by agenesis of the 4 permanent mandibular incisors.

The stages of treatment proposed for the patient were:

1. Insertion of a customized, functional, esthetic, fixed mandibular appliance, with acrylic teeth in the anterior region and bands in the primary second molars—at 8 years, 8 months of age (Figure 3A-C).
2. Bonding of brackets to the maxillary central incisors to close the diastema and direct the mesial eruption of the maxillary canines—from 9 years, 6 months to 10 years, 4 months (Figure 3D).
3. After diastema closure, insertion of a removable appliance with acrylic teeth for the maxillary lateral incisors and an anterior bite plane to improve the overbite—from 10 years, 5 months to 11 years, 8 months (Figure 3E).
4. Monitoring of the replacement of the primary teeth by their permanent successors—from 11 years, 8 months to 13 years, 6 months.
5. After the permanent canine eruption, replacement of the removable appliance by a fixed acrylic bite plane with bands in the permanent maxillary first molar region and brackets in the maxillary incisors to prevent the diastema from opening—from 11 years, 9 months to 13 years (Figure 4A-B).
6. Replacement of the lower lingual arch with a removable mandibular appliance with acrylic teeth in the anterior region before the primary mandibular second molars exfoliated—at 12 years, 2 months (Figure 4C).

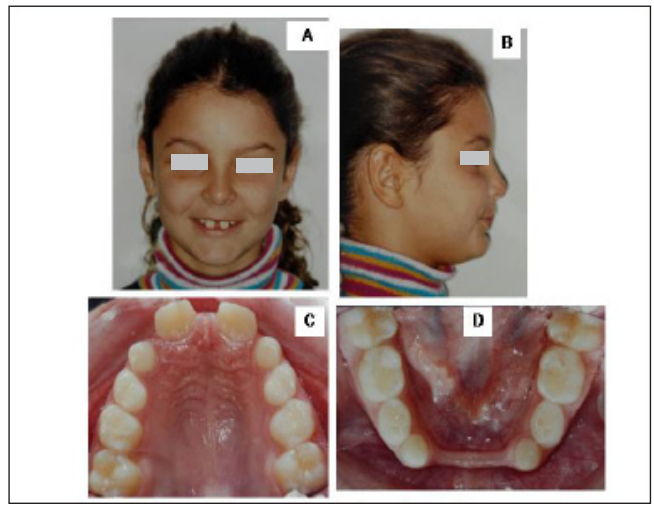


*Figure 1. (A) Extraoral photograph showing the patient smiling. (B) Extraoral photograph showing the concave facial profile. (C) Maxillary occlusal view showing agenesis of the permanent lateral incisors. (D) Mandibular occlusal view showing agenesis of the permanent central and lateral incisors.*

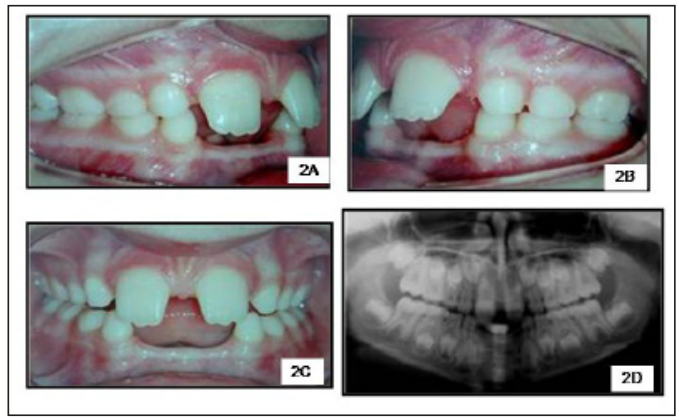


*Figure 2. (A) Right intraoral view. (B) Left intraoral view. (C) Intraoral frontal view. (D) Panoramic radiograph showing the missing teeth.*

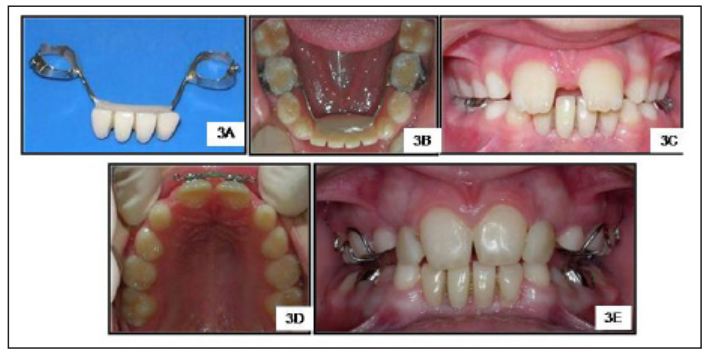


*Figure 3. (A–C) Modified mandibular lingual appliance for function and esthetics. (D) Bonded brackets for diastema closure. (E) Removable appliance with acrylic teeth for the permanent maxillary lateral incisors.*

The patient finished the first treatment phase at 13 years of age and is currently ready to start the second phase with a full fixed orthodontic appliance (Figure 4D). Panoramic radiographs taken at 11 and 13 years of age showed an unexpected mesioversion and impaction of the mandibular left canine (Figure 5). The patient will be referred to an oral maxillofacial surgeon for tooth exposure followed by orthodontic traction.

The second phase of treatment with a full fixed orthodontic appliance was delayed due to the patient's personal issues. In this forthcoming phase, maxillary spaces will be closed by mesial movement of the posterior teeth. After completion of the orthodontic treatment, the patient will be monitored with maxillary and mandibular removable retainers. Implants in the mandibular incisor area will be placed after alveolar growth has ceased.

## DISCUSSION

Early diagnosis of dental development abnormalities can significantly alter the treatment plan and prognosis of the affected teeth. Panoramic radiography in the mixed dentition is important to identify any abnormalities in tooth development, because the permanent dentition is affected by tooth agenesis in the primary dentition.[1,8,18] The dentist must be aware of a delay in tooth eruption, presence of erupted homologous teeth, presence of large anterosuperior diastema, and dentoalveolar ankylosis of the primary molars, all of which are indicative of future dental anomalies.[19,20]

Treatment options for tooth agenesis should be customized for each patient, considering age, esthetics, and the need for rehabilitation.[21,22] In the present case, the use of a fixed lingual arch-type appliance with acrylic teeth in place of the missing permanent teeth was considered suitable for maintaining the space required for future rehabilitation and for restoring the patient's smile. Given the patient's age, it was necessary to use a functional appliance that preserved the mandibular space until final prosthetic rehabilitation could be performed without interfering with the growth and development of the involved craniofacial structures.[22]

In the maxillary arch, considering the patient's age, the proposed treatment was central incisor alignment and diastema closure, which were the main esthetic complaints. Simultaneously, the vertical dimension could be improved. The next stage will consist of completion of the treatment with fixed appliances to finalize the space closure and the subsequent recontouring of the permanent maxillary canines as lateral incisors, and maxillary first premolars as canines.[23]

In conclusion, dentists should evaluate each case of tooth agenesis individually and over the long-term to prioritize the concerns of the patients and their parents while striving to achieve an esthetically and functionally favorable outcometaking into consideration the patient's growth and development.

## REFERENCES


1. Kolenc-Fusé FJ. Tooth agenesis: In search of mutations behind failed dental development. Med Oral Patol Oral Cir Bucal 2004;9:385-95.
2. Stockton DW, Das P, Goldenberg M, D'Souza RN, Patel PI. Mutation of PAX9 is associated with oligodontia. Nat Genet 2000;24:18-9.
3. Dermaut LR, Goeffers KR, De Smit AA. Prevalence of tooth agenesis correlated with jaw relationship and dental crowding. Am J Orthod Dentofacial Orthop 1986;90:204-10.
4. Chai WL, Ngeow WC. Familial cases of missing mandibular incisor: Three case presentations. Dent Update 1999;26:298-302.
5. Schalk-van der Weide Y, Beemer FA, Faber JA, Bosman F. Symptomatology of patients with oligodontia. J Oral Rehabil 1994;21:247-61.


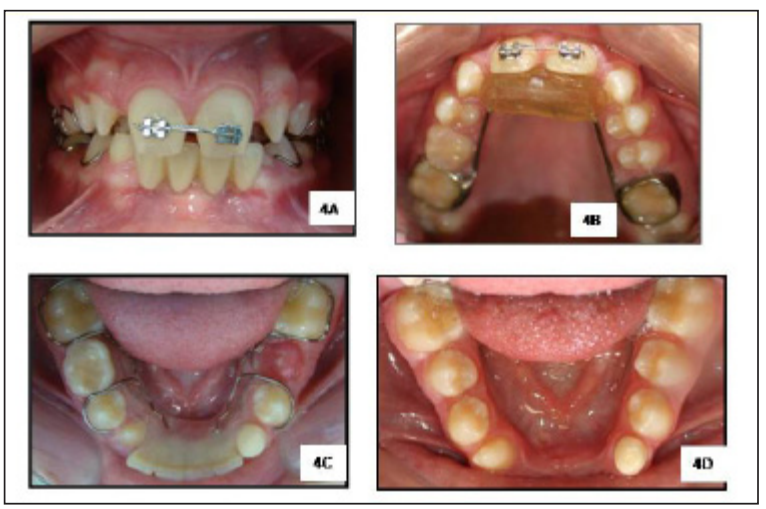


*Figure 4. (A) Removable maxillary bite plane. (B) Fixed maxillary bite plane. (C) Removable mandibular appliance for tooth repositioning. (D) Mandibular occlusal view.*

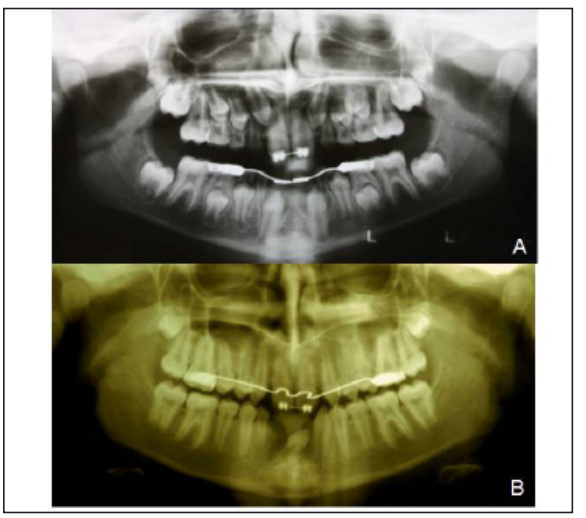


*Figure 5. Panoramic radiograph control showing mesioversion of the mandibular right canine at: (A) 11 years, 2 months old; and (B) 13 years, 6 months old.*

6. Frazier-Bowers SA, Guo DC, Cavender A, et al. A novel mutation in human PAX9 causes molar oligodontia. J Dent Res 2002;81:129-33.
7. Lammi L, Arte S, Somer M, et al. Mutations in AXIN2 cause familial tooth agenesis and predispose to colorectal cancer. Am J Hum Genet 2004;74:1043-50.
8. Vastardis H. The genetics of human tooth agenesis: New discoveries for understanding dental anomalies. Am J Orthod Dentofacial Orthop 2000;117:650-6.
9. Sarnäs K-V, Rune B. The facial profile in advanced hypodontia: A mixed longitudinal study of 141 children. Eur J Orthod 1983;5:133-43.
10. Nieminen P, Arte S, Pirinen S, Peltonen L, Thesleff I. Gene defect in hypodontia: Exclusion of MSX1 and MSX2 as candidate genes. Hum Genet 1995;96: 305-8.
11. Pirinen S, Kentala A, Nieminen P, Varilo T, Thesleff I, Art S. Recessively inherited lower incisor hypodontia. J Med Genet 2001;38:551-6.
12. Arte S, Nieminen P, Apajalahti S, Haavikko K, Thesleff I, Pirinen S. Characteristics of incisor-premolar hypodontia in families. J Dent Res 2001;80: 1445-50.
13. Goya HA, Tanaka S, Maeda T, Akimoto Y. An orthopantomographic study of hypodontia in permanent teeth of Japanese pediatric patients. J Oral Sci 2008;50:143-50.
14. Symons AL, Strizel F, Stamation J. Anomalies associated with hypodontia of the permanent lateral incisor and second premolar. J Clin Pediatr Dent 1993;17:109-11.
15. Kokich VG, Kokich VO. Congenitally missing mandibular second premolars: Clinical options. Am J Orthod Dentofacial Orthop 2006;130:437-44.
16. Nunn JH, Carter NE, Gillgrass TJ, et al. The interdisciplinary management of hypodontia: Background and role of pediatric dentistry. Br Dent J 2003;194:245-51.
17. Rune B, Sarnäs K-V. Tooth size and tooth formation in children with advanced hypodontia. Angle Orthod 1974;44:316-21.
18. Tunç ES, Bayrak S, Koyutürk AE. Dental development in children with mild-to-moderate hypodontia. Am J Orthod Dentofacial Orthop 2011;139: 334-8.
19. Baccetti T. A controlled study of associated dental anomalies. Angle Orthod 1998;68:267-74.
20. Garib DG, Peck S, Gomes SC. Increased occurrence of dental anomalies associated with second-premolar agenesis. Angle Orthod 2009;79:436-41.
21. Fiorentino G, Vecchione P. Multiple congenitally missing teeth: Treatment outcome with autologous transplantation and orthodontic space closure. Am J Orthod Dentofacial Orthop 2007;132:693-703.
22. Tulunoğlu O, Cinar C, Bal C, Bal BT. Two-year study of alternative conservative treatment modalities for early anterior permanent tooth loss. NY State Dent J 2010;76:27-30.
23. Kokich VO, Kinzer GA. Managing congenitally missing lateral incisors. Part I: Canine substitution. J Esthet Restor Dent 2005;17:5-10.