Case Report

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Bimaxillary fixed implant-supported zirconium oxide prosthesis therapy of an adolescent patient with non-syndromic oligodontia and two WNT10 variants: a case report

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Introduction and importance: Oligodontia is a rare genetic condition characterized by more than six congenitally missing teeth, either as an isolated non-syndromic condition or in association with other genetic syndromes. The impact of *WNT10A* variants on dental development increases with the presence of the c.321C > A variant and the number of missing teeth.

Case presentation: A 21-year-old man with non-syndromic oligodontia was diagnosed at 15 years of age with misaligned teeth, speech problems, and the absence of 24 permanent teeth. Interdisciplinary collaboration between specialists was initiated to enable comprehensive treatment. DNA analysis confirmed that the patient was a carrier of the known pathogenic *WNT10A* variant c321C > A and *WNT10A* variant c.113G > T of unknown clinical significance.

Clinical discussion: Dental implants are a common treatment; however, bone development challenges in adolescent patients with non-syndromic oligodontia necessitate careful planning to ensure implant success. Many WNT variants play crucial roles in tooth development and are directly involved in non-syndromic oligodontia, especially the *WNT10* variant c.321C > A.

Conclusion: A full-arch implant-supported monolithic zirconia screw-retained fixed prosthesis is a viable treatment option for young adults with non-syndromic oligodontia. Further studies are needed to clarify the possible amplifying effect of the *WNT10A* variants c321C > A and c.113G > T on the pathogenic phenotype of non-syndromic oligodontia.

Keywords: case report, implant-supported, monolithic zirconium-fixed prosthesis, non-syndromic oligodontia, screw-retained, WNT10

Introduction and importance

Oligodontia is a very rare dental agenesis that affects the permanent dentition more often than the primary dentition and is characterized by the absence of six or more teeth (excluding the third molars), affecting only 3.14% of the population[1]. Oligodontia may occur in isolated forms or non-syndromic and as part of syndromes[2], such as Witkop syndrome[3], Down syndrome[4], Axenfeld–Rieger syndrome[5], ectodermal dysplasia[6], and lip–jaw-joint cleft[7,8].

The clinical consequences of missing teeth in patients with non-syndromic oligodontia are prolonged retention of primary teeth, infraocclusion, ankylosis, unstable occlusion, microdontia, or conical teeth[9], which affect the patient's chewing function, speech, psychological, and social well-being. The absence of teeth also has a negative impact on skeletal growth and alveolar bone volume[10].

The etiology of non-syndromic oligodontia is multifactorial and includes genetic factors. There are many well-known

HIGHLIGHTS

- A 21-year-old man with non-syndromic oligodontia was diagnosed with misaligned teeth, speech problems, and the absence of 24 permanent teeth.
- DNA analysis confirmed the known pathogenic *WNT10A* variant c321C > A and *WNT10A* variant c.113G > T of unknown clinical significance.
- Full-arch implant-supported mandibular and maxillary monolithic zirconia screw-retained fixed prosthesis is a viable treatment option for adolescents and young adults with non-syndromic oligodontia.

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Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

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mutations of causative genes for non-syndromic oligodontia involving craniofacial bone and tooth development, including wingless-type MMTV integration site family, member 10A (*WNT10A*)[11], wingless-type MMTV integration site family, member 10B (*WNT10B*)[12], low-density lipoprotein receptor-related protein 6 (*LRP6*)[13], ectodysplasin A (*EDA*)[14], ectodysplasin 1 (*ED1*)[15], muscle segment homeo-box 1 (*MSX1*)[16], paired box gene 9 (*PAX9*)[17], *AXIN2*[18], and *LTBP3*[19,20].

Dental treatment of non-syndromic oligodontia, which is often associated with other changes in the orofacial complex, for example, tooth morphology and size, malocclusion, growth disorders of the maxillofacial skeleton, and facial appearance, is complex[21]. Early intervention in these patients can significantly improve oral function. It requires wide interdisciplinary cooperation among many specialists and clinicians[22,23]. According to the World Health Organization, the decision-making process for surgical intervention with the insertion of dental implants in adolescents aged 10–19 years is a major challenge because skeletal growth must be carefully considered[24].

We describe a unique case of non-syndromic oligodontia missing 24 permanent teeth documented over 7 years, with complex oral rehabilitation in close interdisciplinary collaboration with a full-arch implant-supported mandibular and maxillary monolithic zirconia screw-retained fixed prosthesis. We present this case report in accordance with SCARE 2023 criteria[25].

Case presentation

A 21-year-old Caucasian patient presented at an orthodontic practice complaining of poor dental aesthetics with diastema and abnormalities in the shape, size, and orientation of the anterior teeth, speech problems, and 24 permanent missing teeth (Fig. 1A). Physical examination revealed maxillary and mandibular hypoplasia, a concave facial profile, skeletal class III malocclusion, a circular crossbite, and oligodontia (Fig. 2). A panoramic radiograph confirmed the absence of teeth 17,15, 14, 13, 12, 11, 21, 22, 23, 24, 25, 27, 37, 35, 34, 33, 32, 31, 41, 42, 43, 44, 45, and 47. This indicated that only the remaining deciduous teeth and first permanent molars were present. Lateral cephalometric radiography and tracing demonstrated an underlying skeletal class III nature of her malocclusion. As the examination revealed no abnormalities in other parts of the body, including the nails, skin, hair, salivary glands, or sweat glands, the patient was diagnosed with non-syndromic oligodontia, and her family history was unremarkable.

Owing to the complexity of the case, interdisciplinary collaboration between pediatricians, psychotherapists, speech therapists, physiotherapists, general dentists, dental hygienists, geneticists, orthodontists, oral surgeons, and dental technicians was maintained from the beginning. After multidisciplinary input, preoperative orthodontic treatment began with the aim of preservation of primary teeth for as long as possible and to create an optimized space occlusal height for dental implant restorations.

To treat the prognathic mandible at 15 years of age, a head and chin cap with pins was used to protrude the maxilla. An implant-based forced palatal suture appliance was used in the maxilla (Fig. 3A). After successful orthodontic protrusion of the maxilla, a multi-bracket appliance was used to align the alveolar ridge in preparation for subsequent implant placement (Fig. 3B) The goal of this treatment phase was to achieve protrusion in the maxilla and retrusion in the mandible and to ensure the preservation of the teeth through fixation. As expected, preoperative orthodontic decompensation treatment closed the diastema and resulted in a slight overjet with a consistent dental and facial midline. To achieve sufficient vertical occlusal dimensions, bite turbos were used in the patient's molar region (Fig. 4A). This procedure resulted in a vertical height gain that was sufficient for the patient to adapt to the subsequent bite elevation caused by a fixed prosthesis with a postoperative temporary denture. At 18 years of age, with the help of orthodontic preparation, the digital dental planning with digital impression, cone bean computed tomography, and Facehunter, the placement of dental implants at our university in collaboration with the dental laboratory was prepared.

Surgical treatment is carried out by a university professor and begins with the extraction of the primary tooth. Then, after administering local anesthesia with Ultracaine and forming a mucoperiosteal flap, the two temporary palatal expansion implants and the abutment in the maxilla were placed together with the surrounding bone using a trephine drill. Next, deciduous teeth 55, 54, 53, 52, 51, 61, 62, 63, 64, 65, 75, 74, 73, 72, 71, 81, 82, 83, 84, and 85 were surgically extracted (Fig. 5A, B). The positions of the 12 implants, six per jaw, were digitally planned and fixed using a drill guide. A minimum torque of > 30 Ncm was achieved and used according to the standard drilling protocol. Implants measuring 11.5×3.25 mm were placed in regions 11 and 21 and implants measuring 11.5×4.3 mm were placed in regions 13, 23, 15, and 25. In addition, 3.25×13 mm implants were placed in regions 31 and 41 and 4.3×11 mm implants were placed in regions 33, 43, 35, and 45 (Fig. 5C, D). To increase the stability of the buccal bone structure, a bone-graft substitute was placed and covered with a pericardial membrane. The flaps were closed using 6-0 Prolene monofilament interrupted sutures.

The patient was given postoperative oral care instructions, which included rinsing the oral cavity twice daily for 1 week with a 0.2% (volume) chlorhexidine gluconate mouth rinse solution and using an extra-fine toothbrush to clean the temporary restorations. He received an antibiotic, amoxicillin/clavulanic acid, twice daily for 5 days. He was also prescribed 100 mg of mefenamic acid for pain control. The patient was advised to minimize trauma to the surgical site, but no special diet was recommended. The sutures were removed 10 days postoperatively. No complications were noted on subsequent postoperative examinations (Fig. 5F). For immediate postoperative care, two conventional full dentures were fabricated to allow a closed healing period of 7 months. No abnormalities were found during the subsequent monthly examinations.

In January 2022, the implants were exposed (Fig. 5G, H), and with the help of a transmission system using cone beam computed tomography, digital impressions and facial scans were performed in conjunction with the S600 ARTI scanner in the modeling software stored. The Facehunter allowed photorealistic three-dimensional digitization of the patient's face, enabling physiognomic work, such as axial positioning of the facial scan data with the models in the virtual articulator. Facial images and situations were related to the model based on facial features. This allowed the production of a prototype in combination with the CAD/CAM Reality Mode software module (Fig. 6A, B). The prototype consisted of a screwed polymethylmethacrylate (PMMA) tray divided into quadrants (Fig. 6C, D). The prototype

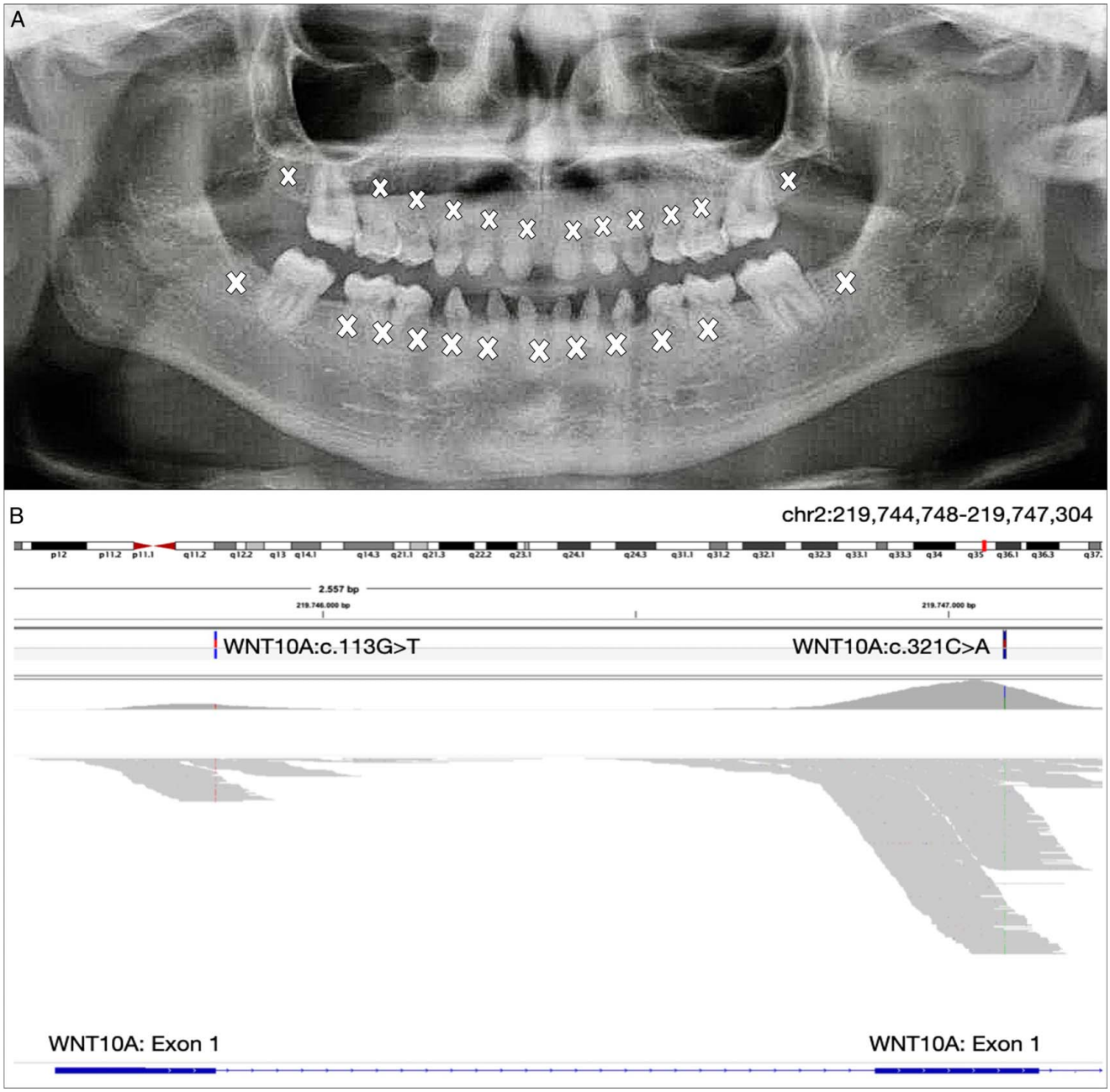
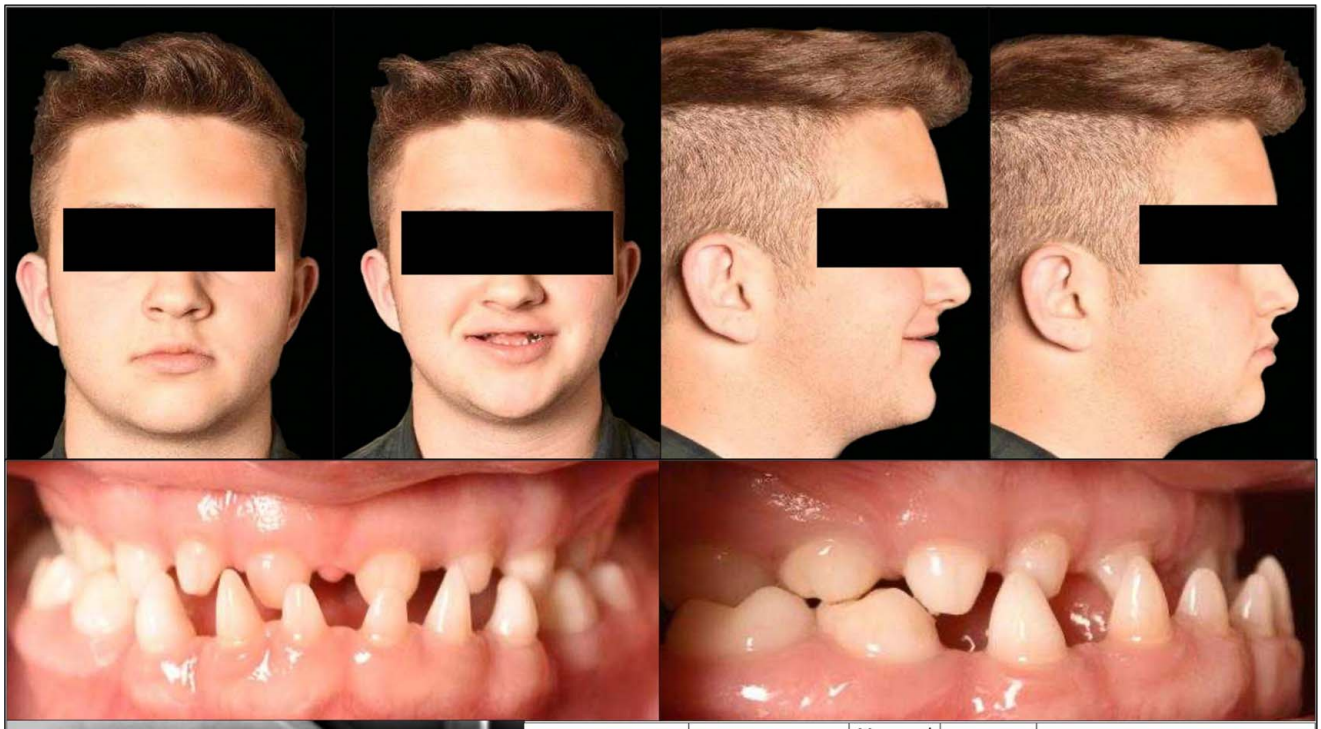


Figure 1. Radiographs and DNA analysis. (A) Panoramic radiographs with sites of oligodontia in the absence of the 24 permanent teeth are marked with stars. (B) DNA analysis reveals the pathogenic wingless-type MMTV integration site family, member 10A (*WNT10A*) variant c321C > A and an *WNT10A* variant c.113G > T of unknown clinical significance.

screw-retained PMMA restoration was worn by the patient for 5.5 months to assess their ability to maintain proper hygiene and bite elevation. The impressions in the PMMA restoration could then be used to adjust and control height and occlusion. As no difficulties were encountered, no changes were made to the design, and the prototype was converted into a complete, implant-supported, screw-retained, fixed prosthesis made of monolithic zirconium dioxide for the lower and upper jaws (April 2023) (Fig. 5I, J; Fig. 6E, F). To carefully monitor patient compliance, given that most bone remodeling occurs within 12–16 months after prosthetic rehabilitation, follow-up visits were initially scheduled four times in the first year (2023) and three times in the second year (2024). To date, good oral hygiene has been demonstrated, no abnormalities have been observed and the patient is satisfied with the result (Fig. 4B).

Owing to the 24 missing teeth, a human genetic analysis of the dental agenesis panel genes *WNT10A*, *WNT10B*, *LRP6*, *EDA*, *ED1*, *MSX1*, *PAX9*, gremlin 2 (*GREM2*), *LIBP3*, selective tooth agenesis 5 (*STHAG5*), and selective tooth agenesis 2 (*STHAG2*) was performed to identify the gene mutation responsible for this case of non-syndromic oligodontia. Whole-exome sequence and copy number analyses of the entire coding regions of genes from



Analysis	Standard/tolerance	Measured value	Deviation	Findings
Jaw relation				
SNA	81° +- 3.5°	75.83°	-5,17°	*Upper jaw retrognath
SNB	78° +- 3°	79.77°	+1.77°	Lower jaw orthognathic
SNPog	78.5 +- 3°	80.89°	+2.39°	Averagely pronounced chin
Skeletal class				
ANB	2° +- 2°	-3.93°	-5.93°	*Skeletal class III
iANB/ANB	0.15° +- 1°	-3.93°	-4.08°	*Skeletal class III
Growth pattern				
NSAr	123.5° +- 5°	118.98°	-4.52°	Average position of the temporomadibular joint fossa
SArGo	142° +- 6°	142.07°	+0.07°	Lower jaw orthognathic
ArGoMe	128.5° +- 6°	123.86°	-4.64°	Balanced growth pattern
Go1	55° +- 4°	57.13°	+2.13°	Normal
Go2	72.5° +- 4.5°	66.74°	-5.76°	*Reduced
Summen_W	394° +- 5°	384.91°	-9.09°	*Horizontal growth pattern
SN_SpP	7° +- 3°	4.85°	-2.15°	Normoinclination of the upper jaw
SN_MeGo	33.5° +- 5°	24.9°	-8.6°	*Horizontal growth pattern
SpP_MeGo	26.5° +- 5°	20.05°	-6.45°	*Skeletal deep bite
GHV	65% +- 4%	71.48%	+6.48%	*Horizontal growth pattern
Dental Analysis				
1_SN	103° +- 6.5°	109.18°	+6.18°	Maxillary front axis-aligned
1-SpP	70° +- 6°	65.97°	-4.03°	Maxillary front axis-aligned
1_MeGo	93° +- 6°	95.44°	+2.44°	Mandibular front axis aligned
1OK_1UK	130° +- 9°	130.47°	+0.47°	Stable
Soft tissue analysis				
NLW	102° +- 7°	96.89°	-5.11°	Average

Figure 2. Extraoral photographs, oral photographs, and radiographs. Oral photographs of skeletal class III malocclusion and lateral cephalometric radiograph and tracing:

the ectodermal dysplasia spectrum were chosen as the testing techniques. DNA analysis confirmed that the patient carried the pathogenic *WNT10A* variant c321C > A (ACMG class 5) and *WNT10A* variant c.113G > T of unknown clinical significance (ACMG class 3) (Fig. 1B). Additional DNA analyses revealed no variants in *WNT10B*, *LRP6*, *EDA*, *ED1*, *MSX1*, *PAX9*, *GREM2*, *LIBP3*, *STHAG5*, or *STHAG2*.

Clinical discussion

The absence of multiple teeth can have a serious impact on oral health-related quality of life, affecting the patient's chewing function, speech, and psychological and social well-being. Moreover, the absence of teeth has a negative impact on skeletal growth and alveolar bone volume[10].

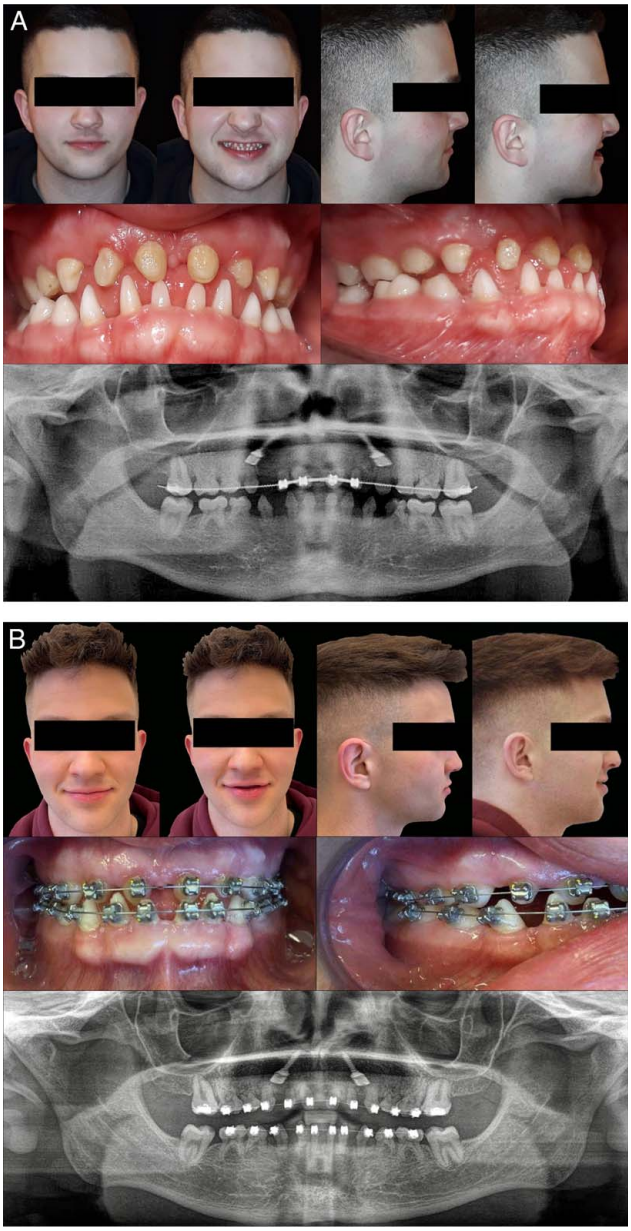


Figure 3. Extraoral photographs, oral photographs, and radiographs. (A) Implant-based forced palatal suture appliance in the maxilla and the start of orthodontic treatment. (B) A multi-bracket appliance is used to align the alveolar ridge for subsequent implant placement.

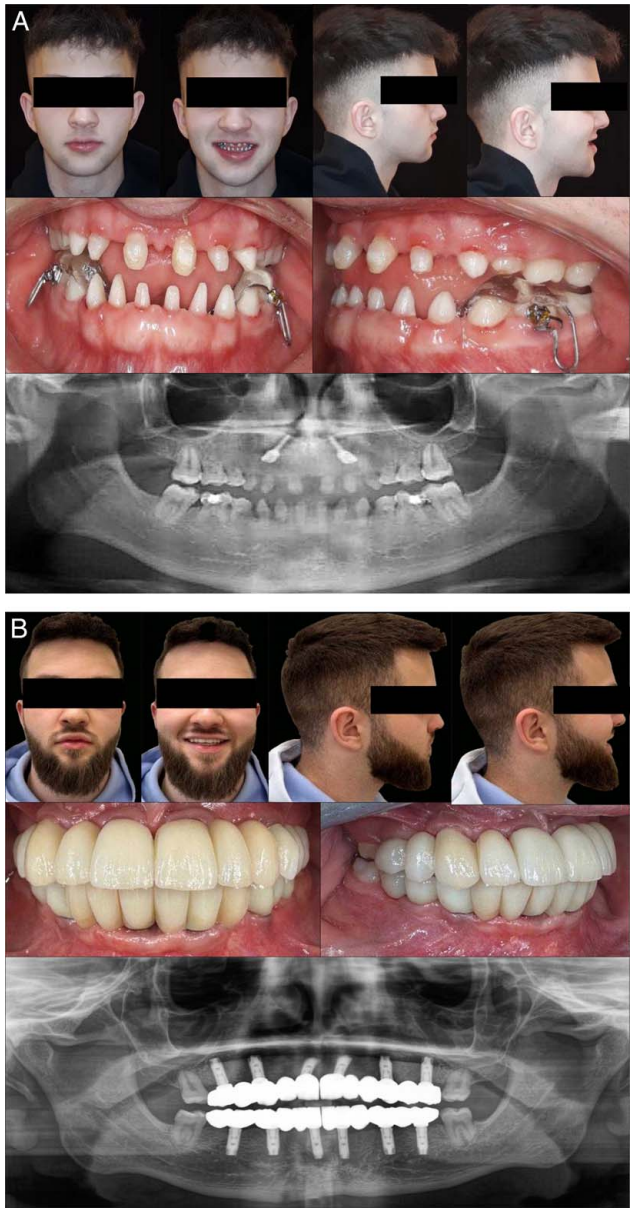


Figure 4. Extraoral photographs, oral photographs, and radiographs. (A) Bite turbos are placed in the molar region to achieve sufficient vertical occlusal dimensions. (B) Implant-supported screw-retained fixed prosthesis made of monolithic zirconium dioxide.

The consequences of missing teeth include prolonged retention of primary teeth, infraocclusion, ankylosis, unstable occlusion, microdontia, or conical teeth[23,26]. According to the International Statistical Classification of Diseases for Epidemiological Purposes, dental agenesis is recognized as the failure of the tooth germ to develop and, in addition to oligodontia, is classified as hypodontia, partial anodontia, or complete anodontia[27]. The cause of oligodontia is thought to be genetic, hereditary, or due to trauma, radiation overdose, glandular dysfunction, drug use, infection, systemic disease, or syndromes[28].

MSX1 plays a critical role in mediating epithelial–mesenchymal interactions during craniofacial bone and tooth development. In humans, genetic linkage analysis of a family with autosomal dominant selective hypodontia revealed a mutation in *MSX1*[29]. Such mutations predominantly affect the second and third molars and sometimes in combination with other tooth types, including the first molar. However, in more common cases of incisor–premolar-type hypodontia, the *MSX1* gene has been found. *PAX9* promoter polymorphisms are associated with hypodontia in humans[30]. *PAX9* is essential for the development of a number of organs and skeletal elements and is required for the condensation of tooth mesenchyme around the tooth bud epithelium[31]. PAX9-deficient teeth are arrested at the bud stage,

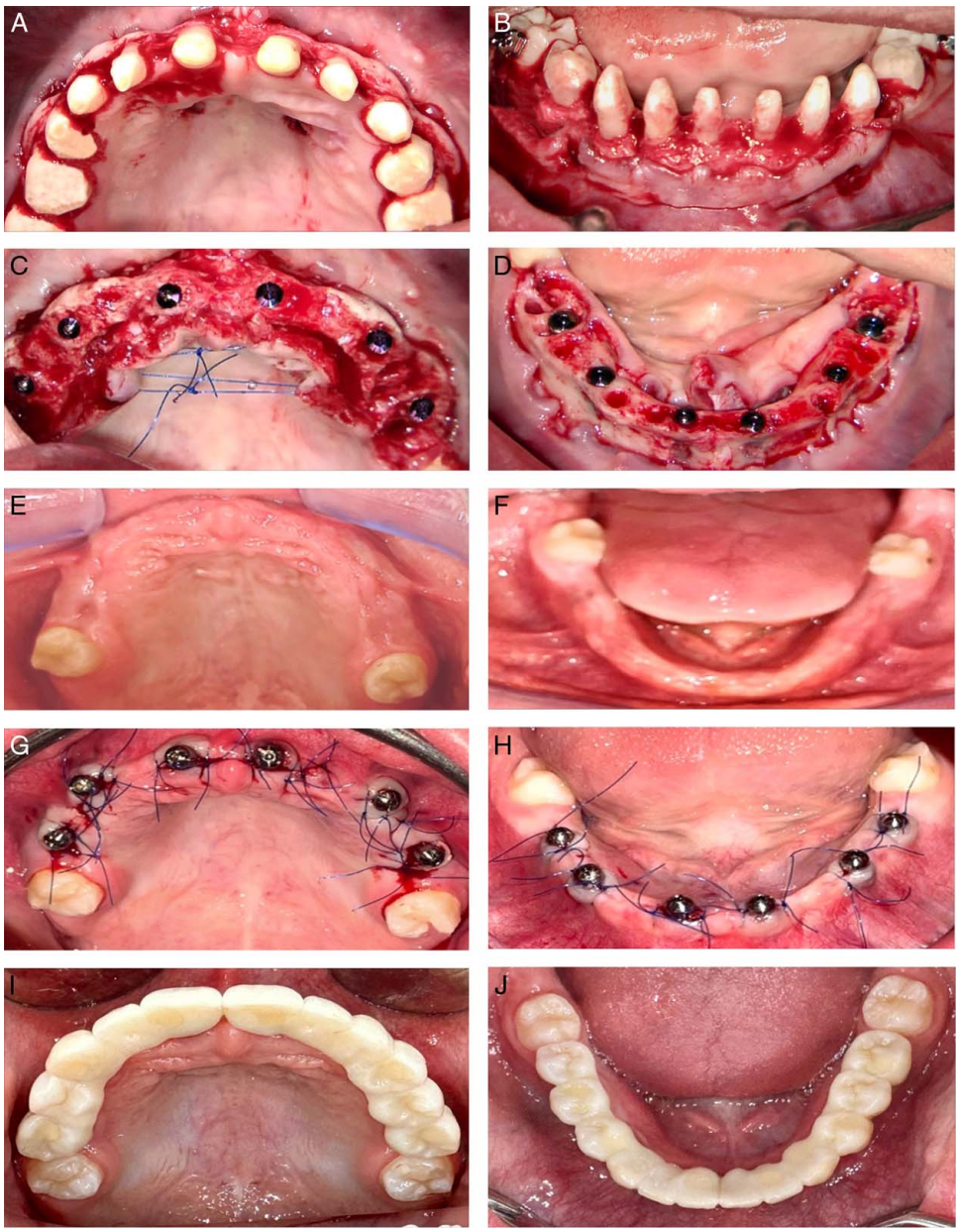


Figure 5. Oral photographs. (A, B) Surgical extraction of deciduous teeth 55, 54, 53, 52, 51, 61, 62, 63, 64, 65, 75, 74, 73, 72, 71, 81, 82, 83, 84, and 85. (C, D) Insertion of 12 dental implants. (E, F) No complications are observed on subsequent postoperative examinations. (G, H) Implant exposure in the maxilla and mandible. (I, J): Implant-supported screw-retained fixed prosthesis of monolithic zirconium dioxide in the lower and upper jaws.

and *PAX9* is required for the mesenchymal expression of MSX1, bone morphogenetic protein 4, and lymphoid enhancer binding factor 1[32]. Thus, *PAX9* plays a role in establishing the induction capacity of the dental mesenchyme. In humans, *PAX9* mutations are associated with unique phenotypes of familial dental agenesis, mainly affecting the posterior teeth.

In recent years, research on the etiology of tooth agenesis has focused on genetic background. Several genes and genetic pathways involved in the etiology of isolated hypodontia have been identified: *WNT10A*, *WNT10B*, *LRP6*, *EDA*, *MSX1*, *PAX9*, *AXIN2*, and *GREM2*[33,34]. Mutations in *WNT10A* were present in more than half of patients with isolated oligodontia, indicating that the Wnt pathway is particularly important for tooth formation[35]. Among the members of the WNT family, *WNT10A* is involved in the development of ectodermal derivatives, including teeth[36]. It has previously been associated with dental syndromic and non-syndromic diseases[37], and some single nucleotide polymorphisms have been linked to changes in the dentin matrix and dental abnormalities[38–40]. In the present study, *WNT10A* variants were found in patients with delayed tooth development and more missing teeth. Another recent finding suggested that the association with *WNT10A* was stronger with an increasing number of missing teeth and the presence of the nonsense

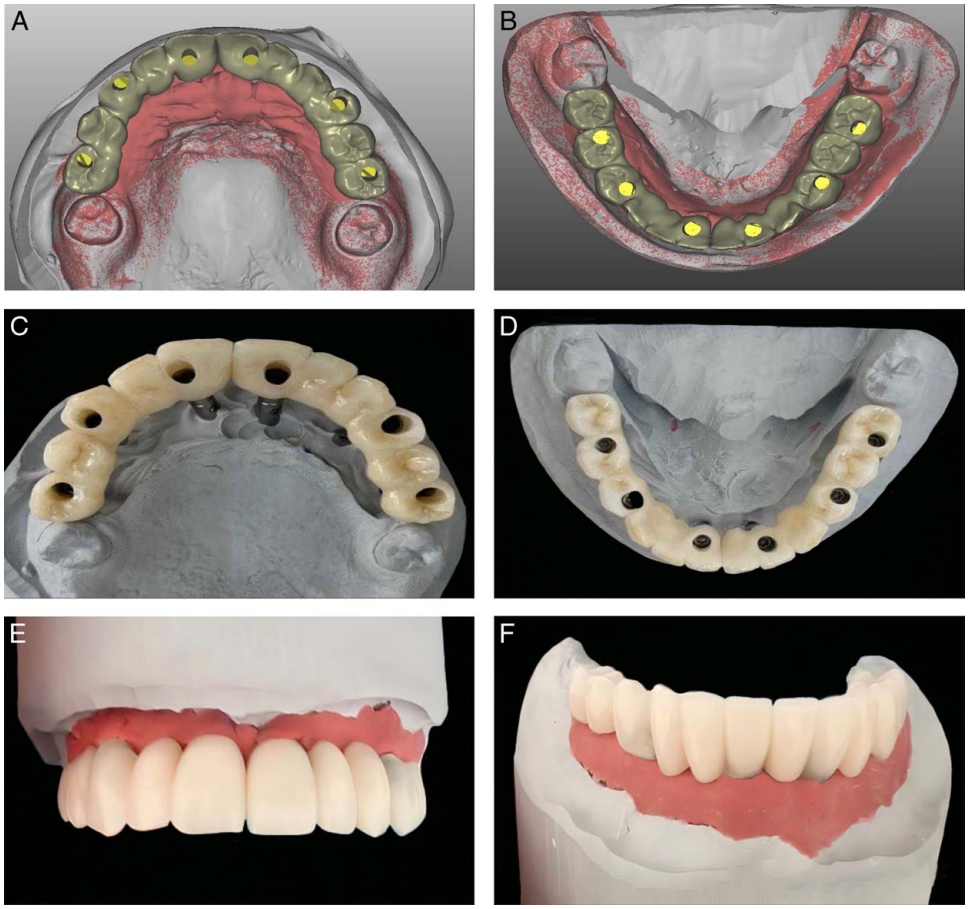


Figure 6. (A, B) Prototype combined with the CAD/CAM Reality Mode software module. (C, D) The screwed polymethylmethacrylate (PMMA) prototype. (E, F) Implant-supported screw-retained fixed prosthesis of monolithic zirconium dioxide.

variant c.(321C4A p.(C107*))[41,42]. *WNT6* is another important gene involved in tooth development and is associated with the migration and differentiation of dental papilla cells and the formation of dentin bridges.

In our patient, DNA analysis of panel genes *WNT10A*, *WNT10B*, *LRP6*, *EDA*, *ED1*, *MSX1*, *PAX9*, *GREM2*, *LIBP3*, *STHAG5*, and *STHAG2* was performed to screen for sequence variants and copy number alterations. DNA analysis confirmed that the patient carried the pathogenic *WNT10A* variant c321C > A (ACMG class 5) and *WNT10A* variant c.113G > T of unknown clinical significance (ACMG class 3). This indicates that the variant NM_025216.3:c.321G > A, which leads to the premature termination of protein synthesis due to a premature stop codon (NP_079492.2:p. Cys107Ter), was found to be heterozygous in *WNT10A*. This variant is stored in dbSNP as rs121908119, ClinVar as ID 19500, and HGMD as CM094234, and it is thought to cause ectodermal dysplasia-type *STAHG4*, which follows both autosomal recessive and autosomal dominant inheritance patterns. Therefore, heterozygous carriers can also develop a phenotype. This indicates that the clinical suspicion of hereditary dental agenesis of the *STAHG4* type can be confirmed using molecular genetics. In the same gene, the variant NM_025216.3:c.113G > T was found in a heterozygous state, resulting in the amino acid exchange NP_079492.2:p.Arg38Met. This variant has not yet been found in databases and has not been described in the literature; therefore, it cannot be interpreted. On the basis of predictive algorithms, it was classified as a variant of unclear significance (ACMG class 3). The amplifying effect of this variant on the pathogenic phenotype could not be confirmed or ruled out. Thus, further studies are needed to clarify the possible enhancing effects of this variant on pathogenic phenotypes. The phases of these variants are also unknown. If they are trans, both recessive and dominant inheritance are possible, and every child of the patient with this variant will inherit one of both variants. If they are in cis, the inheritance is dominant; 50% of the children will inherit both variants, and 50% will not inherit them. Additional DNA analysis revealed no variants in *WNT10B*, *LRP6*, *EDA*, *ED1*, *MSX1*, *PAX9*, *LIBP3*, *GREM2*, *STHAG5*, or *STHAG2*.

Dental implants are widely accepted treatment options for patients with congenital tooth loss. However, our patient had oligodontia associated with other substantial oral maxillofacial diseases, including maxillary and molar hypoplasia, a concave facial profile, and skeletal class III malocclusion. Given the complexity of the patient's oral, jaw, and facial conditions, optimal aesthetic and functional rehabilitation could not be achieved with dental implant treatment alone. Therefore, a multidisciplinary, step-by-step approach was necessary.

Orthodontic treatment can improve the pre-implantation and prosthetic status of adolescent patients with non-syndromic oligodontia[43]. It aims to provide an optimized restorative space for dental implant placement.

The decision to extract all the primary teeth instead of exploring the possibility of retaining these teeth with sufficient root length, such as the second primary molars, to reduce the invasiveness of implant-prosthetic rehabilitation must be viewed critically in times of the trend toward minimally invasive prosthetic treatment in young adults. In the present case, the health insurance company approved only a single reimbursement for implant-prosthetic rehabilitation, but not for repeated minimally invasive prosthetic treatments. The decision to extract all primary teeth and perform implant-prosthetic rehabilitation was therefore made together with the patient and the parents, with the ulterior motive of having the costs covered by the health insurance company.

Considering the psychological burden on young patients with non-syndromic oligodontia, implant treatment may be considered during the growth phase if alternatives, such as Maryland bridges or orthodontic gap closure, are not possible[44].

Implantation in adolescent patients with non-syndromic oligodontia carries inherent risks[45]. When considering the timing of dental implant placement in adolescent patients, it is important to assess skeletal age and avoid crossing a suture line when using a fixed implant-supported prosthesis. It is also essential to ensure that patients and their guardians are fully informed of these considerations.

Owing to the lack of tooth eruption, the alveolar bone is typically underdeveloped in patients with oligodontia. An insufficient amount of bone poses enormous challenges and risks for implant therapy, such as insufficient initial stability and long-term peri-implant bone loss[46]. However, aligning the bone in its physiological position reduces the pressure on the implant, resulting in a higher success rate and a lower risk of incorrect loading of the temporomandibular joints. To minimize the risk of marginal bone loss, interim prostheses were used during the integration phase to stabilize the patient's new occlusal height and occlusion. Implant loading gradually increased, and screw-retained plastic interim prostheses were placed after a 5.5-month integration period to prevent overloading of the temporomandibular joints. Physiological implant loading increases peri-implant bone density, whereas infraocclusion and implant overloading lead to peri-implant bone loss[47].

Herein, we described the successful dental management of an oligodontia patient with complex dentofacial abnormalities. Collaborative interdisciplinary care by pediatricians, psychotherapists, speech therapists, physiotherapists, general dentists, dental hygienists, geneticists, orthodontists, oral surgeons, and dental technicians was required to successfully restore oral function, form, and comfort and achieve desirable outcomes. Given the long treatment duration, clinicians must avoid patient burnout by actively involving patients and parents in open discussions regarding treatment options and goals at different stages of treatment. For this patient, it took 7 years to complete the treatment. The patient and her parents were actively engaged in each phase of treatment, and the goals and results of each phase were clearly communicated and reviewed by the patient and her parents. The patient understood the predicted results of each step and appreciated the importance of each procedure. At the end of each treatment phase, the results and plan of the next phase were reviewed by the patient and her parents, so that they could appreciate the progress and understand what to expect next; thus, the patient was motivated to continue treatment. To optimize the prosthetic results, we used a diagnostic tooth setup, interim removable denture, and interim implant prostheses to streamline the treatment at different treatment phases. The continuous provision of patient-satisfactory temporary prostheses played a key role in the successful execution of this long-term treatment.

Conclusion

Dental implants are a viable treatment option for adolescents and young adults with non-syndromic oligodontia. Careful treatment planning and interdisciplinary collaboration enable the use of complex full-arch implant-supported mandibular and maxillary monolithic zirconia screw-retained fixed prostheses for oral rehabilitation. However, the genetic component should not be ignored, and further studies are needed to clarify the possible amplifying effects of the known *WNT10A* variant c321C > A and the unknown *WNT10A* variant c.113G > T on the pathogenic phenotype of non-syndromic oligodontia.

Ethical approval

This study was approved by the relevant ethics committee.

Consent

The patient received a thorough explanation of this report and gave oral and written informed consent to be included in this report as well as for publication of this case, anonymous data, and pictures. A copy of the written consent is available for review on request.

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This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author contribution

All authors contributed to the conception and design of the study; P.G., F.P.-M., N.L., C.M., M.G., and D.T.: contributed to the data collection, analysis, and discussion of the data; D. T.: contributed to the surgical treatment of the patient; D.T. and P.G.: wrote the manuscript; C.M.: made the orthodontic contribution to the manuscript and provided intensive orthodontic and dental care to the patient; M.G.: performed the genetic examination and made the genetic contribution to the manuscript; F.P.-M. and P.B. contributed to the coordination of the patient; and N.L. and D.B. were responsible for the long-term photographic documentation and design of the graphics. All authors have approved the final version of the manuscript submitted for publication.

Conflicts of interest disclosure

The authors declare that there are no conflicts of interest.

Research registration unique identifying number (UIN)

Not applicable.

Guarantor

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