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Prosthetic Rehabilitation of Familial Non-syndromic Oligodontia: A Case Report

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Abstract

Oligodontia, or congenital teeth agenesis, is an uncommon developmental defect characterised by the absence of more than six teeth, with the exception of the third molars. Oligodontia is frequently associated with specific congenital disorders. Oligodontia's specific cause is unknown, but it is thought to have hereditary roots. Patients who are missing teeth may experience issues with appearance, functionality, or mental health. In the current study, a 13-year-old patient with non-syndromic familial oligodontia and the lack of 16 teeth is described. The ultimate diagnosis of non-syndromic familial oligodontia was determined based on the study of the case where the patient reported a family history of oligodontia, and the lack of any clinical symptoms associated with any syndrome. The case was given the option of multidisciplinary dental therapy.

Keywords: Anodontia, Tooth agenesis, Hereditary

Introduction

Teeth are crucial in this, as they play a major part in one of humanity's most essential face expressions—smiling. Missing teeth are one of the most prevalent anomalies in the permanent dentition. There are many possible effects of this. A variety of terms have been used to describe the congenital absence of teeth in the permanent and primary dentition. The absence of one to six teeth is referred to as hypodontia, the loss of more than six teeth (apart from the third molars) as oligodontia, and the total absence of teeth (anodontia) as hypodontia. These diseases have a genetic basis.[1,2]

The percentage of hypodontia in the Indian populace has only been shown to be 4.19% in prior studies. Oligodontia only occurs in 0.36% of the population, illustrating how uncommon the condition of greater than six teeth agenesis is.[3]

Oligodontia can occur alone, seldom, or in combination with a number of genetic syndromes, including ectodermal dysplasia, Van Der Woude syndrome, Down syndrome, Reiger syndrome, and an unrelated nonsyndromic illness. Oligodontia is characterised by conical-shaped teeth, microdontia, delayed eruption of permanent teeth, enlarged freeway space, and retention of deciduous teeth. By conducting a thorough physical examination of the hair, nails, sweat glands, eyes, and screening for any congenital disorders, oligodontia can be divided into syndromic and non-syndromic variants.[4]

Case Report

A 13-year-old girl patient who had never had any dental work done before visited the dentist for the first time. The patient had a chief complaint of missing posterior teeth in the upper and lower, left and right back region of the jaw for 8 years. The patient's medical history was irrelevant. The patient was the eldest of two children of nonconsanguineous parents who gave birth to their children normally. Further research found that other family members have also experienced irreversible posterior teeth loss in the past. (Figure 1)

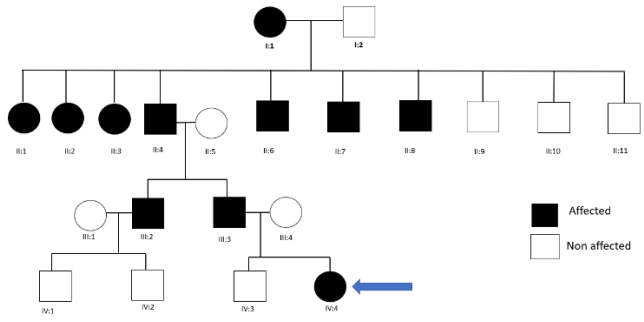

Figure 1: Pedigree chart of the patient

The patient had a straight physique and was about average height. A concave facial profile and normal skeletal dental base relations were visible on the face during extraoral examination. (Figure 2)

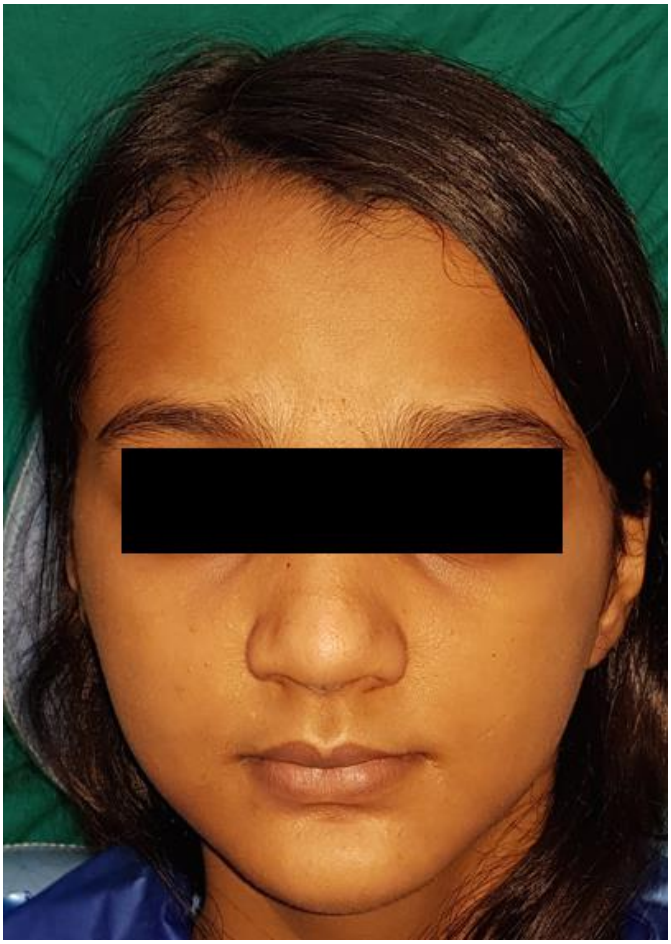

Figure 2: Extraoral frontal view of the patient

Prior to the thorough examination, the patient's parental permission and assent were obtained.

It was discovered during an intraoral clinical examination that the patient had bilateral permanent maxillary canines, bilateral permanent maxillary first premolars, and bilateral permanent maxillary central incisors. There were also permanent mandibular central incisors and bilateral permanent mandibular first premolars.

The primary mandibular right central incisor, primary mandibular canines, and primary maxillary lateral

incisors were also present in the dentition and were all retained.

The dentition's remaining teeth were congenitally absent. Grade II mobility was also present in the right central incisor of the primary mandible. Additionally, the patient exhibited class III frenum attachment. Additionally, the maxillary arch had a fibrous growth from ongoing trauma from the retained number 81.(Figure 3)

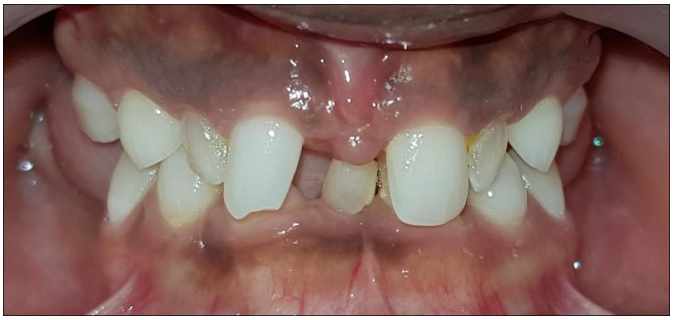

Figure 3: Intraoral frontal view of the patient in occlusion

An orthopantomogram (OPG) revealed the congenital deficiency of third molars in all four quadrants and radiographically confirmed the loss of all teeth. (Figure 4)

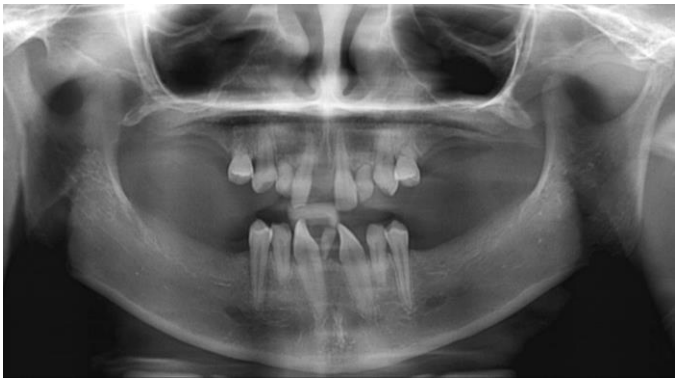

Figure 4: OPG of the patient

Other orthopantomogram findings were within normal ranges for the patient's age.

With Ectodermal Dysplasia, Rieger syndrome, and Van der Woude syndrome in the differential diagnosis, non-syndromic oligodontia was assigned a provisional diagnosis. Physical examination results excluded ectodermal dysplasia since the patient's hair was not thin and sparse, nails were not brittle, and there was no trouble sweating. Ocular examination results also excluded Rieger syndrome because there was no cleft palate or mucosal cysts in the lower lip. The radiographic assessment of the hands and wrists was clear. Finally, based on the facts above, the final diagnosis of familial non-syndromic oligodontia was justified.

The grade II mobile primary mandibular right central incisor will be extracted as part of the patient's treatment plan, which will also involve a class III maxillary frenectomy.

Frenectomy was performed before undergoing prosthetic rehabilitation and was done under local anaesthesia and was accomplished with the help of electrocauterization surgery.

Due to the patient's low socioeconomic situation, removable prosthodontic therapy was recommended after healing.

In order to enhance function and appearance, prosthetic therapy involved replacing the anterior teeth and other deficient teeth. Using a condensation-type polyvinyl siloxane silicone impression material, putty-wash technique was used to create impressions of the maxilla and mandible. A Type II dental stone was used to pour the impressions. On the primary cast, the tray's contour is marked. The cast is modified with a 2 mm thick wax spacer, and at minimum three tissue stops are made in non-critical regions to create room for the impression material. Acrylic resin autopolymerizes to create a bespoke tray.The greenstick compound is used for border moulding. A tray is filled with assembled impression material. Until the material settles, the tray is firmly held in the mouth.

The pick-up impression method described by Mclean is used. After beading and boxing, the master cast is poured. The master tray had wax rims made for it. The casts were put on an articulator after the jaw relation was measured and recorded. Artificial teeth were chosen and placed in the mouth in accordance with the size of the real teeth already there. Both an aesthetic try-in and a verification of the jaw relation records were completed. Heat cure acrylic resin was used to produce the denture bases. Denture base final polishing and finishing were completed. (Figure 5,6 and7)

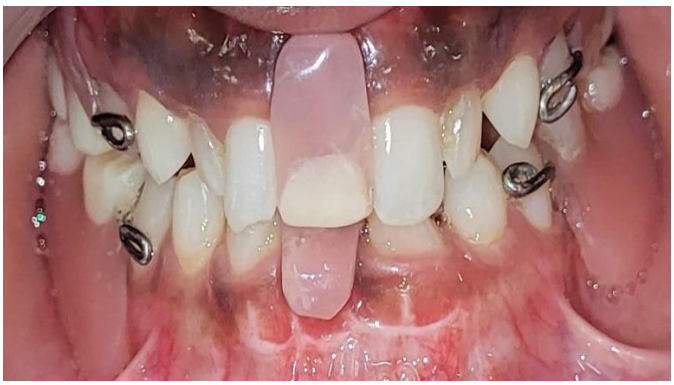

Figure 5: Post-treatment intraoral frontal view of the patient in occlusion

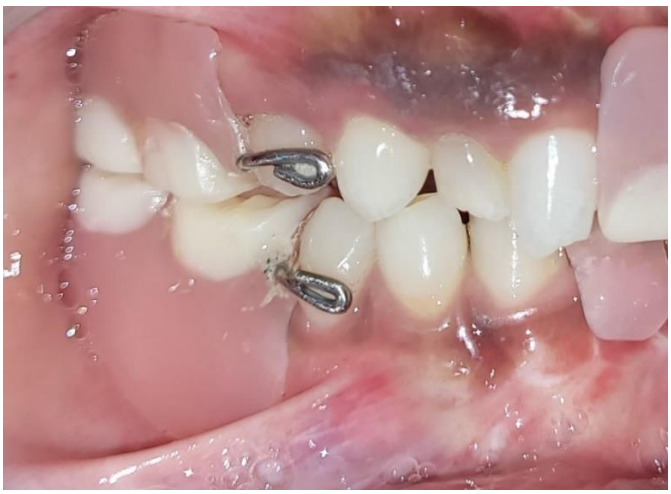

Figure 6: Post-treatment right lateral view of the patient in occlusion

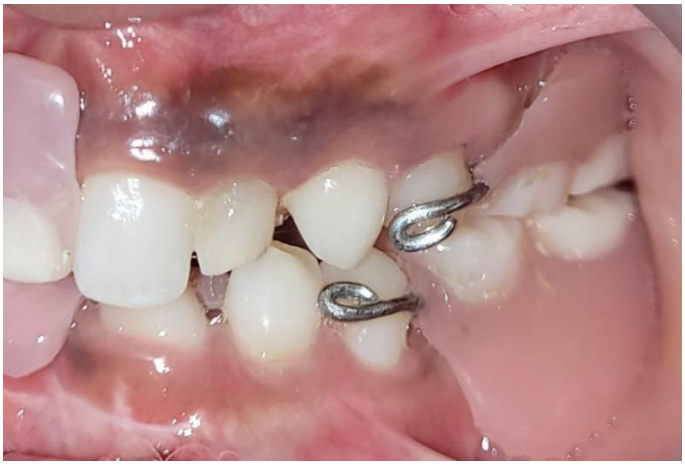

Figure 7: Post-treatment left lateral view of patient in occlusion

Discussion

A common developmental defect that affects 2.2-10% of the general population is tooth agenesis (excluding agenesis of third molars).[5]

Oligodontia, according to Schalk-van-der-Weide, is defined as the agenesis of six or fewer permanent teeth apart from third molars.[6]

There is a wide range of information on the prevalence of oligodontia, from 0.08–0.16% among Danish schoolchildren as reported by Rlling et al. to 1.1% across a Caucasian population as described by Gábris et al.[6]

In research done in Indore, India, the percentage of familial non-syndromic oligodontia was found to be 0.36%, which is slightly higher than the 0.08% to 0.16% global incidence of the condition. In a 3:2 ratio, girls are more likely than boys to experience it.[4,5]

Congenital toothlessness is thought to have genetic roots in its genesis. Oligodontia is controlled by genetics, but other factors, including X-ray therapy, specific drugs, infectious infections, trauma, endocrine and intrauterine abnormalities, cannot be ruled out. Familial tooth agenesis can be passed down as an autosomal dominant, recessive, or X-linked disorder and can appear as a single abnormality or as a component of a genetic syndrome. The MSX1 and PAX9 genes have been

linked to early tooth genesis, according to studies. According to Sun K. et al., MSX1 mutations have been linked to non-syndromic cleft lip with/without cleft palate and autosomal dominant hypodontia, while PAX9 has a role in some human populations' lack of wisdom teeth.Missing maxillary first premolars, second premolars, and mandibular premolars are common in oligodontia linked to MSX1. The lack of the maxillary first premolars is a defining characteristic of MSX1-associated oligodontia, whereas PAX9-associated oligodontia is linked to the absence of both the maxillary and mandibular second molars.[7]

The purpose of treatment is to enhance look, mastication, and speech since oligodontia patients' unsightly countenance, absent teeth, and overclosure can lead to depression and psychosocial issues, especially in children.

According to the severity of the disorder and the patient's perceived need for care, the therapy is necessary not only to enhance aesthetics but also to rehabilitate function and psychological elements. The therapy not only enhances the child's ability to speak and chew, but it also has psychological effects that may substantially aid in restoring the child's sense of self and social perception.

The choice and planning of the patient's treatment are significantly influenced by their age. A successful treatment outcome and the selection of an appropriate treatment strategy depend on the timeliness of the diagnosis.

Oligodontia has a variety of prosthetic treatment options, including overdentures, implants, cast partial dentures, detachable partial dentures, and fixed partial dentures. The state of the residual teeth and alveolar bone will determine the course of treatment.

Once the time of active growth is completed, dental implants can be placed at a later age. The patient's parents were informed of the protracted rehabilitation plan involving dental implants, but due to financial restrictions, the parents chose a less expensive kind of therapy. Implants might be viewed as a permanent treatment in the future.

A removable partial denture was given to the patient by Basoya S et al. in their case report as a prosthetic treatment option because the patient was more focused on aesthetics.[8]

When there was no evidence of the development of teeth 31, 32, 33, 41, 42, or 45 in a case report of non-syndromic oligodontia by Pannu P et al, the fixed prosthetic replacement of the congenital absence of mandibular teeth was carried out using acrylic teeth linked to a lingual arch.[9]

In our situation, the child was more concerned with appearance, so we started by giving her a detachable partial denture. As a result, a detachable prosthesis was chosen as the course of treatment in the current clinical situation.

The patient’s parents were informed that prosthesis may occasionally need to be replaced due to changes in growth, and that dental implants would be the preferable alternative for full mouth prosthetic rehabilitation as the patient’s growth is completed.

A patient who has missing teeth not only experiences masticatory and cosmetic issues, but also psychological stress because it can undermine a healthy person's self-esteem. Clinicians should carefully assess cases of oligodontia, especially paediatric dentists as an early diagnosis enables correct treatment planning and enhances the child's mental, oral and general health.

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