

Accidently Perceived Condylar Aplasia- A Rare Case Report

Dr.Ranjeet.Surwase¹, Dr.Abhay.Kulkarni², Dr.Pratik.Parkarwar³, Dr.Nahush Chaudhari⁴ and Dr.Ashutosh Tervankar⁵

¹Sr.Lecturer in Dept of Orthodontics at Pandit Deendayal.Upadhyay Dental, College, Kaegaon, Solapur, India

²Sr.lecturer in Dept of Oral Medicine & Radiology at Pandit Deendayal, Upadhyay Dental college, Kaegaon, Solapur, India

³P.G student in Dept of Oral Medicine & Radiology at Pandit Deendayal, Upadhyay Dental college, Kaegaon, Solapur, India

⁴5.M.D.S Prosthodontics

Accepted 15 Feb 2016, Available online 20 Feb 2016, Vol.4 (Jan/Feb 2016 issue)

Abstract

Mandibular condyle aplasia is an incongruity usually manifests in association with a variety of syndromes. It is considered as an extremely exceptional stipulation, if it is not usually seen in union with any other developmental anomalies. The incidence is expected 1 in 5600. It occurs due to growth instability in the development of condyle in utero late in the first trimester. The case here reported with malaligned teeth and wants to get it corrected. He had anterior teeth proclined with anterior deep bite. On radiographic examination we noticed absence of right side condyle. The etiology of this condition was unknown, on clinical and radiographic examination we revealed complete absence of condyle on right side. Proper diagnosis along with differentiation from the syndromic cases is important. The aim of this article is to present a peculiar type of mandibular asymmetry with non syndromic condylar aplasia.

Keywords: Condyle, Syndrome, Aplasia, Mandibular Asymmetry, Growth Instability.

Introduction

TMJ is made up of the mandibular condyle and the articular eminence of the temporal bone. The condyle is very particular because the idiom of mandibular growth is provided by mandibular condyle. At birth the joint is largely undersized, in contrast to other diarthrodial joints. [1,2].

The TMJ first becomes apparent in the 8th week of gestation, when two separate areas of mesenchymal blastemas appear near the eventual location of the mandibular condyle and glenoid fossa .[1,2]. Bone and cartilage are first seen in the mandibular condyle at about the 10th gestational week. First condylar blastema build up. The mandibular condyle cartilage, the aponeurosis of the lateral pterygoid muscle, and the disc and capsule component joint are derived from condylar blastema . The temporal blastema next to develop and sooner or later forms the articular surface of the temporal component and the structures of the upper portion of the joint. The mandibular condyle and temporal blastemas begin their growth at comparatively distant sites; they then travel towards each other as the joint develops by the 12th week. Fibrous connective tissues swathe the articular surfaces of both the mandibular condyle and temporal bones at birth. Later, this tissue is leisurely transformed to fibrocartilage as the fossa deepens and the mandibular condyle develops under functional influences [3, 4].

Aplasia or hypoplasia or hyperplasia of the mandibular condyle may result due to growth instability in the augmentation of mandibular condyle that by and large take place in utero late in the first trimester .It is not discernible at birth and seems to be steadily acquired during growth [5]. Mandibular condyle aplasia is a tenure used to illustrate total absence of the condyle. Prior the term 'agenesis' was used, telltaling absence of an organ but it has now been changed by the term aplasia i.e. lack of development of a tissue, as the condylar cartilage is considered a tissue to a certain extent than an organ. Condylar aplasia is due to the growth and developmental abnormality of the TMJ [6].

Case report

A 29 years male came to the department of oral medicine and radiology with chief complaints of proclined teeth in upper front region of jaw since birth. The patient was under impression that as the permanent teeth will erupt , all teeth will get aligned. (fig1,2). He noticed that due to proclined teeth he was unable to close the lips.(fig 2). He had undergone extraction of 36,37 2 years back due to caries..He also had undergone RCT and crown prosthesis with 15 before 1 year. (fig 3,4) and was told to get proclined teeth corrected. Due to unfavorable socioeconomic conditions, the treatment was not done by the patient. There was facial asymmetry with bird face appearance. There was pain on vertical percussion

cariously destracted 27. The maxillary anterior were proclined with anterior deep bite.(fig 2). On opening the mouth there was deviation on right side. TMJ movements were not appreciated on the right side. The gingiva was light pink, resilant with gingival recession with lower anterior and 13,23 in upper anterior.

Investigation

Orthopantomogram (OPG) revealed total absence of the condylar head and the neck on the right side (fig 5). The mandibular angle was placed abnormally close to the base of the skull on the affected side also the head of the condyle on the left side appeared more rounded than normal. There was edentulous area in 36,37,47 region. Impacted 48. On these clinical and radiographic finding we gave the diagnosis of unilateral condylar aplasia along with anatomical facial asymmetry was established .

The patient was then further examined for the presence of any other syndromic features which are usually associated with condylar aplasia, but no syndromic features were apparent

Differential Diagnosis

Hemifacial microsomia was ruled out as no deficit in the neuromuscular pattern or in the soft tissues was present. Other types of acquired mandibular hypoplasia, such as rheumatoid arthritis and Parry Romberg syndrome were also ruled out as no definite clinical history or radiological features were present in our patient. In view of above findings this case was suspected of having a peculiar type of non syndromic condylar aplasia on right side rather than a syndromic one.

Discussion

The development of TMJ launch at 8th intrauterine week. The preliminary functions of mouth opening movements start appearing by the 20th week at some point in fetal stage, but the development course continues until the 12th year of life[7].

The congenital misshapenness and developmental aberrations of the mandibular condyle can be classified as hypoplasia or aplasia, hyperplasia, and bifidity. (6).Due to various craniofacial abnormalities Hypoplasia or aplasias of the mandibular condyle which may point towards underdevelopment or non-development coupled .These may be either congenital or acquired [5].

According to Shafer et al “congenital hypoplasia that is idiopathic in origin is portrayed by unilateral or bilateral underdevelopment of the condyle beginning early in life.” In these cases (eg, dysostosis otomandibularis), the condyle for the most part is small. Secondary or acquired condylar hypoplasia may be caused by local factors (trauma, infection of mandibular bone or middle ear or irradiation) or by systemic factors (infection, toxic agents, rheumatoid arthritis, mucopolysaccharidosis). Either

primary or the secondary hypoplasia may be unilateral or bilateral. [8,9].

Congenital (primary) condylar hypoplasia is illustrated by unilateral or bilateral underdevelopment of the mandibular condyle. This usually occurs as a part of some systemic circumstance originating in the first and second branchial arches, such as Mandibulofacial dysostosis (Treacher Collins syndrome), which demonstrates the traits like Mandibular hypoplasia, hypoplasia/aplasia of the zygomatic arch, coronoid process and condyle, underdeveloped/malformed ears, ocular anomalies, hearing loss . Hemifacial microsomia (first and second branchial arch syndrome) it embraces skeletal traits like underdevelopment of the temporomandibular joint, mandibular ramus and other traits includes underdevelopment of ear and mastication muscles, hearing loss, cardiac anomalies. Oculoauriculovertebral syndrome (Goldenhar syndrome) it is the most relentless form of microfacial microsomia with eye tumours, fused spine. Oculomandibulodyscephaly (Hallermann- Streiff syndrome) shows skeletal and other traits like Dyscephalia and bird face, hypoplastic mandible/ramus, missing condyle ,cataract, microophthalmia, skin atrophy, dental anomalies. Proteus syndrome illustrates atypical growth of the bones, cranial hyperostosis thickening of skin, vascular anomalies, verrucous epidermal naevi. Morquio syndrome contains abnormal development of bones, including the spine, a prominent lower face, respiratory, cardiac, ocular and hepatic abnormalities, enamel hypoplasias [5,10].

Acquired condylar hypoplasia takes place if the condyle is offended during active growth, because of which development may be detained. The most widespread causes are mechanical injury, such as trauma, infection of the joint or sometimes the middle ear infection. Childhood rheumatoid arthritis,radiotherapy and parathyroid hormone-related protein deficiency which impinge on bone formation and chondrocyte differentiation Latest reports have publicized that various extracellular matrix proteins, such as transforming growth factor-b (TGF-b) is accountable by affecting Meckel's cartilage for normal mandibular development[11] whereas some instigators assert that mandibular condyle deficiency can occur with no defined aetiology[12]. Aplasia of the mandibular condyle without any other facial malformations is an extremely rare condition [13].

The cases of non syndromic mandibular condyle aplasia have been in the past reported by Krogstad [6], Prowler and Glossman [14], Akihiko et al. [15], Santos et al. [12], Bowden Jr. and Kohn [16], Canger and Celenk [17] and so forth.The case presented here is in consistant with condylar aplasia without any other features reminiscent of any syndrome. The total absences of the condyle and glenoid fossa on the right side in our case comprise an evidence that the blemish originated in the prenatal period.

The management could be a costochondral graft transplant, if at all probable prior to the growth spurt,

orthognathic surgery at the end of the growth period, or both [18]. Krogstad stated that effective outcome were obtained through the application of a form of orthodontic activator which aimed to swing the mandible to the unaffected side and promote formation of a mandibular condyle, albeit irregular in shape [6]. Surgery is habitually mandatory, but the timing and regimen of this choice is still an issue to be resolved [17].



Fig.1 Profile pic



Fig.2 pt pic at occlusion



Fig.3 mandibular teeth



Fig.4 maxillary teeth



Fig.5 OPG

References

- [1]. 1] D. Buchbinder and A. S.Kaplan. Biology, in Temporomandibular Disorders Diagnosis and Treatment. *A. S. Kaplan and L. A.Assael, Eds*: 1954;11–23.
- [2]. 2] J. W. Choi, J. T. Kim, J. H. Park . gp130 is important for the normalmorphogenesis of Meckel's cartilage and subsequent mandibular development. *Experimental & Molecular Medicine*, 2007: 39; 295–303.
- [3]. J. F. Cleall, .Postnatal craniofacial growth and development. *Oral and Maxillofacial Surgery D. M. Laskin*, :1;70–107.
- [4]. S. Pruzansky.Postnatal development of craniofacial malformations, *Journal of Dental Research*, 1968: 47(6); 936.
- [5]. K. Kaneyama, N. Segami, and T. Hatta.Congenital deformities and developmental abnormalities of the mandibular condyle in the temporomandibular joint. *Congenital Anomalies*, , 2008: 48(3); 118–125
- [6]. Krogstad O. Aplasia of mandibular condyle. *European Journal of Orthodontics* 1997;19:483- 489.
- [7]. Buchbinder D, Kaplan AS Biology. In: Kaplan AS, Assael LA (eds). *Temporomandibular Disorders Diagnosis and Treatment*.Saunders Philadelphia, PA:1991;11–23.
- [8]. Joel Ferri, Eric Lemier, Marie-Madeleine Baralle.Severe Congenital Hypoplasia of the Mandibular Condyle—Diagnosis and Treatment: A Report of 2 Cases.J *Oral Maxillofac Surg* 2006; 64:972-980.
- [9]. Hovel1 JH: Condylar hyperplasia. *Br J Oral Surg* 1963;1:105.
- [10]. EM Canger and P C, elenkAplasia of the mandibular condyle associated with some orthopaedic abnormalities: a case report .*Dentomaxillofacial Radiology* (2012) :41; 259–263
- [11]. Shibata S, Suda N, Fukada K, et al. Mandibular coronoid process in parathyroid hormone- Related protein-deficient mice shows ectopic cartilage formation accompanied by abnormal bone modeling. *Anat Embryol (Berl)* 2003; 207; 35–44.
- [12]. Santos KCP, Dutra MEP, Costa C et al. Aplasia of mandibular condyle: case report. *Dentomaxillofac Radiol* 2007; 36: 420–22.
- [13].S. Ozturk, M. Sengezer, S. Isik, D. Gul, and F. Zor.The correction of auricular and mandibular deformities in auriculocondylar syndrome. *Journal of Craniofacial Surgery*, 2005:16(3); 489–492.
- [14].J. R. Prowler and S. Glassman.Agenesis of the mandibular condyles. Diagnostic findings and treatment of deformity by polyethylene implant. *Oral Surgery, Oral Medicine, Oral Pathology* 1954: 7(2); 133–139.
- [15].I. Akihiko, T. Ritsuo, F. Junichi, I. Nobuyuki, and T. Yutaka.Agenesis of bilateral mandibular condyle- report of a case. *Journal of the Japanese Stomatological Society*. 2006:55; 187–192. C. M. Bowden Jr. and M.W.Kohn.Mandibular deformity associatedwith unilateral absence of the condyle.*Journal of OralSurger.*, 1973: 31(6); 469–472
- [16].E. M. Canger and P. Celenk.Aplasia of mandibular condyle associated with some orthopedic abnormalities: a case report.*Dentomaxillofacial Radiology*.2011;1–5.
- [17].K.Vargervik and L. B. Kaban.Hemifacial microstomia diagnosis and management. *Modern Practice in Orthognathic and Reconstructive Surgery*, W. H. Bell. 1992: 2; 1533–1560.