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Developmental Temporomandibular Joint Disorders In Children

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ABSTRACT: Temporomandibular joint is a diarthrodial, synovial joint formed by the articulation of condyle with the glenoid fossa. It is the only joint in humans with fibrocartilage covering the articular surfaces. Growth of TMJ continues even after birth. TMJ development continues even after birth. Developmental problems such as condylar hypoplasia, hyperplasia, and bifid condyle originate from any disruptions or damage during its development. With correct diagnosis and treatment plan, these disorders are addressed by a multidisciplinary team that includes pediatric dentists.

KEYWORDS:TMJ, condyle, congenital, hypoplasia, hyperplasia, diarthrodial joint.

I. INTRODUCTION

Temporo-mandibular joint (TMJ) is a bilateral, synovial joint formed by the articulation of mandibular condyle with the glenoid fossa of temporal bone[1]. The TMJ development starts around the 7th week of inutero[2]. Thisjoint is significantly underdeveloped at birth, as fetal growth lags in terms of appearance and progression[3]. Fibrous connective tissue covers the articular surfaces of the joint at birth,which slowly gets converted to fibrocartilage under functional influences like mastication[4]. TMJ is considered a ginglymus diarthrodial joint that allows for both translation and rotation movements. TMJ deformities in-utero is most common in the late first trimesterand has adeleterious effect on mastication and speech functions. The purpose of this article is to bring information about the basis of developmental disorders affecting the joint and its functions.

II. CLASSIFICATION

Kaneyama and colleagues (2008) classified developmental disturbances as follows[5],

1. Hypoplasia or aplasia of mandibular condyle
- Primary condylar aplasia and hypoplasia
- Secondary condylar hypoplasia
2. Hyperplasia
3. Bifidity (double mandibular condyle, double-headed condyle)

Condylar Hypoplasia And Condylar Aplasia: Condylar aplasia is the failure of development of the mandibular condyle, and condylar hypoplasia is the underdevelopment of the condyle[6]. Aplasia and hypoplasia can be either congenital or acquired, and may occur unilaterally or bilaterally[7].

Congenital hypoplasia / primary hypoplasia is characterized by unilateral or bilateral underdevelopment of condyle. This usually occurs as a part of systemic conditions originating from first and second branchial arches i.e, disturbances to condylar cartilage results in congenital hypoplasia[5].

Acquired or secondary condylar hypoplasia occurs when condyle is injured during active development resulting in arrested growth of condyle. Developmental hypoplasia arises because of the lack of migration of neural crest cells to the joint area, because of a lack of proliferation of these cells or possibly their destruction[2]. Disturbances are caused by local factors (trauma, infection of the mandibular bone or middle ear, irradiation)[5,6] or by systemic factors (toxic agents, juvenile arthritis, mucopolysaccharidosis)[7,8].

Clinical features: In unilateral condylar hypoplasia or aplasia, the continued growth of the contralateral side causes deviation toward the affected side (facial asymmetry) and results in a cross-bite relationship of the teeth. On the affected side, the

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ramus and body of the mandible remain underdeveloped. In bilateral condylar hypoplasia or aplasia, crossbite is present on both sides with no deviation. Bird face, micrognathia is the prominent feature[6].Imaging:Panoramic radiograph is used for initial imaging[9]. 3D CBCT, series of lateral cephalographs provides an advantage over conventional radiography to evaluate theprogression of the disease[10].

Radiographic features: The condyle will be normal in shape and structure but is diminishedin size, with a proportionally small mandibular fossa. The ramus and mandibular body on the affected side may also be small, resulting in a mandibular asymmetry The antegonial notch is deepened.

Treatment: Treatment of condylar aplasia or hypoplasia is difficult since there is no available means of stimulating its growth locally or compensating for its failure. Immediate treatment must be instituted in life-threatening conditions i.e, infants with condylar aplasia who exhibit respiratory difficulty due to obstruction of the airway by falling back of the tongue may require tracheostomy[6].Early treatment intervention to be performed by grafts to prevent worsening of the condition. Costochondral rib grafts are transplanted as growth center replacement to facilitate mandibular growth. Distraction osteogenesis is a technique for lengthening the bone as an alternative for grafts[11]. In severe cases, osteoplasty can be considered.

Conditions that show condylar hypoplasia/aplasia includes[6],

- Treacher Collins syndrome
- Oculo-auriculo vertebral syndrome
- Hurler syndrome
- Hemifacial microsomia
- Nager syndrome

Treacher Collins syndrome (Mandibulo-facial dysostosis)[5,6,12]: Mandibulofacial dysostosis (MFD) is an autosomal dominant inherited disorder characterized by bilaterally symmetrical abnormalities derived from the first and second branchial arches. This results from the destruction of neural crest cells before they migrate to form the facial processes.When the syndrome is fully expressed, the diagnosis is made based on clinical characteristics alone, as follows.

(i) The facial profile is convex, reflecting hypoplasia of zygoma, maxilla, and mandible with variable effects on the temporomandibular joints and muscles of mastication

(ii) The mandible is underdeveloped, resulting in a retruded chin. These facial features have been described as birdlike or fishlike in morphology.

(iii) The eyes have a lateral downward sloping of the palpebral fissure.

(iv) External ears are often absent, malformed, or malposed.

(v) Hearing is impaired as a result of the variable degrees of hypoplasia of the external auditory canals and ossicles of the middle ears.

Hemifacial microsomia(first and second branchial arch syndrome)[6,12]:Hemifacial microsomia (HM) is characterized by aplasia or hypoplasia of the mandibular ramus and/or condyle. The clinical picture ranges from a slight facial asymmetry to severe underdevelopment of half of the face with orbital implications and a partially formed ear. The chin and facial midline deviate toward the affected side. The masticatory muscles are hypoplastic on the affected side.

Oculo-auriculo vertebral syndrome (Goldenhar syndrome)[5,12]:A variant of hemifacial microsomia. OAVS includes the characteristic hypoplastic mandible along with epibulbar dermoids and vertebral anomalies. It is not limited to the face and includes skeletal, genitourinary, renal,and cardiac abnormalities.

Hurler syndrome[6,12,13]: Hurler's syndrome belongs to the category of mucopolysaccharidosis (MPS). These errors are caused by defects in the lysosomal enzyme (a-L-iduronidase) that degrade mucopolysaccharides. Patients with Hurler's syndrome have growth and mental retardation, corneal cloudinggargoyle-like facies with hypertelorism, a prominent forehead and supraorbital ridges, scaphocephaly, flattening of the nasal bridge,large nostrils, and a short neck. Lack of development of the mandibular condyles has been noted unilaterally or bilaterally, allowing limited rotation.

Nager syndrome[12]:Nager syndrome is a rare form of acrofacial dysostosis, The condition is characterized bycraniofacial and upper limb abnormalities Nager syndrome is downward slanting of the palpebral fissures. In addition to this, affected individuals exhibit micrognathia, midface retrusion, cleft palate, and external ear anomalies.

HYPERPLASIA OF THE MANDIBULAR CONDYLE

Condylar hyperplasia is excessive unilateral growth of the mandibular condyle leading to facial asymmetry and occlusal

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disturbance[14]. This occurs during disturbances in the growth pattern of the mandibular condyle because the condyle is considered to be a site for compensatory growth and adaptive remodelling[3].This is observed in patients between 10-30 years of age[14]. The etiology is unclear, it is suggested that neoplasm, trauma, infection, abnormal loading, hormonal influences, hypervascularity, heredity, or mild chronic inflammation promotes the development of condyle or other neighboring tissues resulting in a condition similar to proliferative osteomyelitis[7,9,15]. The mechanism may be overactive cartilage or persistent cartilaginous rests, which increase the thickness of the entire cartilaginous and precartilaginous layers[10].

Clinical features: The patients usually exhibit a unilateral, slowly progressive elongation of the face with a deviation of the chin away from the affected side with resulting cross-bite malocclusion. The enlarged condyle may be clinically evident or felt on palpation and presents as a striking feature in both coronal and sagittal sections[9]. The facial deformities associated with condylar hyperplasia involve the formation of a convex ramus on the affected side and a concave shape on the normal side[10].

Imaging:Panoramic radiographymay be used as an initial imaging method for identifying condylar hyperplasia, but for quantitative evaluation and follow-up, 3D imaging using CT or CBCT is necessary[9]. Bone scans are used as active condylar hyperplasia exhibits increased uptake of radionuclide (Technetium 99m diphosphate) on the hyperplastic side[5].

Radiographic features: The condyle may appear relatively normal but symmetrically enlarged, or it may be altered in shape (e.g., conical, spherical, elongated, lobulated) or irregular in outline. It may be more radiopaque because of the additional bone present. The condylar neck may be elongated and thickened and may bend laterally when viewed in the coronal (anteroposterior) plane[10].

Histopathologic features: The histopathology picture shows abnormally rapid chondrogenesis with subsequent ossification. Typical histological findings have also included the presence of an uninterrupted layer of undifferentiated germinating mesenchymal cells, hypertrophic cartilage, and islands of chondrocytes in the subchondral trabecular bone[16].

Treatment: Treatment consists of orthodontics combined with orthognathic surgery which is performed before condylar growth is completed, to avoid functional problems (mastication and speech) and worsening esthetic disfigurement[10].Resection of the condyle (condylectomy), condylar shave (condyloplasty) is performed to ensure removal of growing cartilage[11].

BIFID CONDYLE:

Bifid mandibular condyle (BMC) is an uncommon condition and is characterized by the duplicity of the mandibular condyle head[17]. Two theories are postulated for its etiology. One theory states that bifidity originates in an embryo, where there is the limited blood supply to the condyle. Another theory suggests that bifidity could be due to birth trauma or fracture of the condylar head[6]. Teratogenic drugs, trauma,infection, radiation exposure are the various other reasons for bifidity[18]. It is characterized by a varying depth of groove or depression around the midline of the condylar head. This depression may be visible on coronal or sagittal orientation[10]. A deep groove may result in an appearance of the duplicity of the condylar head.

Usually, bifidity is unilateral. To accommodate the altered condylar morphology, mandibular fossa remodels by the expression of vascular endothelial growth factor promoting neovascularization and new bone development[19].

Clinical features: Usually asymptomatic. Pain, restricted movements, limited mouth opening can be present when symptomatic. The diagnosis relied on more radiological findings rather than clinical evidence[6].

Imaging: Imaging of bifidism is better detected on CT coronal or sagittal sections of cross-sectional imaging and 3D reconstruction

Radiographic features: A depression or notch is present on the superior condylar surface,giving the anteroposterior silhouette a heart shape. In more severe cases a duplicate condylar head is present in the mediolateral plane. The orientation of the bifid condyle may be anteroposterior or mediolateral.[10].

Treatment: Patients with bifid condyles do not require any treatment unless pain or functional impairment is present. Patients with associated articular ankylosis might need surgical condylectomy or arthroplasty[14].

III. CONCLUSION

Several disorders can cause congenital growth disruptions in the TMJ. In today's world, these diseases are managed by a multidisciplinary team dealing with anatomical, functional, and psychological issues. Proper management necessitates a detailed investigation and precise

planning. Its the responsibility of pediatric dentists to inspect as well as make decisions whether to observe, treat or refer. It is always necessary to coordinate the best care plan possible for each patient and give them the best possible treatment.

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