CONDYLAR HYPERPLASIA - AN INSIDIOUS DEVELOPMENTAL DISORDER OF MANDIBLE

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ABSTRACT

Condylar hyperplasia is a developmental disorder of mandible characterised by accelerated growth of condylar head of mandible either unilateral or bilateral. Rate of abnormal growth of the condyle determines the severity of facial asymmetry causing distress and rarely pain. It has a multifactorial etiology with increased prevalence in females. Two basic growth vectors occur with CH: horizontal growth vector (type 1) and vertical growth vector (type 2). The prevalence ratio between types 1 and 2 is approximately 15:1. Type-1 condylar hyperplasia increases anteroposterior dimension of mandible causing crossbites and Type-2 increases vertical height of mandible causing openbite. Facial asymmetry due to growth disturbances of the mandible mostly requires orthognathic surgical corrections, and asymmetries treated by this method have a high success rate and long-term stability. However, in very severe cases of facial asymmetry associated with condylar hyperplasia (CH), a partial or complete condylar resection may be necessary for correction of the condition.

KEYWORDS: Condylar hyperplasia, anteroposterior dimension, mandible characterised.

INTRODUCTION

Condylar hyperplasia is a developmental disorder of unknown etiology characterized by persistent or accelerated growth of the condyle even after its growth caesation. Growth eventually stops without treatment. It often results in appreciable facial asymmetry and so can be extremely distressing for the patient. Central to its management is the assessment of the presence or absence of active growth in the condylar head. Condylar hyperplasia is a pathological condition that presents a challenge to both orthodontists and oral and maxillofacial surgeons because of its progressiveness and tendency of causing a severe dentofacial deformity. Adams in 1836 first described Condylar Hyperplasia as overdevelopment of the mandible creating significant functional and esthetic deformities.[1] Rushton in 1946 first reviewed 29 reported unilateral cases, and by 1968, a total of 150 cases had been reported in the literature, most of which were isolated. However, Condylar Hyperplasia is much more evident than clinicians realize, and failure to recognize this condition can result in unfavourable functional and esthetic treatment results.[2]

ETIOPATHOGENESIS

CH at the temporomandibular joint (TMJ) is a rare pathology that occurs at the head of the condyle leading to facial asymmetry affecting occlusion and possible association with pain and dysfunction. It may occur at any age and is more prevalent in females. Till date the pathogenesis of CH affecting the TMJ remains obscure, with factors ranging from a reactive growth response, trauma, excessive proliferation in the repair process, increase in functional load or a Y-linked or autosomal dominant trait being implicated in the development of CH.[3]

CH usually develops during puberty and rarely begins ing of a TMJ after the age of 20. The identification of sex hormone receptors in and around the temporomandibular joint and the pubertal onset of CH strongly suggest a hormonal influence in the etiology. Trauma, infection, heredity, intrauterine factors, arthrosis and hypervascularity have also been implicated as causative factors.[4,5]

CLINICAL & RADIOGRAPHICAL FINDINGS

About one third of bilateral CH patients have a family history of the condition. Two basic growth vectors occur with CH: horizontal growth vector (type 1) and vertical growth vector (type 2). The prevalence ratio between types 1 and 2 is approximately 15:1. Distinct radiographic and clinical features differentiate the 2 types. A normal condyle is approximately 15-20 mm in mediolateral dimension and 8-10 mm wide anteroposteriorly. In type 1 CH, although the condyle retains a relatively normal architecture, an increase in
length of the condylar head, neck, and mandibular body is evident. Type 2 CH may demonstrate a condylar head and neck much larger in length and diameter than a normal condyle, prominent medial and lateral poles, but the condylar surface is smooth with a uniform contour.

Unusual morphological characteristics of the condylar head, such as bony outgrowths, globular enlargements, saddle-shaped cavities, hockey-stick-like exostoses, and bulbous enlargements, are most likely not CH but, rather, an osteochondroma or other pathological condition of the condyle.

Slowly progressive unilateral enlargement of the head and neck of the condyle causes facial asymmetry, cross bite malocclusion, and shifting of the midpoint of the chin to the unaffected side. The patient may have a prognathic facial profile with convexity in the lower border of the mandible on the affected side. Chondroma and osteochondroma may cause similar symptoms and signs, but they grow more rapidly and may cause even greater asymmetric condylar enlargement.

On x-ray, the temporomandibular joint may appear normal, or the condyle may be proportionally enlarged and the mandibular neck elongated. Computerized tomography is usually done to determine whether bone growth is generalized, which confirms the diagnosis, or localized to part of the condylar head. If growth is localized, a biopsy may be necessary to distinguish between tumour and hyperplasia. [6]

Histological observations of the proliferative layer in a hyperplastic condyle demonstrate a greater thickness in some areas and lesser in others, but cartilage-producing cells are everywhere at its lower border. In some regions, the cartilage is very thick and is being actively generated and replaced by new bone. The activity of the proliferative layer appears to regulate the rate at which the condyle and the condylar neck will grow which is formed from the condyle by remodelling. In normal condyles, the formation of cartilage from the proliferative layer and the replacement of cartilage by bone cease by approximately 20 years of age. The narrow cavity is entirely occluded from the remaining cartilage by the closure of the bone plate. The inability of this plate to close in the presence of an active proliferative layer may be a major etiologic factor in CH and may correlate to our observation that cessation of growth related to CH may not occur until the middle to late twenties. Conditions that initiate excessive accelerated mandibular growth after the age of 20 are most often related to an osteochondroma, an osteoma, or another type of proliferative condylar pathology. [7]

DISCUSSION

Type-1 Condyler Hyperplasia
Type 1 CH is not well accepted as a form of CH by many clinicians. It is usually termed as symmetrical or deviated prognathism, laterognathia, or mandibular hyperplasia. However, the basic cause of many mandibular prognathic cases is type 1 CH, i.e., excessive mandibular growth originating in the mandibular condyles. Type 1 CH occurs with equal frequency in males and females, as well as unilaterally and bilaterally. These patients usually demonstrate a Class I or mild Class III skeletal and occlusal relationship before the onset of CH and develop into a Class III or severe Class III relationship, respectively, as their growth accelerates. Type 1 CH rarely occurs in skeletal Class II patients.

Common clinical and radiographic characteristics observed in bilateral, symmetrically growing type 1 CH patients usually include: Increased length of the condylar head and neck, without a significant volumetric increase in the size of the condylar head. Accelerated mandibular growth, Mandibular growth continuing beyond the normal growth years, Worsening Class III skeletal and occlusal relationship, Worsening esthetics, Obtuse gonial angles, Decreased angulation of the lower incisors and possibly increased angulation of the upper incisors (dental compensations), Decreased vertical height of the posterior mandibular body, High mandibular plane angle and Narrow anteroposterior (A-P) dimension of the symphysis in more severe cases.

Additionally, unilateral cases may have: TMJ articular disc displacement, Worsening facial and occlusal asymmetry, with the mandible progressively shifting toward the contralateral side, Unilateral posterior crossbite on the contralateral side, Transverse bowing of the mandibular body on the affected side; and transverse flattening of the mandibular body on the contralateral side.

Type-2 Condyler Hyperplasia
Type 2 CH is often known as hemimandibular hypertrophy. However, it can be a form of CH and usually occurs unilaterally. More severe is the pathology, greater is the clinical asymmetry and the degree of morphological alterations. Most patients have a low mandibular plane angle before the onset of CH.

Specific characteristics of type 2 CH include: Unilateral elongation of the face, causing facial asymmetry and worsening esthetics, Increased length, size, and diameter of the condylar head and neck, Increased vertical height of the entire mandible on the involved side (except for the coronoid process), Open bite on the involved side, Compensatory vertical overdevelopment of the maxilla on the involved side and dental compensations.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for type 1 CH includes Maxillary hypoplasia, Mandibular prognathism without CH (patients start out as skeletal Class III in early childhood and maintain harmonious growth between maxilla and mandible, with growth ceasing at the normal age), Dislocation of the condyles anterior to the articular eminence, Dental interferences or habitual posturing.
causing anterior positioning of the mandible, Acromegaly, Macroglossia and other TMJ pathology such as osteochondroma, osteoma, or contralateral condylar resorption.

The differential diagnosis for type 2 CH includes: Osteochondroma or other condylar enlarging pathology, Hemifacial hypertrophy, Contralateral condylar hypoplasia, resorptive TMJ pathology, or condylar fracture; and other pathology such as unilateral fibrous dysplasia, Sturge-Weber syndrome, arteriovenous malformation, hemangioma, or lymphangioma.

The most common and similar pathology to type 2 CH is an osteochondroma of the condyle. The radiographic appearance of the two processes can be similar, unless the osteochondroma is large, with exophytic growths. Clinically, the condylar surface of an osteochondroma is lumpy and irregular, unlike the smooth surface seen in CH. Histopathologically, unlike CH, an osteochondroma usually has islands of cartilage within the medullary bone of the condyle; however, in slow growing lesions, these may be difficult to identify. Treatment of an osteochondroma usually requires a complete condylectomy and TMJ reconstruction to stop the abnormal growth, whereas CH requires only a high condylectomy (removal of the top 3 to 5 mm of the condyle).[8]

INVESTIGATIONS
Active CH growth can usually be determined by worsening functional and aesthetic changes with serial assessments (preferably at 6- to 12-month intervals) consisting of: Clinical evaluation, Dental model analysis with orthodontically trimmed models or models mounted in centric relation and Radiographic evaluation by superimposition including Lateral cephalometric radiographs, Frontal cephalometric radiographs (for unilateral CH cases) and Lateral cephalometric tomograms showing the TMJ, mandibular ramus, and the body.

The normal pubertal mandibular growth rate is approximately 2 mm for males and 1.5 mm for females, measuring from condylion to Point B. Bone scanning with Technetium 99M pyrophosphate or Technetium 99M methylene diphosphonate may detect active growth in the condyle. This may be most effective in unilateral cases, especially if applied after the normal growing years, when condylar growth should have ceased. We have found bone scans to be inconclusive in younger patients and those with slow-growing CH. Hand wrist films have no value in CH because the mandible can continue to grow well beyond the normal growth years.

Ideally before finalising a treatment plan for condylar hyperplasia the most preferred investigations include panoramic imaging, cephalogram, computed tomography (axial & coronal) and 3Dimensional reformatted images. Scintigraphy is also a form of investigation which determines high uptake of radiographic traces in the condylar area.[9] After a preferential investigations orthognathic work up is done which includes clinical photography, study models, jaw prototyping and cephalometric analysis.[10]

MANAGEMENT
Larry M.Wolford, Pushkar Mehra, Oscar Reiche-Fischel, Carlos A. Morales-Ryan, and Patricia García-Morales, stated that treatment usually includes condylectomy during the period of active growth. If growth has stopped, orthodontics and surgical mandibular repositioning are indicated. If the height of the mandibular body is greatly increased, facial symmetry can be further improved by reducing the inferior border.

It has been reported that performing isolated orthognathic surgery on patients with coexisting TMJ pathology can lead to unsatisfactory results, such as relapse, malocclusion, jaw and facial deformities, TMJ pain, headaches, myofascial pain, and masticatory dysfunction.[11] One solution advocated to avoid these follow-up problems is to perform the TMJ and orthognathic surgeries during the same operation.[12] Recently, Wolford described the clinical implications for simultaneous TMJ and orthognathic surgery for patients suffering from articular disk dislocation, reactive arthritis, adolescent internal condylar resorption, condylar osteochondroma or osteoma, end-stage of TMJ pathology, and condylar hyperplasia. The surgical procedures can also be done separately, beginning with the TMJ surgery and then conducting the orthognathic surgery about six months later, but this requires increased expenses and a second term of hospitalization.

In some services, plain radiography and bone scintigraphy (BS) are used for the standard evaluation of patients suspected of having CH. The addition of BS to the diagnostic evaluation of patients suspected of having CH is useful since it identifies patients with an active condylar growth center who should undergo high condylectomy. Failure to recognize this condition can result in unfavorable functional and aesthetic treatment results. In the present case report, BS was not included in the evaluation because active CH usually develops during puberty and rarely begins after the age of 20 year old. The clinical findings, in addition to the CT scans, guided us to the diagnosis of no active CH. The decision to perform high condylectomy was made due to the signs and symptoms of end-stage TMJ pathology resulting from CH and to assist in the correction of the mandibular plane.

CH activity as demonstrated by Bone Scintigraphy is also strongly correlated with histological findings. The histological findings of the presented case are compatible with those for a normal condyle, confirming the clinical findings of no active CH. When active, the histopathology of CH shows the presence of abnormal
large masses of hyaline cartilage surrounding large cells and new cartilage formation, along with the constant presence of mesenchymal germinal cells and cartilage islands in the bone under the fibrocartilage. In the vertical type of active CH, cartilage maturation layers and increased growth are also noticeable, and inclusions of cartilaginous tissue with glove fingers extending into the underlying cancellous bone have been described.

Conducting orthognathic and TMJ surgery simultaneously is still controversial. Panula et al. carried out a prospective study in 60 subjects on the effects of orthognathic surgery on signs and symptoms of TMJ dysfunction. These authors reported a significant reduction in pain levels with the orthognathic treatment, and the risk for a new TMJ dysfunction was reported to be extremely low (6.7% or 4 patients), with all the occlusion parameters remaining stable through the follow-up period. In their short review, Stavropoulos and Dolwick concluded that TMJ surgery should be reserved for the small number of patients whose TMJ symptoms do not resolve with conventional orthognathic surgery, and TMJ surgery should be performed only when indicated.

The condyle and ramus can also be affected, showing enlargement caused by hemimandibular hyperplasia and condylar tumors.\textsuperscript{[13,14]} Wolford et al. in a retrospective study, evaluated the efficacy of combining high condylectomy and orthognathic surgery for treating CH.\textsuperscript{[15]} Their study presented better results for patients who also had high condylectomy compared to those who had orthognathic surgery alone, indicating that the combination of both procedures is beneficial for patients with active CH.

Facial asymmetry due to growth disturbances of the mandible mostly requires orthognathic surgical corrections, and asymmetries treated by this method have a high success rate and long-term stability.\textsuperscript{[16]} However, in very severe cases of facial asymmetry associated with condylar hyperplasia (CH), a partial or complete condylar resection may be necessary for correction of the condition.\textsuperscript{[17]}

CONCLUSION
Knowledge of the causes of condylar hyperplasia will enlighten its diagnosis differentiating it with possible other diagnosis like osteochondroma, osteoma, or contralateral condylar resorption. Depending upon the pattern of growth of condyle confirmed by the above mentioned investigations, condylar hyperplasia can be managed by either simple orthodontic interventions or surgical management along with orthodontic management.

REFERENCES
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