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IDIOPATHIC CONDYLAR RESORPTION-A REVIEW

Dental Science

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ABSTRACT

Idiopathic condylar resorption (ICR) of the temporomandibular joint (TMJ) is a condition that is often esthetically and functionally altering, characterized by progressive resorption of the TMJ condylar heads, without a known cause. Studies suggest that patients with ICR remain undiagnosed and unrecognized in the orthodontic clinic owing to the poorly understood etiology of the disease and lack of diagnostic tools. ICR often causes occlusal and skeletal changes, TMJ dysfunction and pain, and maxillofacial deformities. Therefore, precise diagnosis of the disease and proper treatment plan is essential.

KEYWORDS

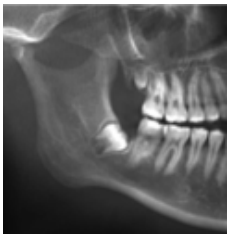
Idiopathic condylar resorption, idiopathic condylysis, Condylar atrophy, Aggressive condylar resorption, Acquired condylar hypoplasia, Progressive condylar resorption, cheerleader syndrome, degenerative disorder.

INTRODUCTION

Idiopathic condylar resorption (ICR) of the temporomandibular joint (TMJ) is a condition that is often esthetically and functionally altering, characterized by progressive resorption of the TMJ condylar heads, without a known cause. It is also referred to as idiopathic condylysis, Condylar atrophy, Aggressive condylar resorption, Acquired condylar hypoplasia, Progressive condylar resorption and cheerleader syndrome.[1] It is a degenerative disease of the TMJ, found mostly in female adolescents and young women. ICR present with loss of condylar mass thereby decreases the height of the ramus and opening rotation of the mandible produces Class II open bite malocclusion.[1]

ETIOLOGY:

Exact cause is unknown . Number of possibilities have been proposed such as hormonal changes, increased mechanical loading from parafunctional habits, trauma, internal joint derangement, orthodontic treatment, orthognathic surgery, occlusal therapy.



PATHOGENESIS:

There are different theories regarding etiology of Idiopathic condylar resorption. The most dominant theory is hormonal theory which is immunologically controlled and arise in genetically susceptible individuals in combination with environmental factors. Sex hormones are thought to modulate biochemical changes within the TMJ, which may then result in condylar resorption. This is further supported by identification of estrogen receptors in female primates.[2]

Estrogen is known to mediate cartilage and bone metabolism in the female TMJ. An increase in receptors may predispose an exaggerated response to joint loading from parafunctional activity, trauma, orthodontics, or orthognathic surgery. In our ICR patient population, the majority of cases occur in young teenage girls in their pubertal growth phase. The sex hormones mediate biochemical changes within the TMJ causing hyperplasia of the synovial tissues that stimulates the production of destructive substrates that initiates breakdown of the ligamentous structures that normally support and stabilize the articular disk in position. This allows the disk to become anteriorly displaced.[3,4] The hyperplastic synovial tissues then assume a position around the head of the condyle, further increasing exposure of the condyle to the substrates that create the resorptive phenomena. In this disease process, the condyle shrinks in all 3 planes of space. The condylar resorption appears to occur in the subcondylar bone without clinically apparent destruction of the fibrocartilage on the condylar head and roof of the fossa, unlike the arthritides where the fibrocartilage is destroyed by the inflammatory disease processes. ICR may eventually go into remission. However, if the condyle and the hyperplastic synovial tissues receive excessive loading.[5,6,7]

Whatever the underlying etiology of ICR, all bone loss at the condylar level involves a *common resorptive pathway* that includes cytokine-activated osteoblasts that then recruit and promote the activity of osteoclasts. This in turn results in the secretion of enzymes that are responsible for the breakdown of hydroxyapatite and collagen. The end result is bone breakdown through the common resorptive pathway. In patients with juvenile idiopathic arthritis (JIA), a current treatment approach is to minimize articular bone loss by using pharmacologic drugs that interfere with specific cytokines and enzymes along the common resorptive pathway rather than open joint or joint replacement procedures. This may include non-steroidal anti-inflammatories (NSAIDS) or other drugs such as methotrexate or a newer class of medications called biologicals. Gunson and Arnett point out that a similar approach to ICR may prove to be useful. [8,9,10]

Another theory of the cause of ICR is avascular necrosis of the condyle as a result of the compression of specific vessels that supply the condyle followed by condylysis, loss of condyle height with secondary jaw deformity and malocclusion. Piper and Choung had speculated that pathologic compressive forces of the posterior aspect of the condyle on the ligamentous retrodiscal soft tissues constrict the small vessels, thereby limiting circulation to the condyles with resulting in aseptic necrosis that accounts for the observed condylysis in patients with ICR. [11,12]They went on to hypothesize that either a chronically dislocated non-reducing disc or specific patterns of malocclusion may also cause this cycle of events. As an extension of this thinking, preventative open-joint procedures were offered to alleviate the theoretical compression of the condylar circulation. Those of us that are skeptical of the non-reducing disc cause of ICR point out that the almost universally observed bilateral symmetric simultaneous nature of this condition and its occurrence only in females make the theory an improbable explanation.[13]

ORTHODONTIST IN IDIOPATHIC CONDYLAR RESORPTION:

Patients with condylar resorption have symptoms of TMD and unstable occlusion. Stabilization of TMJ allows clinicians to identify the true mandibular position and make an accurate diagnosis. It may also alleviate the patient's TMD symptoms.(stabilizing splint is used for these purposes). After stabilization, the occlusion on that position should be reevaluated when establishing an orthodontic treatment plan. During treatment, orthodontists should be aware of the stable position of the condyles when moving teeth, and the occlusion should be finalized in relationship to this position.[14,15]

DIAGNOSIS:

Diagnosis can be through Panaromic radiographs, Cephalograms, Serial cephalometric radiographs for diagnosis of active ICR, cone beam computed tomography (CBCT) images and magnetic resonance imaging.

- Common lateral cephalometric radiographic findings in bilateral TMJ ICR includes:

(1) skeletal and occlusal Class II deformity
(2) anterior open bite
(3) high mandibular occlusal plane angle
(4) high mandibular plane angle
(5) decreased vertical height of the ramus
(6) the lower incisors may appear overangulated and
(7) a significant decrease in the oropharyngeal airway can occur in the more severe cases.[16,17]

- Serial lateral cephalograms will demonstrate slow but progressive retrusion of the mandible during the active resorption phase. Unilateral involvement includes

(1) unilateral skeletal and occlusal Class II deformity
(2) vertical height difference at the mandibular inferior border, ramus, and occlusal plane and
(3) an open bite on the contralateral side.
(4) lower midline shift opposite to affected side

Serial P-A cephalograms may show worsening of the asymmetry. Superimposition of serial cephalometric tomograms will help document the presence of active condylar resorption.[18,19]

- Cephalometric tomographic evaluation of the TMJ usually shows a relatively normal or excessive joint space because of hyperplasia of the synovial tissues. The involved condylar head will appear smaller in size, the degree of which will be dependent on length of time since onset of the pathosis and aggressiveness of the disease. There may be some loss of integrity of the cortical bone on the head of the condyle.[20,21,22]

- The MRI findings include the following:

(1) decreased condylar volume
(2) anterior disk displacement with or without reduction on opening
(3) extreme thinness or loss of continuity of cortical bone on the head of the condyle and
(4) often thick amorphous- appearing soft tissue occupying the space between the condyle and fossa .[23]

The degree of deformation and degenerative changes of the disk will be dependent on the length of time of the disk displacement. Determination must be made relative to salvageability of the disk and adequacy of the remaining condyle to withstand normal functional loading and stress forces

- Observations at surgery reveal an anterior or anteromedial displaced disk. When the disk is pulled back into position, the hyperplastic synovial tissues bunch posteriorly . The tissue may appear amorphous in nature, with little vascular component, and usually no inflammation. The glenoid fossa and the condylar head usually have an intact fibrocartilage covering . The articular disk may or may not demonstrate significant deformation and degenerative changes depending on the length of time of the disk displacement. Histologically, the retrodiskal tissues will demonstrate synovial hyperplasia. If the fibrocartilage is absent and erosion present on the condyle and fossa, the disease process is not ICR.[24]

TREATMENT :

Idiopathic condylar resorption is one of the most difficult conditions to treat due to the variability of expression and the unpredictability of existing management options . Although there are very few cases of patients with condylar resorption that can be corrected merely with orthodontic treatment due to the bone discrepancy caused by condylar rebirth, cases have been documented in which conservative orthodontic treatment is performed achieving adaptive condylar remodeling .[25]

A new non-surgical treatment protocol has been proposed which consists of placing an occlusal guard or splint that increases the height of premature contact, use of elastics during the night and physiotherapy to restore normal occlusion which has shown good results. In order to correct bone class II, open bite and aesthetics in patients with condylar resorption, orthognathic surgery or its repetition has been performed according to the case; however, the rate of recurrence is high, and additional reabsorption can occur .[26]

According to some studies, pharmacotherapy for 2-3years for normal opening, stable occlusion and absence of joint pain. Currently there are 5 agents that block TNF-α which are: etanercept, infliximab, adalimumab, certolizumab and golimumab; In addition, the omega-3 supplement has shown beneficial effects. On the other hand, there are authors who recommend in addition to orthognathic surgery, remove hypertrophied synovial tissue surrounding the condyle, reposition and stabilize the articular disc surgically . There is no scientific information that supports this treatment protocol, but its results are stable . Another treatment option is the reconstruction of the temporomandibular joint with total alloplastic prosthesis which, according to the literature, has had good results. There are several therapeutic options for the management of condylar resorption, each with its advantages and disadvantages.[27,28]

CONCLUSIONS

Studies suggest that patients with ICR remain undiagnosed and unrecognized in the orthodontic clinic owing to the poorly understood etiology of the disease and lack of diagnostic tools. ICR often causes occlusal and skeletal changes, TMJ dysfunction and pain, and maxillofacial deformities. Therefore, precise diagnosis of the disease and proper treatment plan is essential.

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