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Case Report

# Pathogenesis of Idiopathic Condylar Resorption

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## Abstract

Idiopathic condylar resorption (ICR) is an uncommon aggressive condition that targets the temporomandibular joint (TMJ). The condition involves partial or complete degeneration of the condylar head of the mandible. It has a higher female-to-male ratio, especially during the puberty growth spurt period. This paper reviews the pathogenesis of this degenerative disease and reports a unique case of a twenty-nine-year-old female who presented with jaw pain and clicking on the left side. Pain was unilateral, extending from the preauricular area to the angle of the mandible. The patient linked the onset of her symptoms to her visit to the dentist, who performed composite restorations on teeth number #45, #46 and #47. She reported that since then her bite had not felt normal, the restorations were high and uncomfortable and pain had developed. A comprehensive clinical examination was performed, and a panoramic radiograph was taken, which demonstrated degenerative radiographic changes. Several factors can potentially contribute to the development of this condition, some of which are still hypotheses. This case supports the reasoning that was suggested in previous studies that increasing muscle tension as a biomechanical phenomenon is most likely the trigger of ICR. The treatment method used was nonsurgical, including home care instructions designed to optimize pain relief.

***Keywords:*** *Idiopathic Condylar Resorption; Progressive Condylar Resorption; Condylar Atrophy; Biomechanical Overloading of the TMJ*

## Introduction

Idiopathic condylar resorption (ICR) is an uncommon aggressive condition that targets the temporomandibular joint (TMJ). The condition involves partial or complete degeneration of the condylar head of the mandible (i.e. flattening and thinning and or loss of height) resulting in a shift in the facial morphology and jaw alignment associated sometimes with the local remodeling of the underlying bone [1-7]. ICR (also known as condylar atrophy, or progressive condylar resorption) is characterized as a progressive destructive noninflammatory disease of the condyle affecting the underlying subchondral bone and articular fibrocartilage [8]. Pathology of the condition includes a series of lyses and repairs leading ultimately to a progressive change in the mass and shape of the condyle [1,2,9]. ICR affects both men and women, with a female predominance. The female-to-male ratio is 9:1, especially in adolescent girls during pubertal growth before the age of twenty, and rarely after the age of forty [10,11]. Medical articles sometimes refers to it as "cheerleader's syndrome" as it targets physically active teenage girls who engage in sports activities and are more likely to be exposed to jaw trauma and psychological stress [12]. One of the earliest written medical articles goes as far back as the early sixties. In it, Burke describes ICR as acquired condylar hypoplasia. It was only in the late seventies that it was distinguished from congenital condylar hypoplasia and documented as a progressive destructive pathology in nature [13,14]. ICR has been associated with many etiological factors such as reactive arthritis, avascular necrosis, infection, internal derangement, traumatic injuries and autoimmune diseases such as rheumatoid arthritis, scleroderma, sys-

temic lupus erythematosus, and Sjogren's syndrome [15-17]. Others hypothesized reduced bone strength, steroidal use, vascular collagen diseases, the restricted blood supply to the condyle (avascular necrosis), and psychological stress as the etiological cause for triggering the disease [18-20]. Hormones are one of the important etiologies presented. Researchers believe that biochemical changes within the TMJ are modulated by sex hormones, estrogen and prolactin and their receptors present on the condyle, bilaminar zone of the meniscus [20]. Wolford., *et al.* hypothesized that the exposure of those receptors to certain inflammatory mediators results in condylar resorption [12]. Biomechanical overloading of the condyles caused by autorotation of the mandible was a significant contributing etiological factor proposed by Phillips and Bell [21]. They hypothesized that biomechanical overloading in the form of trauma, excessive stress applied to the condyle, or orthognathic surgery is the reason behind the absorption phenomenon.

## Case Presentation

We report a case of a twenty-nine-year-old Caucasian female at The Orofacial Pain and Temporomandibular Joint Clinic at a private hospital in Jeddah, Saudi Arabia. The patient presented with a chief complaint of jaw pain and clicking on the left side. Symptoms started gradually, following a visit to the dentist. Dental work included composite restorations performed on teeth number #45, #46 and #47. The patient reported that "her bite felt off" following that dental visit and that "the restorations felt high and been bothering her since then". Pain was unilateral on the left side extending from the preauricular area to the angle of the mandible. Pain was mild to moderate, 4 out of 10 on a visual analog scale (VAS) and it was intermittent in frequency, lasting for minutes, sometimes hours. The quality of the pain was dull aching triggered by function and pressure. No autonomic symptoms were associated with the pain. She also reported jaw clicking and occasional subluxation of the jaw on the left side. The patient reported progressive worsening of pain and occlusion. A review of systems also revealed that the patient was healthy and clear from any known pathology with no known allergies. Her dental history revealed surgical extraction of tooth #38 in 2009. During extraction, the surgical bur broke and became embedded within the alveolar bone. Dentists failed to remove the bur, and it remained embedded. The embedded bur is theorized to be the source of the pathological biomechanical overload to the joint and the etiology behind the ICR in our patient's case. Intraoral examination revealed abnormal teeth wear of the lower incisors and an visible unbalanced occlusion. A horseshoe articulating paper revealed an unbalanced bite with areas of heavy occlusion (iatrogenic from the high crown/high restorations) consistent with the patient's complaint. A musculoskeletal examination included flat and pincer palpation of muscles of mastication and the upper quarter muscles (upper trapezius and sternocleidomastoid muscle). This examination revealed the presence of trigger points on the right-side superficial masseter muscle. TMJ palpation revealed left side moderate tenderness to palpation, 6 out of 10 on a VAS, mild limitation of range of motion and deflection of the mandible to the affected side during mouth opening, associated with clicking. The radiographic finding suggested deterioration of the articular tissues and remodeling of the underlying subchondral bone. On the panoramic radiograph, severe flattening of the left condylar head and a decrease in the articular joint space are evident. Bilateral elongation of the styloid process is present. ICR have always been a diagnostic dilemma. In our case a diagnosis was made based on a good medical history to exclude all other known causes of condylar resorption, radiological and comprehensive head and neck examination, including muscle palpation and TMJ examination to confirm the diagnosis. The patient received nonsurgical treatment that included home-care instructions, including use of hot/cold pads, self-massage of the muscles and the TMJ rea, and practice of relaxation techniques. Instructions were reinforced that the patient should not chew gum and should resume daily vitamin D, calcium, and some physical therapy. The patient was referred to the hospital prosthodontist for correction of the bite. The prescribed treatment plan was designed to optimize pain relief. Treatment of ICR depends on the clinician's beliefs about the disorder's etiology and pathophysiology. Because mechanical overloading of the joint was found to be the main cause of the disease, the use of an intraoral joint stabilization appliance was important in this case. A joint stabilization device was fabricated after the bite was corrected.

Regarding the ethics of the case report, we should add that informed consent was obtained from the patient for publication of this manuscript.

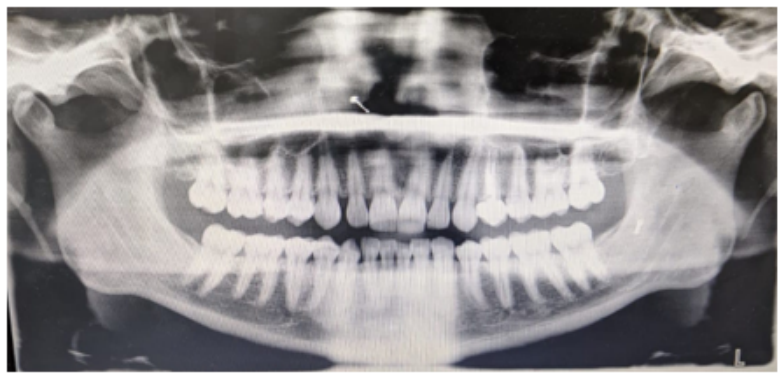

***Figure 1:*** *An OPG taken on Aug 25, 2019, the day of the TMD clinic visit, note the structural bony changes in the left TMJ and the bilateral elongation of the styloid process. An important finding is the presence of a surgical bur embedded within the alveolar bone following the surgical extraction of tooth number 38 in 2009.*

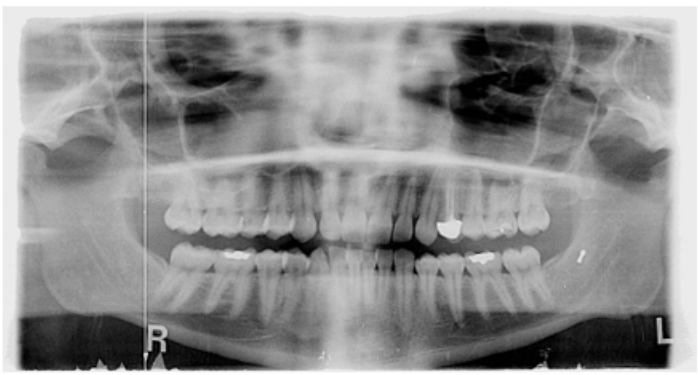


***Figure 2:*** *An OPG taken on Feb 3rd, 2018 on the day of the dental visit but prior to the dental work done on teeth number 45, 46 and 47. Note that the although the radiograph was not diagnostic, it did show early stages of structural bony changes of the disease (Lt condyle).*

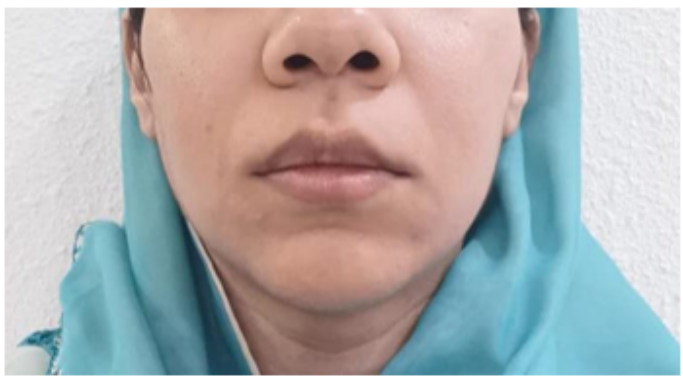

***Figure 3:*** *A frontal view of the patient.*

## Discussion

Many causative factors can contribute to the cycle of events of this pathology and a number of hypotheses have been proposed by authors. The experience with our patient in this paper supports Moore., *et al*. who believed that the most likely causative mechanism behind ICR is the fact that it is a "biomechanical phenomenon, based on increased muscle tension". They hypothesized that pathological compressive forces of the posterior aspect of the condyle on the ligamentous retrodiscal soft tissues constrict the small vessels, limiting circulation to the condyle and resulting in aseptic necrosis. They also believed that chronically dislocated nonreducing disc or malocclusion can be causative factors in this cycle of events. Those skeptical of the "nonreducing disc" risk factor for ICR point out that the almost universally observed bilateral symmetric simultaneous nature of this condition makes it an unlikely etiology [22-26]. Clinical settings of TMJ loading might include compressive orthodontic, surgical, or dental forces, often in the presence of a developmental or acquired malocclusion. In our patient, it was the iatrogenic etiology; during extraction, the surgical bur broke and was embedded within the alveolar bone. Dentists failed to remove the bur and it remained embedded, which we theorized to be the source of the pathological biomechanical overload to the joint and the etiology behind the ICR in our patient's case. Our hypothesis also agrees with Chamberland, who stated that "Functional overloading can facilitate hypoxia and mediate the destructive processes associated with osteoarthrosis as an autocrine factor. Vascular endothelial growth factor (VEGF) induction in osteoarthritic cartilage by functional overloading is linked to activation of the hypoxia-induced transcription factor 1, leading to hypoxia in the joint tissue. Furthermore, VEGF regulates the production of matrix metalloproteinases (MMPs) and tissue inhibitors of these enzymes, which are among the effectors of extracellular matrix remodeling. Overloading also causes collapse of joint lubrication as the result of hyaluronic acid degradation by free radicals. The regulation of hyaluronic acid production is controlled by various proinflammatory cytokines. Of these cytokines, tumor necrosis factors a and interleukin-1 and -6 play crucial roles in the pathogenesis of osteoarthrosis regarding the acceleration and progression of cartilage degradation, because they promote bone resorption through the differentiation and activation of osteoclasts" [27]. Many authors hypothesized that ICR pathology is attributed to a cascade of a series of more than one of those factors, such as loss or disturbance in the physiological condylar remodeling capacity due to age, gender, hormones, systemic diseases, and trauma as mentioned earlier in this paper [3,10,12]. However, the exact cause of this condition remains uncertain; hence the name of the disease [6,10,22].

## Conclusion

ICR is usually an aggressive and rapidly progressing disease, and for that reason an explicit diagnosis, comprehensive examination, and treatment planning are critical. The authors presented many hypotheses behind this disease. The experience with our patient in this paper supports Moore., *et al*. who believed that "pathological compressive forces of the posterior aspect of the condyle on the ligamentous retrodiscal soft tissues constrict the small vessels, limiting circulation to the condyle and resulting in aseptic necrosis". Due to the complicated nature of the disease, the etiology of the patient's condition is difficult to discern. In this case, estrogen-mediated cellular activity, undiagnosed systemic disease, and unstable occlusion are all still possibilities. Several factors can contribute to the disease's development. However, the exact cause of this condition remains uncertain; hence the name of the disease.

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## Conflict of Interest

None.

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