Review Article

Idiopathic condylar resorption: The current understanding in diagnosis and treatment

Andrew Young

Department of Diagnostic Sciences, University of the Pacific, Arthur A. Dugoni School of Dentistry, San Francisco, CA 94103, USA

Abstract

Idiopathic condylar resorption (ICR) is a condition with no known cause, which manifests as progressive malocclusion, esthetic changes, and often pain. Cone-beam computed tomography and magnetic resonance imaging are the most valuable imaging methods for diagnosis and tracking, compared to the less complete and more distorted images provided by panoramic radiographs, and the higher radiation of ^{99m}technetium-methylene diphosphonate. ICR has findings that overlap with osteoarthritis, inflammatory arthritis, physiologic resorption/remodeling, congenital disorders affecting the mandible, requiring thorough image analysis, physical examination, and history-taking. Correct diagnosis and determination of whether the ICR is active or inactive are essential when orthodontic or prosthodontic treatment is anticipated as active ICR can undo those treatments. Several treatments for ICR have been reported with the goals of either halting the progression of ICR or correcting the deformities that it caused. These treatments have varying degrees of success and adverse effects, but the rarity of the condition prevents any evidence-based recommendations.

Key Words: Idiopathic condylar resorption, progressive condylar resorption, temporomandibular disorder

Address for correspondence: Dr. Andrew Young, Department of Diagnostic Sciences, University of the Pacific, Arthur A. Dugoni School of Dentistry, 155 5th Street, San Francisco, CA 94103, USA.
E-mail: ayoung@pacific.edu

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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,[1,2] condylar atrophy,[2] aggressive condylar resorption,[1] acquired condylar hypoplasia,[3] progressive condylar resorption,[1,2] and cheerleader syndrome.[4] ICR is a diagnosis of exclusion given only when all other possible conditions have been ruled out. A significant portion of the reported ICRs followed orthognathic surgery, but ICR can also occur without a history of prior surgery.

Although the exact cause is unknown, a number of possibilities have been proposed, such as hormonal changes,[5,6] and increased mechanical loading from

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parafunctional habits, trauma, internal joint derangement, orthodontics, orthognathic surgery, and occlusal therapy.[7] Preexisting clinically detectable TMJ sounds have also been implicated but have recently been found not to be a risk factor.[8]

CLINICAL PRESENTATION

As the TMJ condyle collapses in ICR, which is usually bilateral[9] but can be unilateral, the mandible begins to the fulcrum on the portion of the occlusal arch that is closest to the collapsing condyle when the mandible is in the closed position. In nonpathological TMJs, this new fulcrum would never exist. The progressively heavier loading on those posteriormost teeth causes them to wear more rapidly than other teeth in the arch. If the condyle collapses faster than the fulcrum teeth wear, the teeth mesial to the fulcrum will become nonoccluding. Theoretically, anterior disclusion would progress more slowly when the ICR is less rapid, when the fulcrum teeth are less hard (due to the crystalline structure of the teeth, acid exposure, etc.,), and when a grinding habit is present. However, studies are needed to confirm or reject this reasoning.

When ICR is bilateral, a bilateral anterior open bite may begin to develop. The degree of occlusal wear on the nonoccluding teeth gives some clue to the approximate age of onset of a patient's ICR. The presence of mamelons suggests ICR started before or shortly after the eruption of the permanent incisors. However, also explanatory would be preexisting Class II occlusion, thumb-sucking/pacifier use habit, or tongue thrust. Conversely, the presence of wear on the anterior teeth, which are now out of occlusion, indicates the patient at one time did not have an anterior open bite. The presence of anterior wear, but minimal to no premolar wear, could be the result of ICR starting after the eruption of the anterior teeth, but before or slightly after the eruption of the premolars.

The jaw exhibits decreased vertical ramus height[2] and posterior facial height,[8,10,11] and the mandibular plane angle is correspondingly high/steep,[2,6] with a dental and skeletal Class II,[2,6,8,12] and anterior open bite.[2,6,8,12] The overjet is increased[9] and the lower incisors are angulated.[2] The backward rotation of the mandible may result in decreased oropharyngeal airway space.[2]

When unilateral, there is a decreased posterior facial height[10] and dental and skeletal Class II,[2] on the side of the ICR. This causes a vertical height difference at the mandibular inferior border, ramus and occlusal plane,[2] and midline shift.[7] Occlusion and wear on the posterior teeth are heavier ipsilateral to the ICR and heaviest on the posteriormost teeth. The ipsilateral posterior teeth may also be in crossbite. The contralateral posterior teeth may become nonoccluding.[7]

Approximately 75% of the ICR patients[7] have symptoms, such as bilateral masticatory muscle, temporomandibular joint dysfunction (TMD) pain or discomfort, and fatigue.[9] The pain intensity is usually roughly 3–4 out of 10.[2,7] In the author's experience, these patients also take longer than the average TMD patient during conservative TMD treatment to achieve a significant reduction in pain. Pain reduction is sometimes ultimately not adequate.

RADIOGRAPHIC PRESENTATION

On radiographic or magnetic resonance imaging (MRI) examination, the volume of the condyle is diminished.[2,6,13-17] The resorption occurs on the superior,[14] anterosuperior,[10,11,14] or all other surfaces[14] of the condylar head. This often results in an alteration of condylar contour,[13,16,18] with flat[6] or peaked[14] condylar surfaces. In some cases the condylar surface, though diminished in volume and height, still retains its rounded shape.[14] When severe, only a remnant of the medial pole may remain.[12] Some cases also present with a loss of integrity of condylar cortex,[2] in the form of erosions.[6,18] Bilateral anterior disc displacement with and without reduction[2] are also seen; although, these are very common in the general population as well.

The loss of condylar height, and resultant formation of a new fulcrum on the posteriormost teeth, causes an increased MPA[8,9,11,18] and a small SNB angle.[8,9,11,17,18] Hoppenreijs *et al.* observed that resorption of the superior surface of condyle tends to present with a deep bite[14] and the resorption of anterosuperior surface tends to present with anterior open bite.[14]

Wolford and Cardenas described the presence of hyperplastic synovium on the superior portion of the condylar head during open-joint surgeries of patients with a pathologic complete response (PCR). This can also be seen either as increased joint space on a tomogram[2] or as thick amorphous soft tissue on MRI.[2] No other authors have mentioned this tissue, despite surgically accessing the TMJ condyle. As the investigation into ICR continues and the scientific understanding increases, it is possible that this may become a subset of the ICRs, pathognomonic, or a condition of its own. It has the potential to be a valuable diagnostic marker.

NATURAL HISTORY

ICR progresses at a rate of approximately 1.5 mm/year.[2] The point at which it stops varies case-by-case and there are currently no ways to predict this point. However, it has been hypothesized that it does not progress beyond the condylar neck.[9]

EPIDEMIOLOGY

The ICR is rare. Sansare *et al.* in 2015 counted a total of 178 reported cases, all originating from only seven countries: The USA, The Netherlands, South Korea, Switzerland, France, Germany, and Japan. The nation of origin of these reports is likely reflective of awareness of the condition, though racial predilection cannot yet be ruled out. As noted by Sansare *et al.*, half of those cases were from only two groups: an oral and maxillofacial surgery group in The Netherlands and one in the USA.[1]

Furthermore, 11 of the 17 publications reviewed by Sansare *et al.* were investigating postsurgical cases, though this could be reflective of reporting bias.[1] In a review[14] of reports published between 1990 and 1998, the incidence of ICR following orthognathic surgery ranged from 5.8% to 20%.[14,19-24] In a more recent retrospective study of 15 patients that underwent condylectomies and costochondral grafts for ICR, 4 of them (26.6%) had prior orthognathic surgery.[9] In a larger study by Hwang *et al.*, of 452 consecutive patients who underwent orthognathic surgery, 16% of those who had combined maxillary osteotomies and bilateral sagittal split osteotomy (BSSO) developed ICR, but only 1% of those who underwent isolated BSSOs developed ICR.[8]

The authors have suggested ICR to be more common in females (9:1 female predominance compared to male), ages 10–40 (predominantly those in their teens to twenties), and those with Class II skeletal and occlusal relationships.[2,7-9] Mehra *et al.* reported that 71% of their 21 ICR patients correlated the initiation of ICR with active orthodontics, though the patient report is less reliable than imaging; often a significant amount of this slowly progressive condylar resorption should occur before the patient notices a clinical change.[7] Hwang *et al.* consider posteriorly inclined TMJ condylar necks to be risk factors.[8]

DIAGNOSIS AND TRACKING

Diagnosis of ICR is primarily made through imaging, though the trigger to order imaging is often clinical or reported findings. Imaging would aid in differentiating a thumb-sucking habit, tongue thrust habit, osteoarthritis (OA), inflammatory arthritis, physiologic condylar resorption, and ICR. Particularly between the latter four, there is overlap and variability in their signs on imaging. Wolford and Cardenas[2] described synovial hyperplasia of the condylar head, which they attributed to ICR, visible on the MRI and implied on radiographs. As stated above, if this finding indeed a feature of ICR, then it would greatly simplify the diagnosing of ICR. MRI would then also be the most conclusive imaging method for diagnosing ICR. However, no other authors have reported that hyperplasia and corresponding widened joint space. Interestingly, in juvenile idiopathic arthritis (JIA), a pannus, which is granulation tissue, forms on the cartilage and degrades the cartilage and adjacent bone.[25]

^{99m}Technetium-methylene diphosphonate (^{99m}TC-MDP) is a radionucleotide that is administered by injection and subsequently imaged in the TMJ, for the purpose of determining whether the ICR is active.[9] This information is important because corrective orthodontics, prosthodontics, and/or orthognathic procedures and some surgeries should not begin while ICR is active. However, a significant disadvantage with ^{99m}TC-MDP is the amount of radiation involved, which is approximately 4–6 mSv.[1] When considered, the patient's age (which is often relatively young) should also be factored into the decision. ICR can also be determined as active or inactive through serial imaging, which with cone-beam computed tomography (CBCTs) would be a fraction of the radiation of ^{99m}TC-MDP, and with MRIs would have no radiation.

CBCTs, like MRI, allow assessment of all surfaces of the condyle, so all locations of resorption are identified and quantified.[26] CBCT also allows measuring of condylar head volume, which is a potentially efficient and comprehensive method for recognizing and quantifying condylar resorption. Moreover, if percent volume loss is in the future is used to differentiate pathologic from physiologic condylar resorption, CBCT will be very valuable in that diagnostic process.[27]

CBCTs and MRIs can be used to assess whether the ICR is active or arrested. However, to assess this, they are taken in series and compared for changes in condylar dimensions. A loss of volume means ICR was active between those two capture dates, but it does not indicate whether ICR continued to be active even at the time of the second image capture, or if it stopped sometime between those two image capture dates. If no volume was lost between the image capture dates, then one can be quite certain that the ICR is no longer active. Deciding how far apart to take the serial images can be challenging. Six months is often sufficient

time to detect a true change in the condylar dimensions, but a year would be more ideal. Yet, the patients often prefer not to wait that long and sometimes the orthodontic or prosthodontic factors also favor less delay.

Panoramic radiographs have limited value in diagnosing and monitoring ICR. They can only show gross changes on the anterior, superior, and posterior surfaces of the condyle. The medial and lateral resorption would generally occur undetected. Distortion, and obscuring from overlapping structures, also prevents precise measurements of condylar loss between serial images.[26]

While clinical measurements may be used to monitor the progression of ICR between serial images, they are not adequate alone. Occlusal wear on the fulcrum teeth will compensate for the resorption of the condyles, lessening the amount of resultant overbite. The patient then loses a significant amount of condylar height but display no change in an overbite because of compensatory posterior occlusal wear. Therefore, imaging is needed to measure actual condylar volume loss. ICR is by definition and origin resorption of the condyle, so its monitoring should be done at the condyle.

COMPARISON TO OSTEOARTHRITIS

While ICR is reported by Wolford and Cardenas (Wolford *et al.* 1999) to exhibit increased joint space superiorly on radiographs because of the cap of synovial hyperplasia, OA does not usually present with this finding. Comorbid anterior disc displacement, or osteophytes on the anterior surface, would result in increased anterior joint space in OA of the TMJ.[26]

OA often presents with sclerosis and generalized sclerosis of the condylar head (as opposed to focal sclerosis) meets the diagnostic criteria for OA.[26] Sclerosis has not been reported in ICR.

Both ICR and OA exhibit a loss of volume in the condylar head. However, the loss of volume in OA occurs in the form of condylar head flattening, either from the superior surface or the anterosuperior surface. These wear patterns seem to reflect the surfaces that experience wears during excessive joint loading. In ICR, flattening can likewise occur not only on the anterosuperior aspect[10,11] or superior aspect but also can occur in all three sides (anterior, superior, and posterior) simultaneously.[14] The presence of loss of volume from the posterior aspect, particularly because it is not an area subjected to physical wear, should make the clinician suspicious of a non-OA condylar resorption.

The presence of cortical erosions, seen radiographically as breaks in the cortical outline, are diagnostic for OA.[26] Subcortical cysts, which are in fact not actually cysts, but focal areas of osseous erosion, are also diagnostic for OA.[26] They can also occur in ICR but seem to be less common.

The presence of osteophytes, which are bony outgrowths that occur in areas of cartilage degeneration,[28] also are diagnostic for OA,[26] but have not been reported for ICR. Calcified loose tissues in the soft tissues around the TMJ condyles are not alone diagnostic for OA, but do occur;[26] they have not been reported in ICR.[2,6,18]

Joint effusion occurs in OA, though alone it is not diagnostic for OA.[26] It has not been reported in ICR.

COMPARISON TO INFLAMMATORY ARTHRITIS

Since ICR tends to occur in the teens and twenties, the most relevant inflammatory arthritis is JIA, which occurs from 0 to 16 years of age.[29] It affects the growth of the mandible, which normally can continue until the third decade[30] and cause condylar hypoplasia and anterior open bites.[31] Thus, it is confused with ICR. JIA occurs in 1 out of 1000 children in Europe aged 0–15.[25] The TMJ is involved 38%–72% of the time[32] and seems to be more common in cases of earlier JIA onset.[33] Most of the patients with TMJ involvement do not report TMJ signs or symptoms.[34-36] Left undetected, the facial functional, and esthetic consequences can be significant;[37] these alterations may become more evident during the growth spurts of ages 9–12 years.[38] Depending on the subtype, systemic findings, and laboratory tests will help distinguish them from ICR.

COMPARISON TO PHYSIOLOGIC CONDYLAR RESORPTION (OR CONDYLAR REMODELING)

The TMJ condyle is resorb, particularly in postsurgical cases, in a manner that might be considered nonpathologic. It has been considered an adaptive osseous response to new functional and passive loads placed on the TMJ following the surgically altered changes.[8] This may also apply to significant occlusal changes with orthodontics and prosthodontics. Xi *et al.*, in a study of 112 patients that underwent BSSO, reported 55% of the patients experiencing postoperative condylar volume loss, with a mean reduction of 6%. The difference between pathologic and nonpathologic condylar resorption may be the degree to which resorption has occurred. Xi *et al.* observed a 17% loss of the condylar volume to be the threshold at which an anterior open bite occurred. When an anterior

open bite is present, there is usually a significant amount of anterior facial height increase, horizontal mandibular body length decrease, and posterior facial height decrease. They, therefore, suggested considering a 17% loss of the condylar volume as the lower threshold for diagnosing PCR, but stated a larger sample size is needed to be more conclusive.[27]

COMPARISON TO CONGENITAL DISORDERS AFFECTING MANDIBLE

A number of congenital conditions exist which can cause condylar hypoplasia. These include hemifacial microsomia, Goldenhar syndrome, Treacher Collins syndrome, acrofacial dysostosis, and Silver–Russell syndrome. These usually can be differentiated from ICR by the presence of other oral or facial anomalies, and anomalies elsewhere in the body.

EFFECT ON ORTHODONTIC TREATMENT

Since this condition usually occurs around the age of puberty, there is an increased likelihood of ICR occurring during orthodontic treatment. This can result in several complications.

Idiopathic condylar resorption begins before orthodontic treatment

If a patient presents with an anterior open bite, the orthodontist needs to determine the cause of the open bite. Besides ICR, several conditions could be causative. Thumb-sucking or pacifier use which was found to continue past the age of 7 in 12.1% of one study's population[39] usually has opposing occlusal planes that curve away from each other; ICR has the usual less-pronounced curve of Spee. Tongue thrust causes similar changes to the occlusal plane curves.[40,41] If the cause of the anterior open bite is ICR, then the orthodontist should determine whether the ICR is active or inactive. If inactive, orthodontics may begin provided there are no other contraindications to beginning orthodontics. If it is still active and continues to be active after orthodontics is completed, the anterior open bite will return shortly after the completion of orthodontics. Therefore, orthodontics should not begin until ICR has become inactive.

A thorough discussion should also be conducted with the patient regarding expectations. If the patient wants orthodontic treatment for esthetic or functional reasons, orthodontics can be reasonably expected to deliver on those expectations, though surgery may also have to be included. However, if the patient hopes to experience a permanent relief of TMD symptoms by restoring balanced occlusion, orthodontics, often in conjunction with surgery, may not be able to deliver on that expectation. Mehra *et al.* did not note a significant lasting decrease in overall pain after occlusion was restored with alloplastic TMJ reconstruction and orthodontics (Mehra *et al.* 2016). Wolford and Cardens reported a significant decrease in pain after removal of the hyperplastic synovium, disc repositioning and ligament repair, and orthognatic surgery, from an average presurgical pain intensity of 3.5 out of 10 (range 0–9), to a postsurgical average of 0.7 (range 0–4), though the *P* value was not given (Wolford *et al.* 1999). Any given patient's TMD can have multiple causes and the lack of even occlusion may only be one. Chronic pain also becomes a disease itself, self-perpetuating by the central sensitization, maladaptive behavior, and maladaptive thinking, which can also result in unsatisfactory symptom relief after orthodontics.

Idiopathic condylar resorption begins during treatment

If a patient develops ICR during orthodontic treatment, it may remain undetected until an anterior open bite forms after orthodontics is complete. The only sign may be some pain and a longer than expected orthodontic treatment time. While the rarity of spontaneous ICR means orthodontists should not routinely suspect it mid-treatment, it should be monitored for during any case that involved orthognathic surgery. This may involve imaging mid-treatment.

EFFECT ON PROSTHODONTIC TREATMENT

Prosthodontic treatment in the presence of suspected ICR involves complications similar to that with orthodontic treatment, but with some differences. The total treatment time for prosthodontics is generally much shorter than orthodontics, making it less likely that spontaneous ICR would develop during prosthodontic treatment. Furthermore, most spontaneous ICRs occur in the teens and twenties, which is younger than most patients undergoing extensive prosthodontic treatment. Therefore, most spontaneous ICRs will affect prosthodontic treatment as a preexisting, but possibly still ongoing condition. The prosthodontist or dentist mainly should confirm that the ICR has stopped and then may proceed with treatment. If, per the patient's history, the ICR seems to have started and ended many years prior, evidenced by no further opening of the overbite and overjet, and no unusually rapid and localized occlusal wear, then no further investigation may be needed. If the onset of ICR was more recent, and the prosthodontist or dentist cannot be certain that it has stopped, serial CBCT or MRI may be indicated to confirm the arrest of ICR before proceeding.

If prosthodontic treatment is following orthognathic surgery, the risk for ICR to develop during or after prosthodontic treatment is higher.

In general, removable prosthodontics cases would experience milder consequences if ICR occurs after treatment is complete. The teeth on removable prostheses allow easier and more extensive occlusal adjustments. Moreover in the event that a new prosthesis needs to be made because of on-going ICR, it is less damaging to the natural dentition than would be a new set of fixed prostheses.

MANAGEMENT

Treatment for ICR is generally done for one of two reasons. If done during active ICR, it is usually for the purpose of halting the progression of ICR. If done after ICR has stopped, it is usually to restore occlusion and esthetics.

Treatment options range from no treatment to condylar replacement and osteotomy. Being a rare and idiopathic condition, there are insufficient data in the literature to make evidence-based recommendations for treatment.

No treatment

When ICR is bilateral, the patients often elect this option; Hoppenreijs *et al.* reported roughly half of their patients doing so.[14] Many have had occlusion only on their second molars for an extended time before finding a provider who diagnoses the ICR and present the treatment options, and by then have already become comfortable and accepting of the occlusal scheme. They, therefore, often feel surgery and fixed prosthodontics to be excessive. Moreover, with most of them being the past age where they and their peers would commonly have braces, orthodontics also is to them not an attractive option.

For these patients, the risks of no treatment should be discussed. This includes excessive wear of the posteriormost teeth and possible resultant pulp exposure. Traumatic occlusion can also potentially cause periodontal bone loss and mobility, fracture, and pulpal death. The risk of these happening can be reduced by training the patient to avoid daytime bruxism and using a full-coverage occlusal splint at night.

Treatments performed to arrest idiopathic condylar resorption

Splint

As with all treatments discussed in this review, splint therapy has limited evidence to support it. It has been proposed that splints may alleviate TMJ loading, and TMJ loading may accelerate ICR. Yet, whether this is in fact the true or not, splints will protect the teeth.

The patients should understand in advance that the night guard will be abnormally thick in all, but the posteriormost section to fill the area of open bite and bring all teeth into occlusion. Hard acrylic should be used because a soft vacuform material cannot be made thick enough for the area of natural open bite. Patients may actually have some difficulty adapting to full occlusion when they have not had it for a long time. The unusual thickness may also be a factor is slower patient acceptance.

Removal of hyperplastic synovium

Wolford and Cardenas describe 14 cases that underwent this open-joint surgery, in which the condyles were repositioned, the discs stabilized by attachment to implants, and BSSO and maxillary osteotomies were performed. All reported cases treated in this way in the literature were done by one group, with no relapse after an average follow-up of 33.2 months (range 18–68).[2] No adverse outcomes were reported.

Condylectomy and costochondral graft

In a study by Troulis *et al.*, fifteen patients with active ICR underwent a minimum of endoscopic condylectomy and costochondral graft. When indicated, Le Forte I maxillary osteotomy and/or genioplasty was done. Orthodontics was done before the surgery. With a mean follow-up of 34 months (range 12–84), no TMD, neuropathy, or any other adverse outcomes were reported. Furthermore, no significant relapse was observed.[9]

Treatments performed after idiopathic condylar resorption has stopped

Bilateral sagittal-split osteotomy

This procedure involves splitting the mandible with bilateral sagittal cuts and re-orienting the mandibular body to achieve full occlusion. Adverse outcomes include mild-moderate pain, and mild hypoesthesia, still persistent at the 1-year follow-up.[42] Relapse also occurs. Hoppenreijs *et al.*[14] published a retrospective study of patients who developed ICR after a minimum of BSSO. For those who had a corrective second BSSO surgery (minimum), 46% had a relapse during the 8–120 month follow-up period. Moreover, of those with relapse, the average amount of relapse was 67%. However, we do not know if the original BSSO surgeries were done for ICR. The reported relapse rate of ICR for BSSO ranges from 46% to 100% for 6–68 months.[10,14,43]

Orthodontic treatment

If the open bite is not too large, full occlusion may be restored with orthodontics.

Prosthodontic treatment

This has never been reported in the literature as the sole treatment for restoring full occlusion. This is likely because

even though it would be more rapid than orthodontics, it requires extensive removal of tooth structure.

Splint

A splint, while advised to begin during active ICR, should continue to be used for the lifetime of the patient. This is discussed in more detail above.

Unspecified as before or after

Alloplastic reconstruction

Mehra *et al.* published a retrospective study of patients 21 patients who underwent TMJ reconstruction with synthetic material. Relapse rates were low. Comparing the 2-week postop period to the furthest follow-up time point (average 6.2 years, range 5–12 years) mandibular point *P* receded 1%, pogonion 0.3%, gonial length 4.1%, and occlusal plane by 3.9°. They reported no adverse outcomes.

CONCLUSION

To better understand this idiopathic condition, the clinical and research community need more data. This needs to start with greater awareness, and then reporting; the small number of centers and regions reporting the majority of the cases indicates most ICR cases are not being used to increase our understanding of the condition. Moreover, because there is some heterogeneity in the presentation of cases reported thus far, we may find that the currently classified ICRs may, in fact, be more than one diagnostic entity.

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