Anterior open bite due to idiopathic condylar resorption during orthodontic retention of a Class II Division 1 malocclusion

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A common dilemma when treating anterior open bite is understanding its etiology. Idiopathic condylar resorption (ICR) can cause open bite in affected individuals. Although it is prudent to not treat patients with ICR until active resorption has ceased, orthodontists may begin treating them because anterior open bite from ICR may not appear before or during their orthodontic treatment. This article reports a 12-year-old female who was diagnosed with ICR 10 months after completion of her orthodontic treatment for a Class II Division 1 malocclusion. When a young patient with a high mandibular angle and previous skeletal or dental Class II malocclusion returns with an open bite during the retention phase, the patient's condyles must be carefully examined to determine whether any temporomandibular joint disorder, such as ICR, is present. Currently, the controversy over the cause and the cure for ICR is continuing to challenge orthodontists in diagnoses and treatments. Orthodontists should closely monitor and offer informed treatment options to patients with risk factors for ICR or signs of its pathology that might develop at any stage of orthodontic treatment, including the retention period. (Am J Orthod Dentofacial Orthop 2019;156:555-65)

Treating open bite has long been considered a profound orthodontic challenge because of the complexity of the treatment and difficulty in achieving stability. Among the causes of anterior open bite, idiopathic condylar resorption (ICR) compounds the challenges to orthodontists because of its negative effect on growth and potential for relapse after surgical and orthodontic treatment.[1-5] Orthodontists encounter patients with ICR at a rate of 1 case per 5000 orthodontic patients,[5] but this low number is possibly due to a poor understanding of the pathology among orthodontists; ICR has only recently begun to be recognized as a significant etiologic factor for anterior open bite.[6]

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ICR, also known as idiopathic condylysis, condylar atrophy, and progressive condylar resorption, results in malocclusion, facial change, musculoskeletal instability, and temporomandibular joint (TMJ) dysfunction.[1,7-9] Early diagnosis and proper treatment may help minimize the compromised growth of the condyle and the change in morphology of the mandible.[10,11]

Adolescent females, predominantly those with high mandibular plane angle and Class II skeletal or dental relationships, are at the greatest risk of ICR.[7,12] It has been theorized that ICR causes mandibular recession into a Class II position in 2 forms of condylar resorption: (1) adult, in which the mandible recedes after growth completion, and (2) juvenile, in which the potential mandibular growth rate is diminished.[13] This case report presents a female adolescent patient with ICR. The onset of ICR began about 10 months after completion of her orthodontic treatment.

CASE REPORT

A 12-year-old girl presented with the chief complaint of crowding. Clinical examination revealed a large overjet (7.7 mm), normal overbite (2.6 mm),

and moderate and mild crowding in the maxillary and mandibular arches, respectively. She had a narrow palatal arch width and bilateral end-on Class II molar relationship. She had a mesocephalic facial form with a convex profile. In the panoramic radiograph, her condyles showed good cortication without beaking. Lateral cephalometric analysis revealed a mild skeletal Class II pattern (ANB, 4.4°) with a retrognathic mandible (SNB, 73.9°) and hypodivergent growth pattern (SN-MP, 27.7°). She did not report any TMJ dysfunction or breathing or sleep problems. She was in good general health with no history of major systemic disease or dental trauma (Fig 1).

The treatment objectives were to expand her palatal arch, obtain normal overjet and overbite, relieve crowding, attain Class I molar relationship, and improve her soft tissue profile.

Treatment started with a Quad Helix for arch expansion. After leveling and aligning, a fixed Class II corrector, Forsus (3M Unitek, Morovia, Calif) appliance was used for 8 months for correction of the Class II malocclusion. Upon removal of the appliance, Class II elastics were used at night. At the finishing stage, anterior box elastics from maxillary lateral incisors to mandibular lateral incisors were engaged along with the Class II elastics at night. The total treatment time was 23 months.

Posttreatment analysis indicated that treatment objectives had been successfully achieved. Class I canine and molar relationships were established with a canine protected occlusion. Profile esthetics improved, and an acceptable overbite and overjet were attained. The posttreatment panoramic radiograph showed acceptable root parallelism and 3 developing third molars. Some teeth showed mild apical root resorption, and slight condylar resorption was noted in the anterosuperior aspect on both sides, which did not exist at pretreatment. The posttreatment lateral cephalometric analysis showed improvement of skeletal Class II pattern (ANB from 4.4° to 3.1°; Wits appraisal from 4.6 mm to 0.7 mm) (Fig 2; Table).

However, during the course of her treatment, the patient's mandible grew with a downward and backward rotation. The posttreatment mandibular length was longer than pretreatment, increasing from 98.5 mm to 102.1 mm (Table). Despite the condylar resorption seen in the final panoramic radiograph (Fig 2), the patient did not show any symptoms related to ICR during treatment or for the 6 months after treatment. The inclinations of her incisors did not change significantly and her lower lip position to E-plane remained unchanged. However, her upper lip became more retrusive with respect to the E-plane, changing from -2.4 mm to -6.1 mm to the E-plane. This appeared to be from the soft tissue change including growth of her nose (Table).[14]

At the 6-month follow-up, the patient had a stable occlusion, and treatment results were maintained. However, 10 months after completion of the treatment, the patient returned for an emergency visit because her general dentist noticed an anterior open bite and suspected a tongue thrust. The patient reported that a dull, aching pain occurred a few times a day on both sides of her jaw when she ate chewy foods. No other TMJ symptoms were reported at this time, and maximum jaw opening was within the normal range. She did not report any systemic health issue.

Cone-beam computed tomography (CBCT) imaging was acquired during this visit, and the radiological findings revealed that the patient had ICR, more aggressive on the left condyle with some characteristics of degenerative joint disease (DJD)[5,8] (Figs 3, *A* and 4, *C*). Her condylar heads were well positioned within the glenoid fossa, but significant flattening and irregular surfaces were seen at the anterosuperior aspect of the condylar head on both sides with anterior lipping, consistent with DJD and osteophyte formation. The joint spaces measured approximately 2.6 mm at the mid-condylar head position bilaterally. Multiple small areas of hypodense lesions, consistent with subchondral cysts, were seen. Although the overall bone volume of the condylar heads was mostly unchanged in the posterior aspect, there was a prominent change in the anterosuperior aspect compared with the appearance in the pretreatment panoramic radiographic image.

The patient was referred to a rheumatologist for further examination. It was concluded that she did not have an autoimmune disease. She was monitored by a temporomandibular disorder (TMD) and orofacial pain specialist while wearing a soft splint known to reduce the muscle pain effectively.[15] Aside from hypermobility of joints and a popping and clicking sound when she opened her mouth, the patient had crepitus on the left side condyle and pain on both condyles when chewing. Based mostly on these clinical symptoms, TMJ osteoarthritis (OA) was suspected as a possible cause for these issues.[16]

A CBCT was acquired 15 months after treatment, and the radiological findings were still suggestive of both DJD and ICR. The superior surface of her condyles remained irregular and flat with small subchondral cysts as noted in the earlier examination. Whereas the condylar volume was still unchanged, the overall bone density had increased, and her condylar heads appeared more sclerotic and hyperdense than they did previously (Fig 3, *B*). Her overjet was now 6.5 mm with an increased anterior open bite of 4 mm (Fig 5, *C*). A lateral

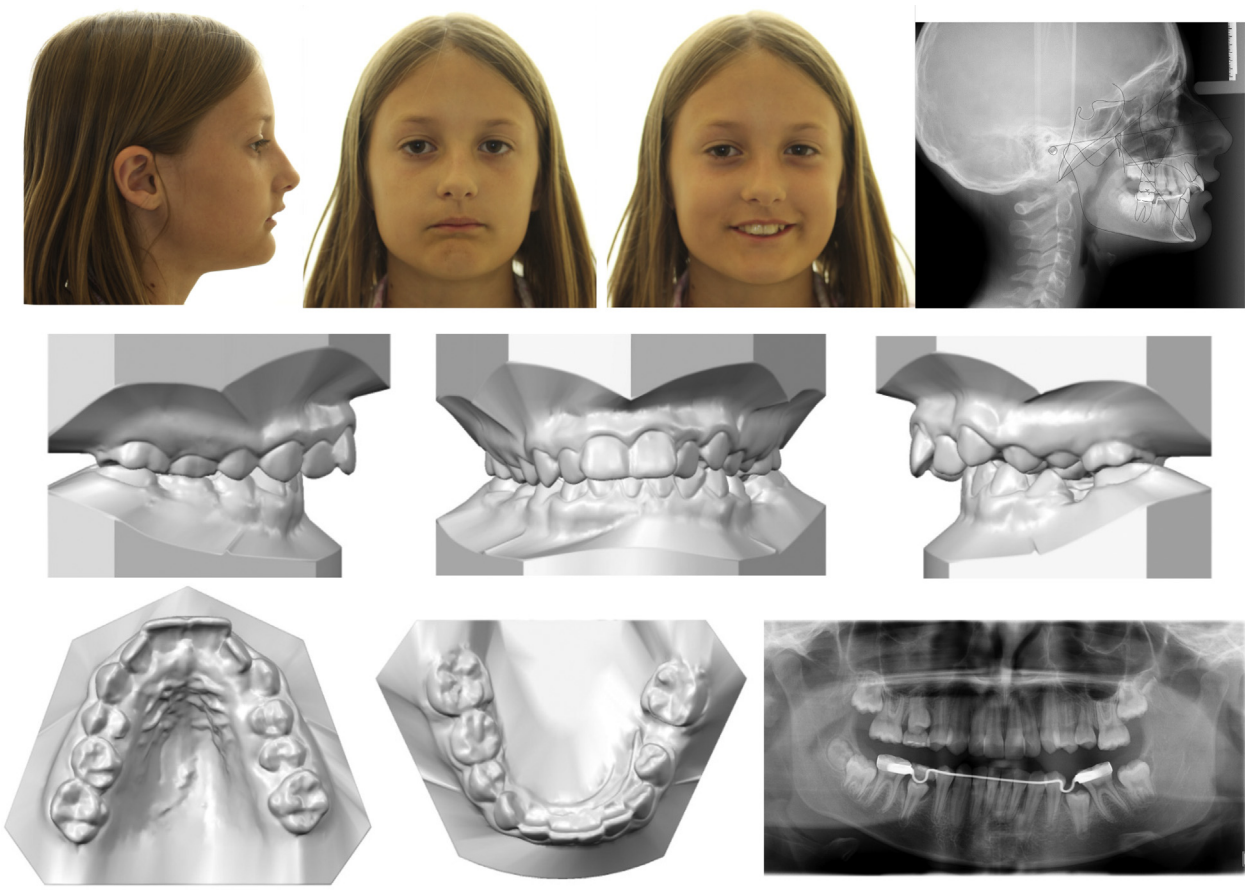

Fig 1. Pretreatment photographs and radiographs.

cephalogram derived from the CBCT suggested that the mandible had decreased in length during retention. The mandibular length, which was 102.1 mm at the debonding visit (Fig 4, *B*), was now 97.9 mm (Figs 4, *D* and 6; Table).

At 16 months after treatment, as the soft splint had worn down, hard splints were delivered for better durability. A flat plane stabilization appliance for the maxillary arch was delivered to be worn indefinitely at night. This device was intended to relax the elevator muscles, protect the teeth, provide joint stabilization, and redistribute the occlusal forces.[17] Then, a mandibular orthopedic repositioning appliance in the form of a mandibular splint was delivered to produce vertical dimension and horizontal maxillomandibular relationship changes.[18] The splint was for daytime use only, designed in a way as to not cover the anterior teeth to facilitate daytime speaking. The patient was instructed to use it for 4-6 weeks to prevent an adverse effect on the occlusion. It was expected that her TMJ would heal during this time.[19]

At 23 months after treatment, the severity of the patient's open bite remained the same (Fig 5, *D*). She still had bilateral jaw pain during mastication. Her headaches, which had worsened over time, were controlled by ibuprofen. She is currently being monitored for burn-out of the condylar resorption. Meanwhile, she has been instructed to perform manual therapy and clenching exercises to reduce pain, relax and reset the masticatory muscle, and possibly autorotate her mandible.[20,21] The resorptive process of the condyle will be monitored by comparing the shape and size of the condyles on the radiographs and measuring the status of open bite using the splint as a landmark. When it has been clinically and radiographically confirmed that active resorption has ceased,[1,13] the patient will be evaluated for further treatment, including conservative management, orthodontic camouflage treatment, and orthognathic surgery as indicated.

DISCUSSION

To diagnose ICR, clinical detection can be first achieved by evaluating changes in occlusion, skeletal

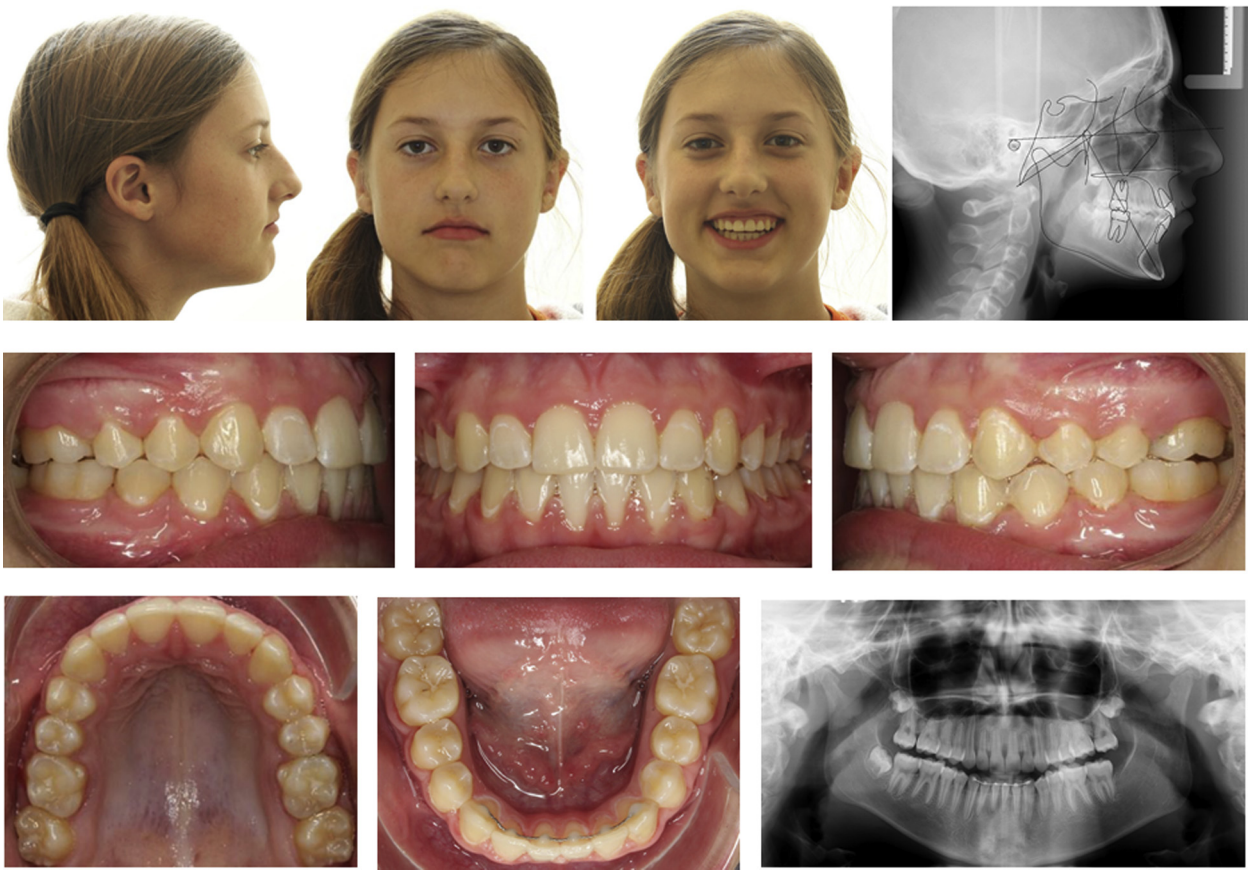

Fig 2. Posttreatment photographs and radiographs.

Table. Cephalometric measurements

Measurement	*Norm*	*Pretreatment*	*Posttreatment*	*10 months after treatment*	*15 months after treatment*
SNA (°)	82.0	78.4	76.0	75.7	75.6
SNB (°)	80.0	73.9	73.0	70.8	70.3
ANB (°)	2.0	4.4	3.1	4.9	5.3
Wits (mm)	0.0	4.6	0.7	0.5	1.1
Mandibular length (Co-Gn) (mm)	106.7	98.5	102.1	100.6	97.9
SN-MP (°)	32.0	27.7	35.0	37.5	37.1
FH-MP (°)	25.0	17.6	23.6	27.5	27.7
LFH (ANS-Me/N-Me) (%)	55.0	54.5	56.8	56.9	57.0
U1–SN (°)	104.0	105.1	103.3	102.2	98.7
U1–NA (°)	22.0	26.7	27.3	26.6	23.1
IMPA (°)	90.0	95.6	96.9	101.9	99.8
L1-NB (°)	25.0	15.5	23.4	30.2	27.2
U1/L1 (°)	131.0	133.4	126.3	118.4	124.4
Upper lip (mm)	−4.0	−2.4	−6.1	−4.9	−5.4
Lower lip (mm)	−2.0	−3.5	−3.3	−1.4	−2.4

relationship, and soft tissue change, especially when there is no anticipated growth or TMJ dysfunction.[5,22-24] Sagittal and posterior vertical deficiency of the lower facial third can result in decreased ramus height and mandibular length with posterior mandibular rotational growth.[9] Increased occlusal and mandibular plane angles, increased overjet, and proclined mandibular incisors are some of the characteristics frequently found in individuals with ICR.[7,25]

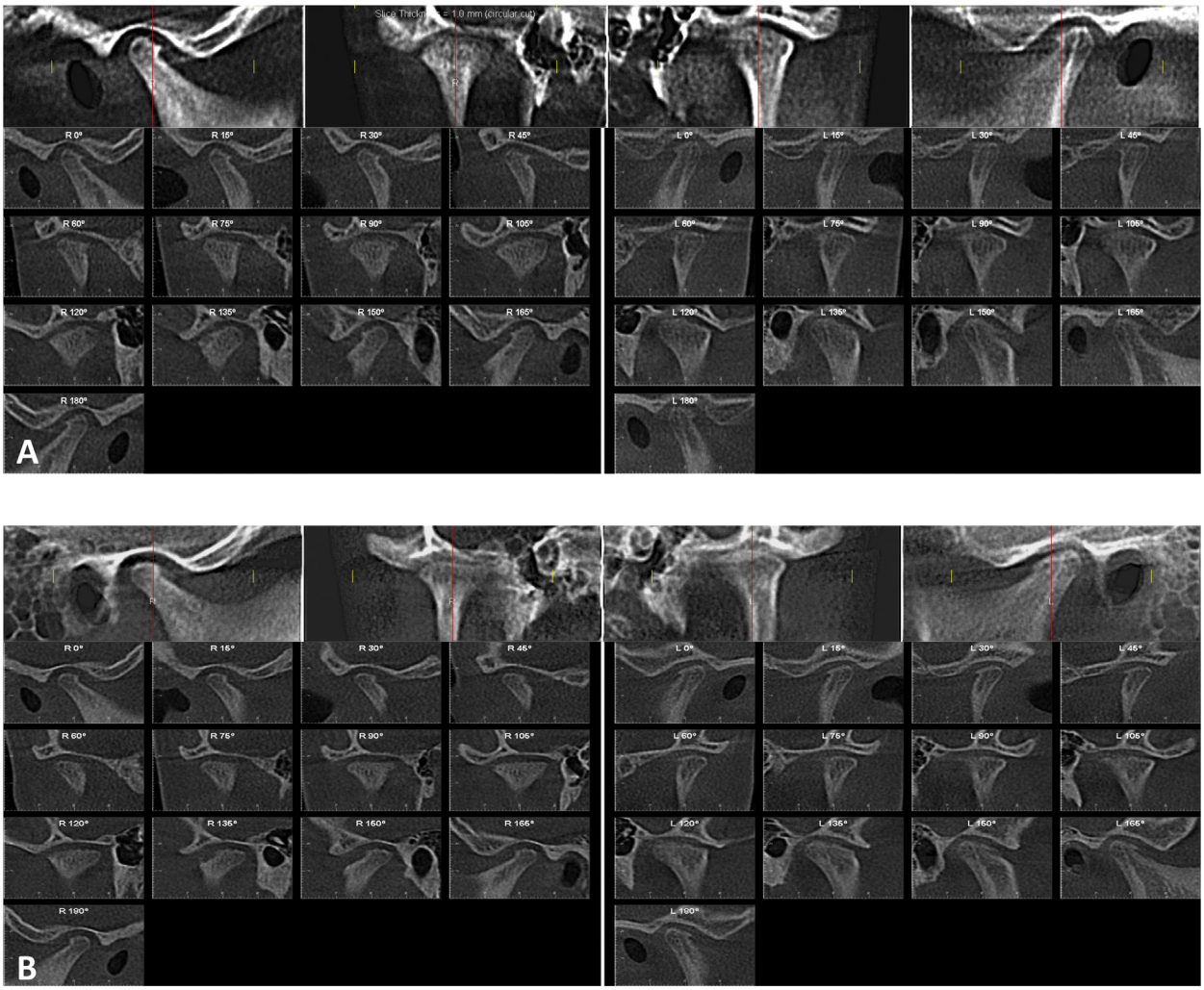

Fig 3. Radiographic imaging (CBCT) of the patient's condylar resorption. **A,** 10 months posttreatment; **B,** 15 months posttreatment.

The patient in this case report did not have any of these clinical symptoms except for a large overjet before treatment. However, the aforementioned characteristics gradually developed after completion of her treatment, as her anterior open bite worsened. More often, bilateral ICR results in a relatively symmetrical posterior shift of the mandible. By contrast, in a unilateral ICR or a bilateral ICR with differing rates of condylar resorption on the right and left sides, the mandibular dental midline and the chin shift to the affected side.[1] In this case, the mandibular dental midline and the chin gradually shifted to the left, and there was more aggressive resorption on the left condyle (Figs 5, *C* and *D*). Unilateral Class II occlusal relationship, crossbite, and posterior occlusal prematurity can also be caused by asymmetrical ICR.[1] The process of resorption in many affected individuals resolves spontaneously in 6 months to 2 years.[12]

TMJ distress is another clinical feature to be considered among patients with ICR.[5] TMJ symptoms range from nonexistent to severe.[26] Wolford and Cardenas[7] reported that TMJ symptoms were mild or nonexistent among patients with ICR. The absence of clicking and popping was explained by hyperplastic synovial tissue largely taking up the joint space as the synovial tissue gives smooth transition. Reyneke and Ferretti[12] also stated that ICR progresses without pain or hypomobility. However, in a more recent study, Kristensen et al[26] concluded that most patients with ICR showed symptoms and signs of TMD, and only 12% of the patients presented without any sign or symptom of TMD in their series. Patients had the following symptoms: arthralgia,

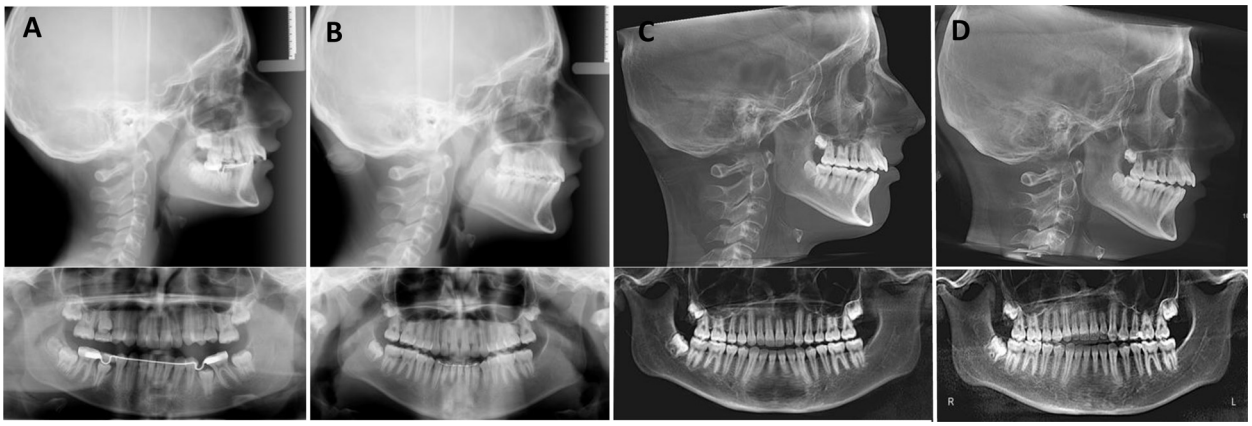

Fig 4. Lateral cephalograms and panoramic radiographs showing the progress of ICR with aggravated open bite, condylar shape change, and shortening of the mandibular length. **A,** pretreatment; **B,** posttreatment; **C,** 10 months posttreatment; **D,** 15 months posttreatment.

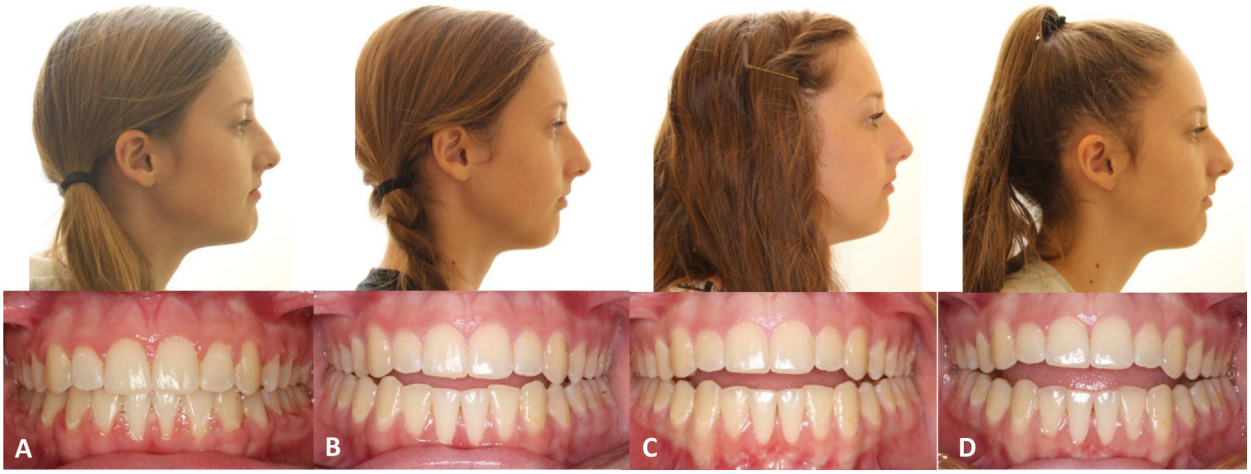

Fig 5. Progress of anterior open bite over time shown in intraoral and profile photos. **A,** posttreatment (14.1 years old); **B,** 10 months posttreatment (14.11 years old); **C,** 15 months posttreatment (15.4 years old); **D,** 23 months posttreatment (16.0 years old).

myalgia, TMJ sound, TMJ locking, and decreased mouth opening.

According to Hatcher,[8] ICR proceeds through a destructive phase of loss of the cortex that begins along the anterosuperior surface of the condyle. A cavitation defect extends into subchondral bone, resulting in loss of condylar volume. This active phase is followed by the reparative phase of condylar flattening and recortication (Fig 7). Radiographic imaging is useful in diagnosing and detecting the phase of condylar degeneration in ICR.

Two-dimensional imaging shows structural changes on the condylar surface, decreased SNB angle, high occlusal and mandibular plane angles, and decreased vertical height of the ramus.[1,9] Panoramic radiographic findings include thinning and shortening of the condyles and an average condylar resorption rate of 0.12 mm per month.[5,7] Magnetic resonance imaging displays the loss of integrity on the cortical bone in the condylar head and illustrates thick soft tissue between the condyle and fossa. Deformation and degenerative changes of the discs can also be evaluated.[1,9] Recently, a CBCT study showed a statistically significant reduction in the condylar width, height, and axial angle, and 84% of the TMJs had a posterior condylar neck angle. Whereas the condylar width, height, and axial angle decreased, the length did not change. Noncongruent shape of the condyle-fossa relationship

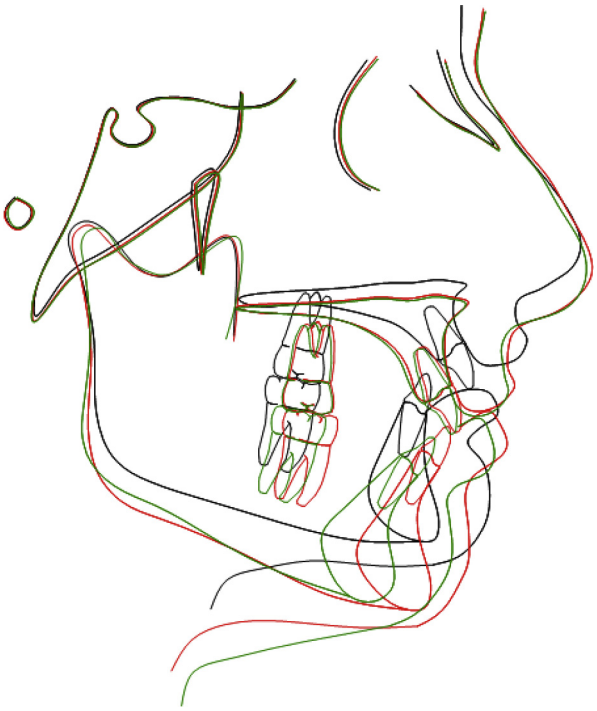

Fig 6. Superimposition of cephalometric tracings from pretreatment, posttreatment, and 15 months after treatment. Pretreatment (*black*); Posttreatment (*red*); 15 months after treatment (*green*).

(72%), condylar resorption (56%), and nonintact cortex (40%) were the most common degenerative changes found.[26]

To diagnose ICR, other potential underlying factors for condylar resorption must be evaluated. Local factors include OA, reactive arthritis, avascular necrosis, infection, and traumatic injuries, whereas systemic factors include rheumatoid arthritis, scleroderma, systemic lupus erythematosus, and Sjögren syndrome.[1] Congenital defects causing condylar hypoplasia are Treacher Collins syndrome, hemifacial microsomia, and hemimandibular hypoplasia with condylar-coronoid collapse.[27,28] Consultation with a rheumatologist is suggested to rule out the possibility of an autoimmune disease.[5] Distinctive characteristics of similar pathologies are important in the diagnosis of ICR. For instance, reactive arthritis appears in older people and causes decreased joint space between the condyle and fossa.[1,29] Juvenile idiopathic arthritis, which involves TMJ at a prevalence of 87%,[30] is often confused with ICR because of condylar hypoplasia and the age of affected patients. Young patients often present without overt TMJ symptoms.[31,32] In our patient, no joints other than the TMJ were involved.

In this study, the patient had clinical characteristics of both ICR and OA with her TMJ. Her symptoms were not typical clinical characteristics, which defined either OA or ICR. Anterior beaking and a flat shape of the condyle are characteristics of OA, but unlike OA cases including osteoarthrosis, DJDs, and posttraumatic arthritis where an open bite develops slowly and is minor, her anterior open bite developed quickly and advanced aggressively, which is consistent with ICR.[32] ICR is often classified as a low-inflammatory arthritic disorder or a severe form of OA.[8,33] However, others classify ICR as an individual pathology, distinct from OA.[1,34] Some clinical findings of ICR overlap those of OA; crepitus and breaks in the cortical outline can occur with both OA and ICR.[26,35] However, diagnostic characteristics, such as osteophytes and effusion, or an increased amount of fluid in the synovial compartment of a joint, are usually found in OA.[35] ICR involves increased joint space and can often have condylar resorption in all surfaces, whereas OA presents flattening of the superior or anterosuperior surface of the condylar head.[7,26,35-39] Therefore, ICR may be regarded as a separate pathology from OA, sharing some of the diagnostic characteristics with OA. Even so, there is a need for research on synovial fluid to confirm whether ICR should be classified as a low-inflammatory arthritic disorder.[25]

As its nickname, cheerleaders syndrome, implies, ICR frequently occurs among young females, possibly with a history of trauma.[2] The potential cause for ICR is a decreased ability of the articulating structures to adapt to excessive physical stress in conjunction with a hormonal effect.[13,40] The presence of estrogen receptors in the TMJs of female primates and the effect of estrogen on cartilage and bone metabolism may explain the high occurrence among females.[1,24,41,42] Low levels of circulating 17ß-estradiol prevent the condyle's ability to overcome local inflammatory factors, resulting in condylar lysis.[41] Mechanical loading of the condyles can increase with parafunctional habits, trauma, orthodontics, or orthognathic surgery.[43] Increased estradiol receptors may predispose an exaggerated response to joint loading while hormones mediate biochemical changes within the TMJ, causing synovial tissue hyperplasia.[7]

The Forsus (3M Unitek) appliance, which was used on the patient in this case report to correct Class II Division 1 malocclusion, has been evaluated for its potential effect on TMJ in different studies. The Forsus Nitinol Flat Spring (3M Unitek) was reported to increase the amount of repositioning of the condyle posteriorly in the glenoid fossa during the pubertal growth period in patients with Class II Division 1 malocclusions.[44] Based on a later

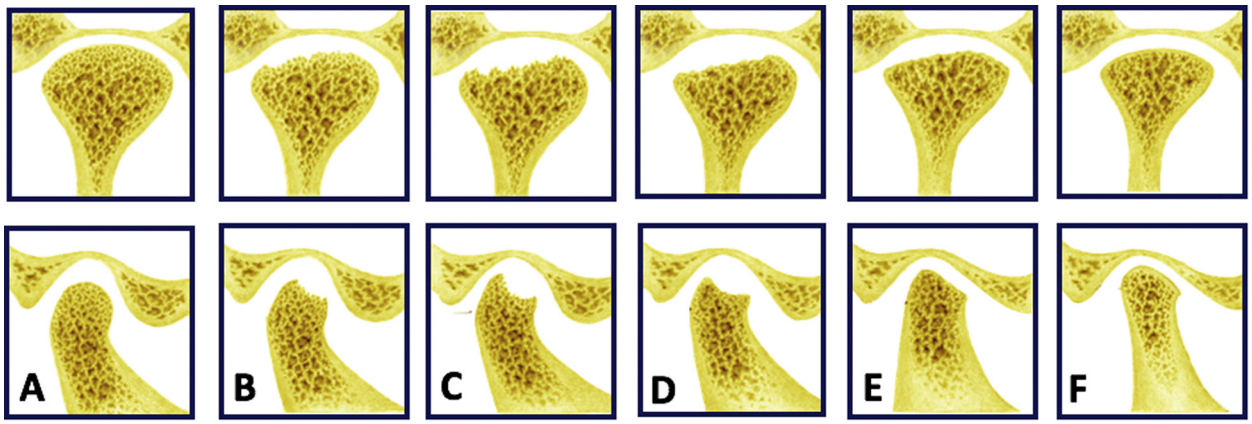

Fig 7. Phases of condylar degeneration from normal TMJ to destructive, repair, and finally stable stage. **A,** normal TMJ; **B,** beginning of active/destructive phase; **C,** "cup" shaped defect in superior surface of condyle (**B** and **C,** stages vulnerable to biomechanical force); **D,** beginning of repair stage; **E,** advanced repair phase; **F,** stable stage. Based on a figure originally published in Hatcher.[8] Modified and used with permission.

study conducted with the Forsus Fatigue Resistant Device (3M Unitek) by Arici et al,[45] treatment with this appliance did not result in statistically significant changes in the condyle position and did not cause a nonphysiological disc-condyle relationship. According to existing evidence, the effects of fixed functional appliances to correct Class II malocclusions seem to be mainly dentoalveolar rather than skeletal.[46] Therefore, the effect of the Forsus in TMJ because of changes in the dentoalveolar section may be better understood as more studies are conducted to confirm the relationship between occlusion and TMJ.

When ICR has been diagnosed as in this reported case, splint therapy is often considered to be the treatment of choice to unload condyles and prevent progression of disease or to relieve discomfort and muscle hyperactivity.[3,6,13] Occlusal splints are also used as a diagnostic tool to determine cessation of the resorptive process by evaluating whether the mandibular incisors are still in registered contact with the splint.[5,6,23] Other noninvasive treatment methods for TMD include the aforementioned exercise, manual therapy, and relaxation training. If these are not sufficient, corrective procedures including prosthodontics, orthodontics, and orthognathic surgery may be needed.[3,6,7]

Current TMD treatment methods are challenging to clinicians who seek to validate their practice with treatment methods that are supported by strong evidentiary standards.[47] Manfredini et al[48] compiled several major systemic reviews and meta-analyses dealing with the diagnosis and treatment of TMDs and failed to find strong evidence that occlusal splints were a benefit to patients with TMD because of the low quality of the studies or insufficient evidence within the studies.[49-51] Reviewed studies on TMD exercise, manual therapy, and orthognathic surgery also need clearer evidence to properly support their claims about the effectiveness on TMD.[52-54] Whereas these common treatment modalities for patients with TMD are apparently helping many patients, strong, scientific evidence is still necessary to confirm that these methods are reliable ways of treating patients with TMD.

Despite these criticisms, positive clinical results have been reported from specific surgical treatments on patients with ICR, but there is no clear consensus on the timing for surgical treatment. Whereas Wolford recommends surgical management at an early stage, another option is that surgical intervention should only be done after the cessation of the active resorption process.[1,9,22,25] If condylar resorption has been stable for at least 1 year, an intact cartilaginous cap over the resorbed condyle has to be confirmed for successful results. To confirm the status of the condyle, previous dental records can be compared with current records. A radioisotope bone scan of the TMJ may also be useful to detect the presence of resorptive activity in the condyle, despite a limitation in distinguishing between active ICR and postresorptive osseous remodeling.[9,12] Surgical treatment modalities include repositioning of the maxilla with Le Fort I osteotomy, ramus osteotomies on the mandible, oblique osteotomy of the chin, condylectomy with costochondral graft, alloplastic TMJ reconstruction, and other orthognathic surgical approaches.[3,9]

Surgical intervention requires careful planning to prevent and treat ICR effectively. The occurrence rate

of ICR after orthognathic surgery is reportedly between 5.8%-20%.[36,55,56] A higher rate of ICR was reported among patients with maxillary osteotomies and bilateral sagittal split osteotomy owing to the sudden repositioning of the condyles, which increases mechanical loading on the joint. For favorable surgical results with ICR, Reyneke and Ferretti[12] recommended surgical correction on the maxilla only if mandibular surgery can be avoided. They recommended a month of intermaxillary fixation rather than rigid fixation if mandibular surgery is needed. No orthodontic treatments that load the condyle, such as Class III elastics, should be used to avoid a possible reinitiation of the ICR.

High success rates without relapse are reported with specific forms of surgical treatments. Troulis et al[25] shared favorable treatment results using minimal endoscopic condylectomy and costochondral graft in their study. As needed, presurgical orthodontic treatment and Le Fort I maxillary osteotomy or genioplasty were undertaken. No adverse outcome was reported, and the relapse rate was insignificant.[25] Wolford and Cardenas[7] devised a treatment protocol to remove hyperplastic synovial tissue from the joint and reposition the articular disc. The condyle was stabilized with a Mitek mini anchor (Mitek Products Inc., Westwood, Mass) placed on the posterior aspect of the condylar head. Orthognathic surgery is then performed afterward to correct the associated jaw and occlusal deformities. Long-term follow-up of about 33 months demonstrated no change in jaw function and a significantly lower level of pain. Five patients younger than 16 years even exhibited small condylar growth with an average increase of 0.43 mm.[7] For growing patients, combining functional treatment and distraction osteogenesis is another treatment modality suggested to manage asymmetries and maldevelopment of the mandible and related structures.[57]

To avoid a treatment plan that "builds a house on sand," orthodontists should stay keen to the condition of the ground where the beautiful house is to be constructed. For an ideal finish and retention of the treatment, orthodontists should consider providing an ongoing monitoring of patients with risk factors for ICR. Throughout the course of treatment, regular clinical and radiographic evaluations along with input from a TMD specialist are recommended for detection of potential ICR. Treatment plans should be modified accordingly.

CONCLUSIONS

Because ICR may appear at the end of orthodontic treatment or during retention, orthodontists need to conduct careful assessments for ICR when young female patients with high mandibular angle and previous skeletal or dental Class II relationship present with open bite during retention. Moreover, despite having intact condyles, pretreatment patients with high risk factors for ICR should also be carefully monitored regularly with clinical and radiographic examinations throughout their orthodontic treatment to look for signs of ICR. Orthodontists should provide informed treatment options to the high-risk patients and the patients already diagnosed with ICR at any stage of orthodontic treatment, including the retention period. Treatment methods currently being used for ICR are reported to be clinically beneficial for patients, but there is a need for high quality scientific evidence to support such treatments routinely.

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