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# Progressive idiopathic condylar resorption: Three case reports

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Progressive condylar resorption, also known as idiopathic condylar resorption, is an uncommon, aggressive, degenerative disease of the temporomandibular joint (TMJ) seen mostly in adolescent girls and young women. This condition leads to loss of condylar bone mass, decrease of mandibular ramal height, steep mandibular and occlusal plane angles, and an anterior open bite. In 3 case reports, we review the pathogenesis of TMJ degenerative disease and the clinical management of TMJ arthrosis. We emphasize that TMJ arthritic disease should be discussed in dental circles as a pathologic entity in the same way that orthodontists discuss arthritic disease in orthopedic circles. Regarding the degenerative pathology of the TMJ, treatment goals include restored function and pain reduction. The treatment methods used to achieve these goals can range from noninvasive therapy to minimally invasive and invasive surgery. Most patients can be treated noninvasively, and the importance of disease prevention and conservative management in the overall treatment of TMJ disease must be acknowledged. The decision to manage TMJ osteoarthrosis surgically must be based on evaluation of the patient's response to noninvasive treatments, mandibular form and function, and effect of the condition on his or her quality of life. (Am J Orthod Dentofacial Orthop 2019;156:531-44)

Progressive condylar resorption (PCR) also known as idiopathic condylar resorption (ICR) is an uncommon, aggressive, degenerative disease of the temporomandibular joint (TMJ) seen mostly in adolescent and young women.[1,2] This disorder has a female prevalence of 9:1. Constitutional risks factors, besides female predilection, are hormonal imbalance (↓estrogen, ↓17β-estradiol), nutritional status (↓vitamin D, ↓dietary omega-3 fatty acids), bruxism, and repetitive oral habits. Iatrogenic causes have been reported to include orthodontics, orthognathic surgery, intermaxillary fixation, and improperly designed and used occlusal appliances, all resulting in condylar displacement and compressive TMJ overloading.[3]

## PATHOLOGIC CHANGES

PCR is best describe as a localized noninflammatory degenerative disorder of the TMJ and is characterized by lysis and repair of the articular fibrocartilage and underlying subchondral bone.[4] This condition leads to loss of condylar bone mass, decrease of mandibular ramal height, steep mandibular and occlusal plane angle, and an anterior open bite. Most of the destructive process is localized to the bone superior to a line bisecting the condylar poles. The active phase of PCR is often associated with limited jaw opening and TMJ pain, followed by condylar flattening. This may form a congruent articulation with the opposing posterior aspect of the articular eminence which permits redistribution of functional loads, thereby restoring some condylar motion and reduction in pain.

PCR occurring before the completion of growth results in a shorter condyloid process, shorter ramus, shorter mandibular body, compensatory growth at the gonial angle, and increased vertical dimension of the anterior facial region. As ramus height is lost, development of an anterior open bite is likely. There is a tendency for a reduction in airway dimension secondary to decreased mandibular growth[4] or, in the case of a patient who has completed growth, progressive mandibular retrusion.

Reduction in airway dimensions can lead to the risk of developing sleep apnea. Increased lower anterior facial height may cause lip incompetence in repose and reduced alveolar bone thickness at the facial aspect of the incisors.

## IMAGING MODALITIES

The orthopantomogram (OPG), as a panoramic radiograph is termed internationally, is the least

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expensive imaging modality for gross evaluation of the condyle. Loss of bone mass or flattening of the anterior or superior aspect of the condyle, as well as a distal inclination of the condylar neck, is easily observable on the OPG.

A cephalogram would display a shortened posterior facial height, increased anterior facial height, increased overjet, and open bite. Serial cephalograms taken during the active stages of PCR would show a more mesial position of the articulare point.[1]

Cone-beam computed tomography (CBCT) permits 3-dimensional evaluation of the condyle and helps to diagnose features such as condylar degeneration, erosion, sclerosis or flattening of the dense cortical layer, and subcortical cyst formation (Ely cyst).[1]

Magnetic resonance imaging (MRI) is the preferred technique for investigation of the soft tissues of the TMJ, including cartilaginous integrity of the articular surfaces, disk derangement, and inflammation. T1-weighted MRI is helpful in identifying disc position and alterations in bone and soft tissue anatomy, and T2-weighted MRI is useful for identifying inflammatory response as well as TMJ condylar bone marrow edema.[2,5,6]

Nuclear medicine bone scanning with the use of technecium-99 can be used to assess whether there are any active bony changes, but the specificity is not sufficient to assess the state of stability or remission of those changes.[1,4]

## PATHOGENESIS OF TMJ DEGENERATIVE DISEASES

The most common joint pathology affecting the TMJ is osteoarthrosis. Unlike rheumatoid arthritis, TMJ osteoarthrosis has a noninflammatory origin. The pathologic process is characterized by deterioration and abrasion of articular cartilage and local thickening and remodeling of the underlying bone. These changes are frequently accompanied by the superimposition of secondary inflammatory changes.[7] Three main etiologies[8] have been proposed for the pathogenesis of the disease: (1) trauma and or aberrant loading; (2) hormonal pathogenesis; and (3) a genetic basis for altered joint extracellular matrix.[7,9,10] These are not mutually exclusive, because a decreased adaptive capacity of the articulating structures, hormonal factors, and excessive physical stress on a joint can all induce dysfunctional remodeling.

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 theses 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 factor $\alpha$ 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[7] (Fig 1).

## CLINICAL MANAGEMENT OF TMJ OSTEOARTHROSIS

The management goals of TMJ osteoarthrosis should be: (1) decreasing joint pain, swelling, and muscle pain; (2) increasing joint function; (3) preventing further joint damage; and (4) preventing disability.

The first management option includes noninvasive modalities such as medications (nonsteroidal antiinflammatory drugs [NSAIDs] and muscle relaxants), physiotherapy, and occlusal appliance therapy. A second set of options includes minimally invasive modalities such as arthrocentesis (washing out the joint), injection of hyaluronic acid or a corticosteroid, and arthroscopic surgery. However, a recent meta-analysis concluded that there was little evidence to support the effectiveness of arthrocentesis in the management of TMJ osteoarthrosis.[11] Therefore, arthrocentesis can no longer be recommended for the management of TMJ osteoarthrosis. The third set of options involve invasive surgical modalities such as arthroplasty, autogenous hemiarthroplasty, discectomy, and disc repositioning by means of orthognathic surgery.[2] Goncalves et al[12,13] reported that articular disc repositioning in patients treated with maxillomandibular advancement and disc repositioning had better long-term outcomes with less relapse compared with a group of patients undergoing only maxillomandibular advancement surgery. However, the disc must be intact and the patient in the early stages of the disease.

End-stage PCR requires salvage procedures to restore jaw function and improve and maintain skeletal alterations.[2,7] These patients require either autogenous or alloplastic total joint replacement.

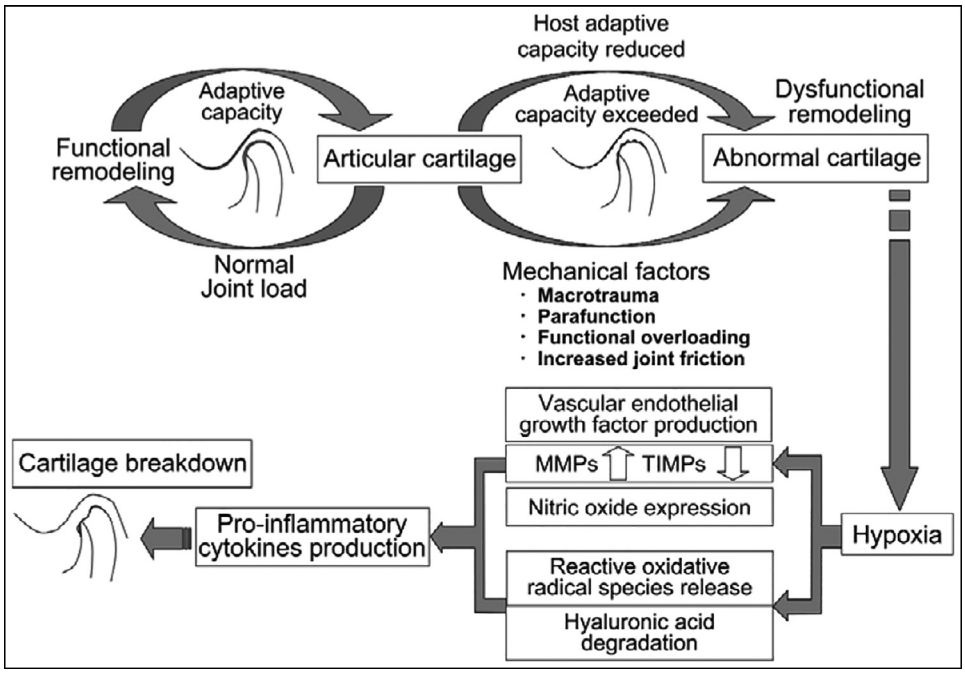


**Fig 1.** The concept of the process of cartilage breakdown in the TMJ. From J Dent Res 2008,87:296-307, used by permission of the publisher, Sage Publications/Corwin.

## CASE REPORTS

### Patient 1

A woman 19 years 1 month of age presented for consultation regarding possible orthodontic treatment for complaints of "crooked teeth" and TMJ pain. Her TMJ pain problems began at age 16. At 17, she had conservative treatment with occlusal appliance therapy, physiotherapy, muscle relaxants, and NSAIDs. A year later, she was diagnosed with disc displacement without reduction, limiting jaw opening to 20 mm. An oral surgeon did an infiltration of a local anesthetic with epinephrine and 40 mg triamcinolone (40 g/mL). Disc reduction was observed after infiltration, with an interincisal opening of 35 mm. TMJ and myofascial pain continued despite this treatment.

She went to a second oral surgeon for consultation, and a month later bilateral TMJ arthrocentesis was performed. One month later, she had persistent pain in the masseters and both joints and limited mouth opening.

A third oral surgeon then infiltrated 200 U Botox (dilution 100 U/mL) into masseter muscle trigger points. This was followed in 1 month by infiltration of 1 mg Decadron into each masseter.

Clinical examination revealed a Class III subdivision left dental relationship, moderate crowding, and the lower midline deviated to the right (Fig 2). The cephalometric analysis demonstrated a dentoalveolar bimaxillary protrusion, Class I skeletal relationship (Wits 1 mm), hyperdivergent (FMA 38°), short ramus, excessive anterior face height, and retrusive chin (Fig 3, *A*). Her profile was convex, and her lips were incompetent in repose (Fig 3, *B*).

The OPG revealed a flattened anterosuperior surface of the left condyle with an anterior osteophyte. The articular eminence also appeared flattened. The right condyle appeared normal. However, both condyles had shortened condyloid processes (Fig 4).

The treatment plan included extraction of the 4 first premolar teeth for maximal anterior retraction and a functional genioplasty to help obtain lip competency and normal anterior facial height. After 13 months of orthodontic treatment, her teeth were aligned and the extraction spaces were ready to close (Fig 5, *Top*). Joint pain and limited mouth opening were still issues. An MRI revealed bilateral anterior disc displacement without reduction.

After 18 months of orthodontic treatment, bilateral disc repositioning, extraction of all 4 third molar teeth, and a functional genioplasty were performed. Orthodontic progress records at 19 months showed a Class I occlusion with some remaining space to be closed (Fig 5, *Bottom*). A profile view showed reduction of the dentoalveolar protrusion, and a genioplasty helped in achieving lip competence in repose (Fig 6).

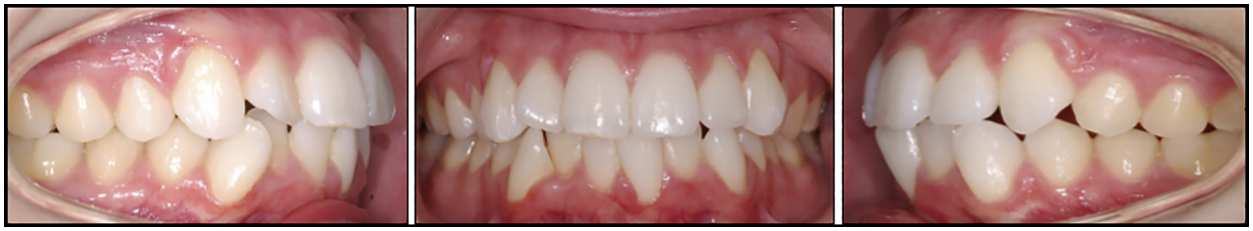

**Fig 2.** Patient 1 was a 19-year-old woman with Class III subdivision left malocclusion and moderate crowding. The lower midline was deviated to the right.

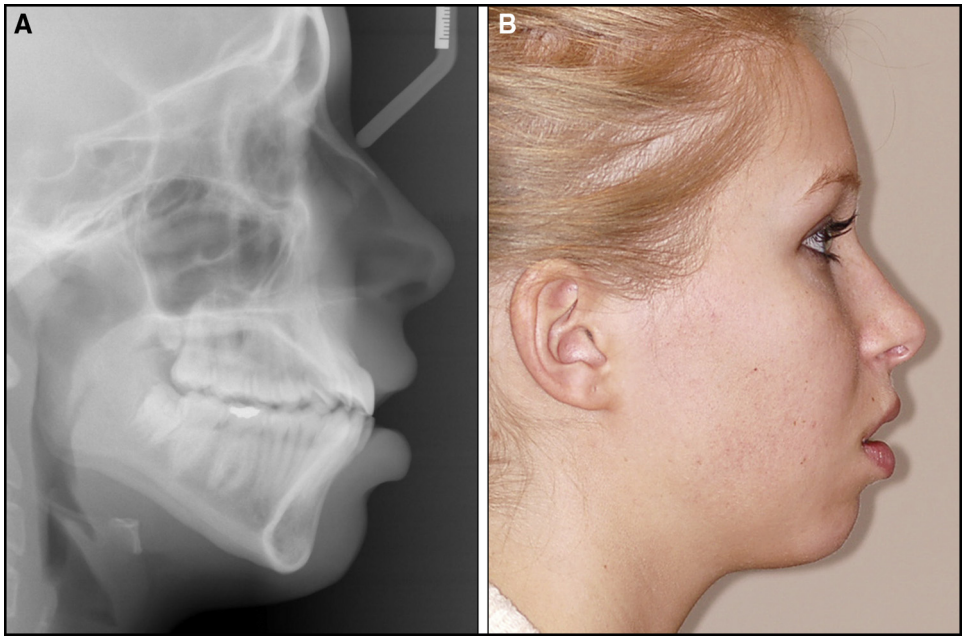


**Fig 3. A,** Lateral cephalogram shows a hyperdivergent short ramus, excessive anterior face height, and retrusive chin. **B,** Facial photograph reveals a convex profile and lip incompetency in repose.

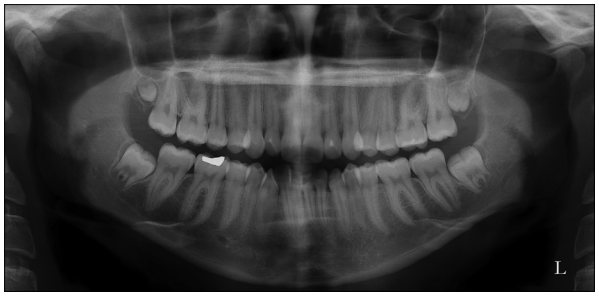

**Fig 4.** OPG taken before orthodontic treatment shows a flattened anterosuperior surface of the left condyle with an anterior osteophyte. The articular eminence also appears flattened.

Two months after disc repositioning, the patient's masseter muscle and TMJ pain increased. CBCT demonstrated a decrease in articular disc space bilaterally (Fig 7). Physiotherapy was recommended, and Flexeril and NSAIDs were prescribed. Three months after surgery, the right TMJ and right masseter muscle pain significantly increased (7/10). One mL dexamethasone was injected into the right TMJ. One week later at follow-up, the TMJ pain decreased to 1/10.

The patient was then transferred to an oral surgeon at Hôpital de l'Enfant-Jésus in Quebec City. A right TMJ discectomy was performed in February 2015. At the September 2015 follow-up, the right condyle showed significant resorption, reduced interarticular space, and flattening of the articular eminence. The left condyle showed progression of the flattening of the anterosuperior surface. In February 2016, a left TMJ discectomy was performed.

At 40 months, orthodontic treatment was completed. A functional occlusion was achieved with minimal

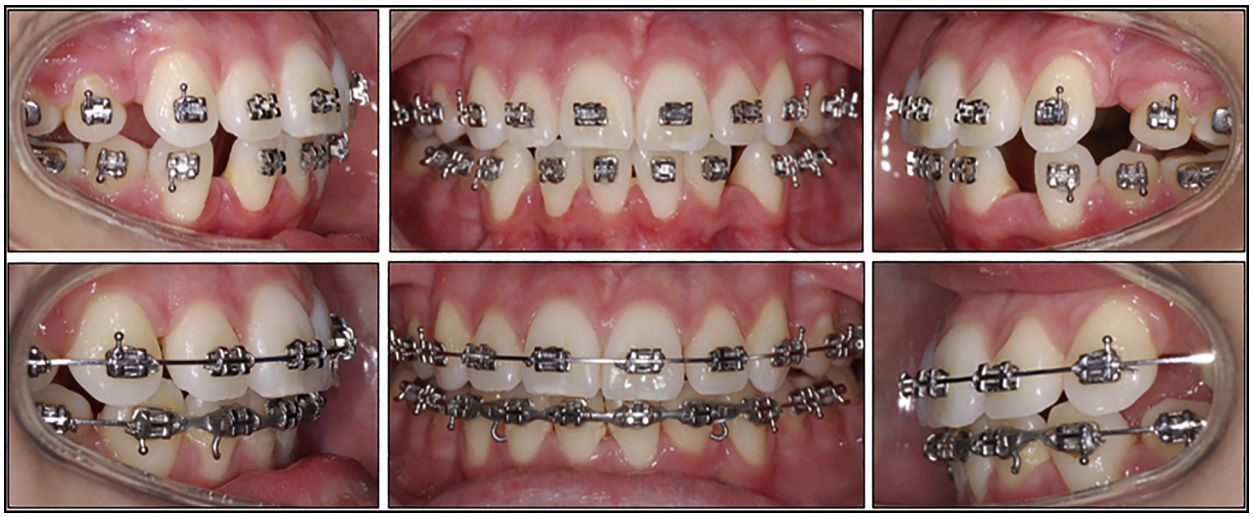

**Fig 5.** Top: Progress records at 13 months. Extractions spaces are ready to close. Bottom: Progress records at 19 months. Class I occlusion with some space remaining.

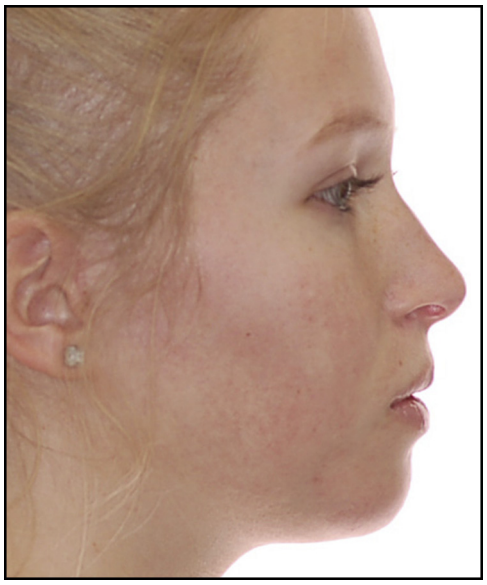

**Fig 6.** Profile view at 13 months shows reduction of the dentoalveolar protrusion and lip competence in repose.

overjet and overbite. The OPG revealed a progressive loss of right condylar bone mass (Fig 8).

Four months after debanding, an anterior open bite had developed and continued to progress to the point that it was decided that total joint replacement was the only management option (Fig 9). Bilateral alloplastic total joint replacement was performed in July 2017 with the use of Zimmer Biomet (Jacksonville, Fla) custom prostheses (Fig 10). Follow-up records demonstrate that a functional occlusion was established (Fig 10), despite the lack of lateral movement of the jaw, and her facial esthetics were improved (Fig 11).

### Patient 2

Patient 2, a young woman, presented with a Class II Division 1 malocclusion and an anterior open bite (Fig 12). She had been followed by her dentist for TMJ pain for the past 7 years. She had undergone orthodontic treatment 12 years earlier for a Class I malocclusion with severe crowding and a mandibular midline deviation to the left (Fig 13, *Top*). Four second premolars were extracted to alleviate crowding and the orthodontic treatment proceeded uneventfully. A Class I occlusion was achieved, although a slight lower midline deviation to the left remained (Fig 13, *Bottom*). The OPG at debanding revealed a shortened left condyloid process that might explain the midline deviation to the left and facial asymmetry (Fig 14, *A*). Follow-up 1 year into retention showed a normal occlusion without TMJ symptoms. The patient's profile was straight, with lip competence in repose. The cephalogram revealed a normodivergent mandibular plane, and the gonial angle was at the level of C2. The airway appeared normal (Fig 15).

During the period between debanding and her visit 7 years later, she had developed significant TMJ pain. Her general dentist made 3 splints over the years. She stated that 1.5 years after debanding, she experienced a sudden left TMJ lock with pain. A TMJ MRI revealed

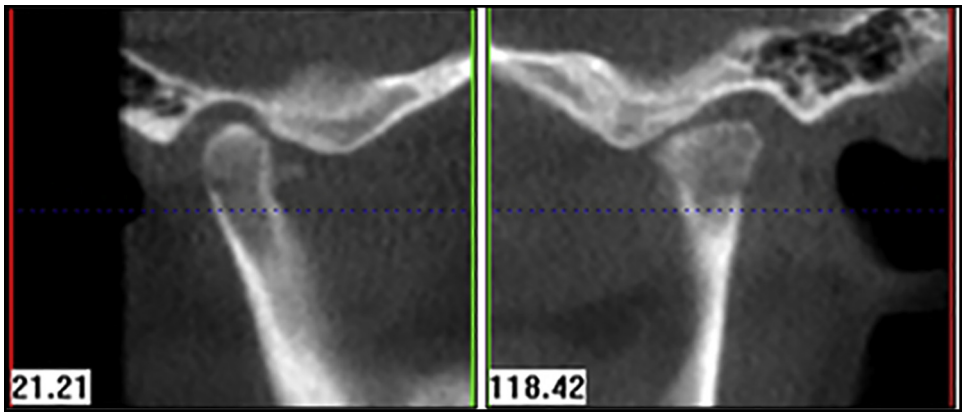


**Fig 7.** CBCT 2 months after disc repositioning. Note the decreased articular disc space bilaterally.

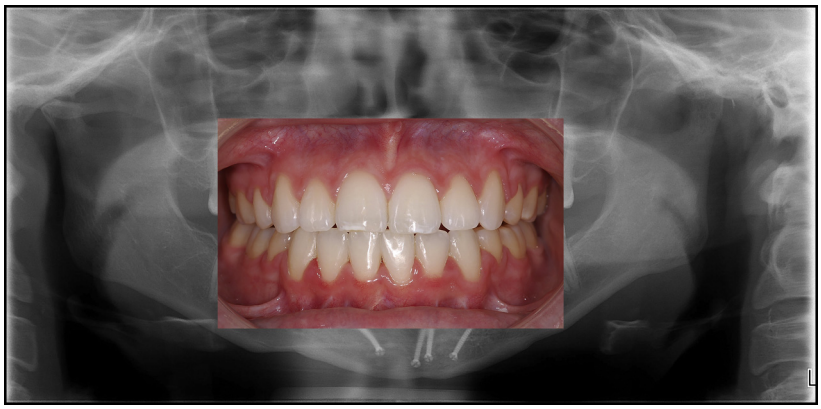

**Fig 8.** Intraoral view shows that a Class I occlusion was achieved. The OPG in the background shows loss of right condylar bone.

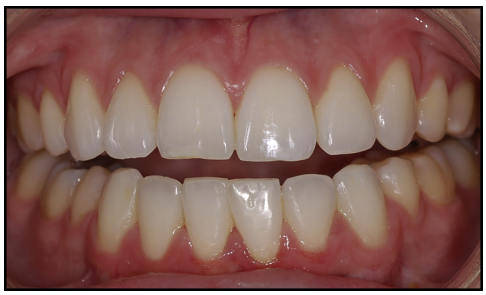

**Fig 9.** Intraoral photograph 12 months into retention shows anterior open bite.

bilateral disc displacements. On opening, the right disc reduced, but the left side did not. No condylar degenerative changes were seen. Occlusal appliance therapy was started.

Over the ensuing years, the patient gave birth to 3 children. New occlusal appliances were made after each birth. The OPG 4 months after the birth of the first child revealed noticeable changes in both condyles (Fig 14, *B*). A concavity was seen on the superior aspect of left condyle, and resorption was seen in the lateral pole of the right condyle. She was referred to a rheumatologist for an autoimmune work-up. The physical examination and serology were inconclusive. However, the patient complained of eczema and reported dull pain in her left wrist and such acute pain in her knees that she could no longer jog.

An OPG made after the third child's birth revealed severe bilateral condylar resorption. A new occlusal appliance was fabricated. The patient also noted that her open bite had increased drastically, even with the oral appliance. Cephalometric analysis demonstrated decreased posterior face height, short mandibular ramus, high mandibular plane angle, anterior open bite, and retrognathic mandible. Study models taken at that time could be hand articulated into a Class I occlusion,

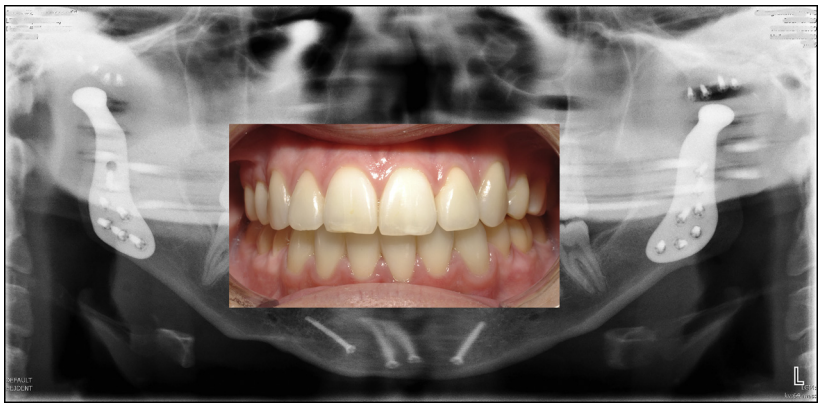

**Fig 10.** OPG 1 month after Zimmer Biomet custom total joint replacement. Class I functional occlusion was achieved.

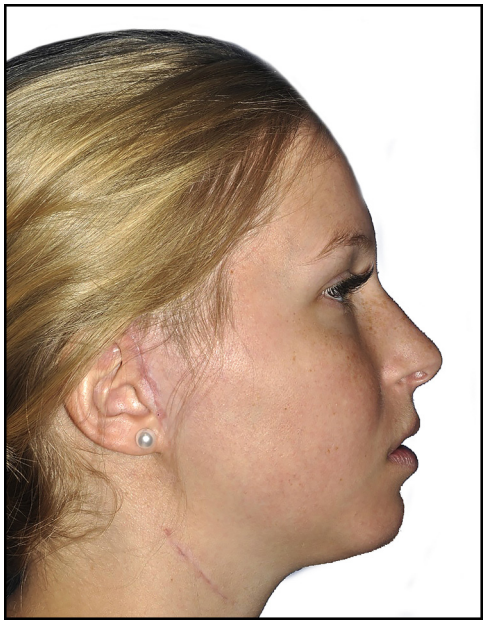

**Fig 11.** Profile view, 1 month after total joint replacement.

demonstrating that no dental changes had occurred. One year later, an OPG revealed complete degeneration of the mandibular condyles bilaterally (Fig 16). The intraoral examination showed a Class II occlusion with a significant anterior open bite (Fig 12). Her profile showed a recessive mandible and chin. She complained of sleep apnea symptoms. The cephalogram revealed significant shortening of the ramus, a gonial angle that had moved from C2 level to C1, and a significantly reduced airway shadow (Fig 17).

After discussion, it was decided that the best management option for this patient would be bilateral alloplastic total joint replacement (Zimmer Biomet custom prostheses; Fig 18).

Fifteen days after surgery the patient had a Class I functional occlusion (Fig 19). Her profile was significantly improved, her chin/throat projection increased, and her lips appeared competent in repose. The lateral cephalogram demonstrated an increased airways profile and a normal overjet and overbite (Fig 20). She had an interincisal opening of 20 mm with 2 mm lateral excursion. This can be considered normal at 15 days after surgery. Physiotherapy was then prescribed.

### Patient 3

A girl 10 years 10 months of age had a Class II subdivision left malocclusion with slight crowding and moderate curve of Spee. Class II correction was achieved with the use of Class II elastic traction. Near the completion of orthodontic treatment, relapse in the occlusion was noted. A fixed twin force bite corrector (TFBC) functional appliance was used.

She returned on an emergency basis 3 weeks later owing to acute pain in the left TMJ and decreased interincisal opening (25 mm). The TFBC was immediately removed. A left TMJ disc displacement without reduction was diagnosed clinically. After physiotherapeutic manipulation of her mandible, normal jaw mobility was regained and 400 mg ibuprofen every 4 hours for 4 days was prescribed, followed by 400 mg every 6 hours for 3 days. Follow-up at 14 days showed an interincisal opening of 45 mm without pain.

Three weeks later, the patient developed another closed-lock with left TMJ pain. She was then referred to a physical therapist and instructed to take the ibuprofen as previously prescribed for another week. Two months later, at debanding, her interincisal

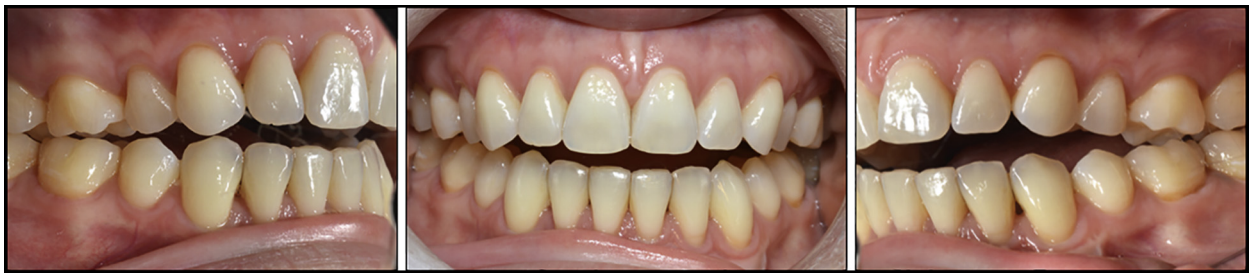

**Fig 12.** Patient 2. Adult woman with Class II Division 1 malocclusion, anterior open bite, and a 7-year history of TMJ pain.

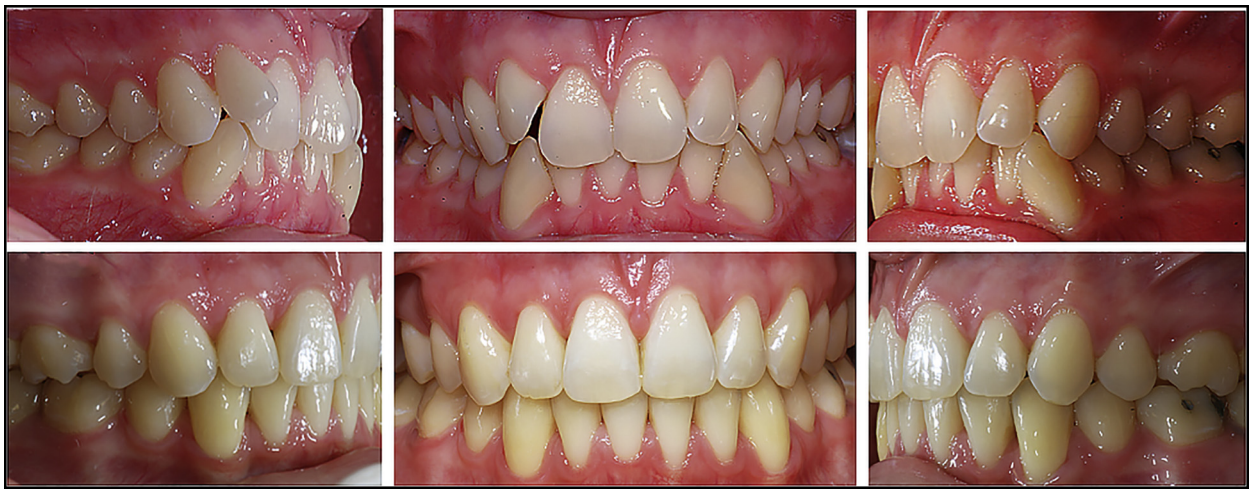

**Fig 13.** Initial photos (top) show class I occlusion with moderate crowding. Final photos (bottom) show Class I occlusion after extraction of 4 second premolars.

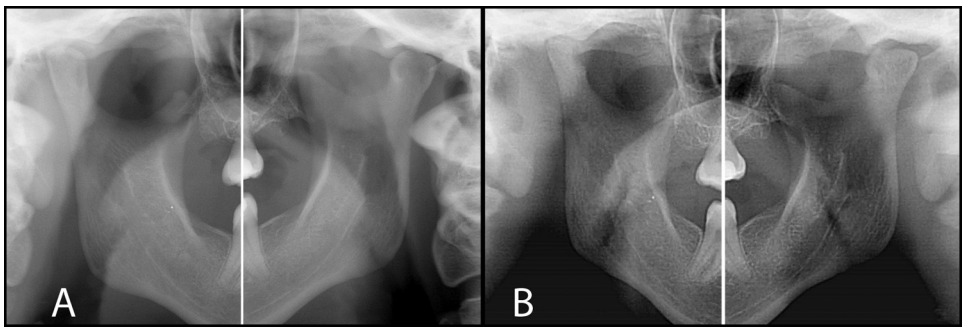


**Fig 14. A,** OPG after at debanding shows a shortened left condyloid process, similar to initial OPG. **B,** OPG 4 months after the birth of her first child shows concavity on the top of the left condyle and resorption of the lateral pole of the right condyle.

opening was 41 mm with no pain and a Class I occlusion (Fig 21, *A*). However, the OPG revealed flattening of the anterosuperior surfaces of both condyles (Fig 22, *A*). This suggested incipient condylar morphology change likely due to resorption. Follow-up records 3 and 9 months into retention showed progressive bite opening (Figs 21, *B* and *C*). The OPG 9 months into retention revealed signs of significant TMJ PCR (Fig 22, *B*), and the OPG at debanding showed bilateral condylar flattening. This

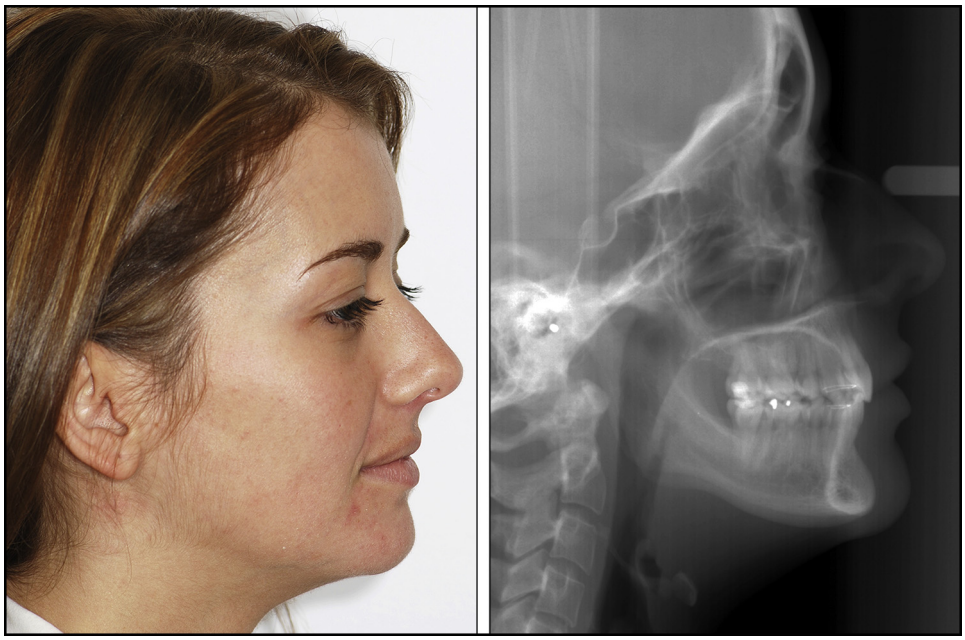

**Fig 15.** Profile and cephalogram at the end of orthodontic treatment revealed a normodivergent mandibular plane as well as normal airway width and lip competence in repose.

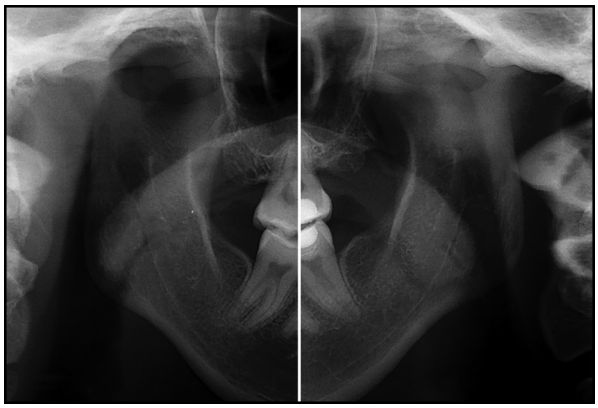

**Fig 16.** OPG 1 year after the birth of her third child shows complete degeneration of mandibular condyles bilaterally.

may explain relapse toward class II that was noted toward the end of treatment.

Calcium (500 mg) plus vitamin D (1000 UI) once per day was prescribed for 30 days. She was referred to an oral surgeon and a rheumatologist. Blood testing was negative for any systemic pathology (rheumatoid factor, anti–cyclic citrullinated peptide, C-reactive protein, antinuclear antibody, 17β-estradiol, and vitamin D levels were normal).

Twenty months into retention, she had an anterior open bite of 3 mm, slightly more severe on the left side (Fig 23). She reported often feeling dull pain in the right joint. On OPG, both condyles showed significant flattening of the anterosuperior aspect and the condylar neck was inclined posteriorly. The right joint showed more flattening than the left joint, suggesting more resorption (Fig 24). This might explain why the bite was more open on the left side.

Instructions were reinforced that the patient should not chew gum and should resume daily vitamin D and calcium. Despite the rheumatologist finding no evidence, we suspected that this could be a case of oligoarticular subtype of juvenile idiopathic arthritis (JIA) rather than ICR. Further investigation is necessary to obtain a final diagnosis.

## DISCUSSION

Patients 1 and 2 had end-stage condylar resorption resulting in end-stage diseased TMJs requiring salvage total joint replacement. Patients 1 and 3 had the onset of symptoms at ages 13-15 years and could be diagnosed with ICR. Case 2 developed symptoms in her mid-20s when she reported sudden locking of her left TMJ. Because she also reported mild knee and wrist pain and eczema, a form of inflammatory arthritis was suspected. Further, 3 pregnancies likely played a role in dysfunctional TMJ condylar remodeling and resorption.[9,14]

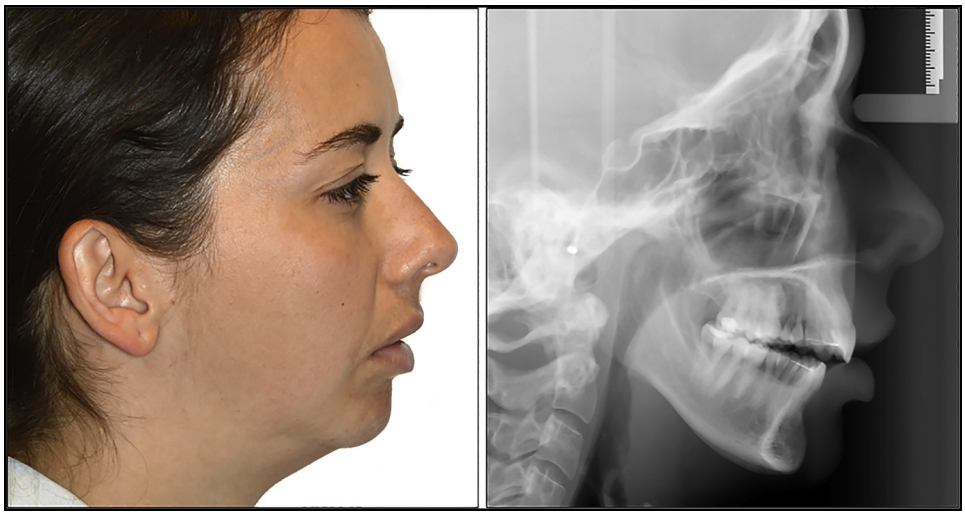

**Fig 17.** Profile view shows recessive mandible and chin. Cephalogram reveals significant shortening of the ramus. Gonial angle moved from C2 to C1 and reduced oropharyngeal airway shadow.

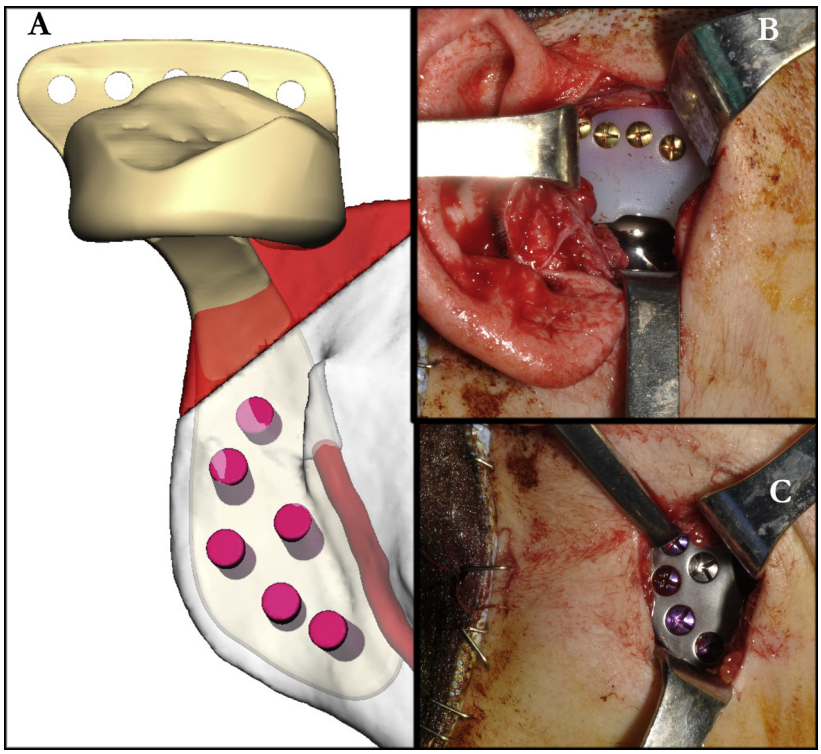


**Fig 18.** Left total joint prosthesis (Zimmer Biomet).

There is growing evidence that sustained inflammation induces degeneration of the TMJ[15-17] and can lead to deterioration of the joint's mechanical properties as well as alteration of the disc ultrastructure which might contribute to TMJ disc displacement.[16]

This agrees with Wolford's hypothesis[2] that female hormones can influence biomechanical change within the TMJ, causing hyperplasia of the synovial tissues. This would stimulate the production of cytokines that initiate breakdown of the ligamentous structure that normally support and stabilize the articular disc with

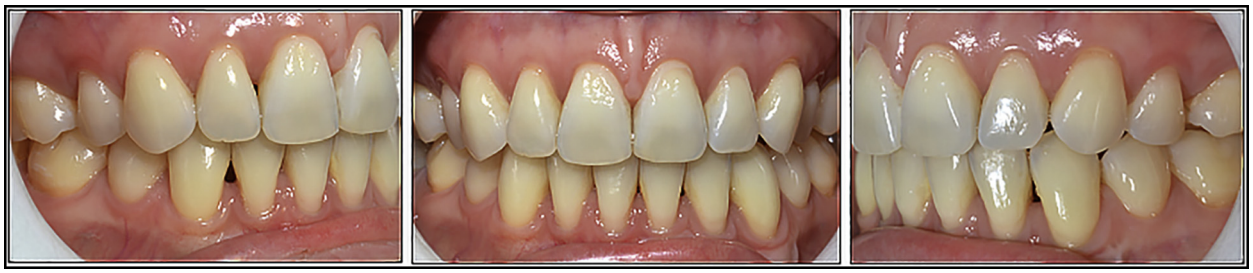

**Fig 19.** Intraoral photograph 15 days after surgery shows a Class I occlusion.

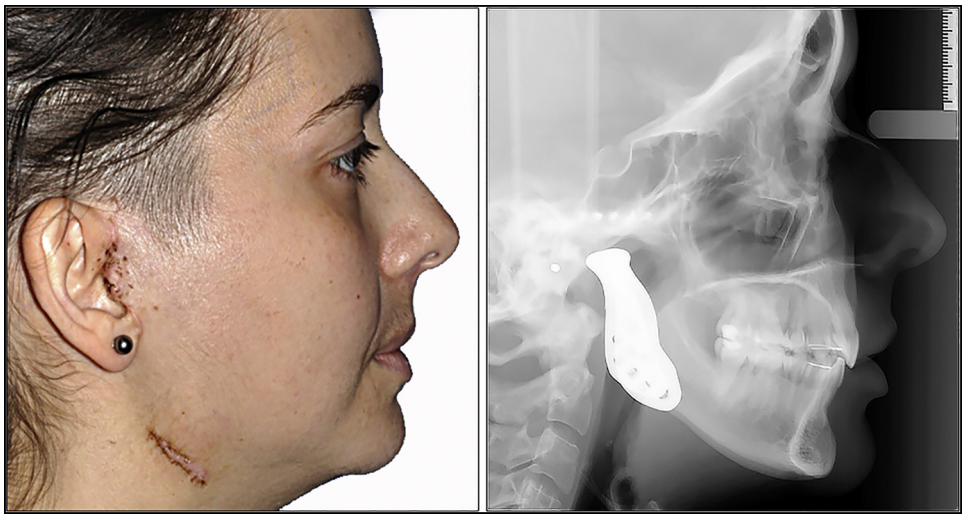

**Fig 20.** Profile photograph 15 days after surgery shows improved chin projection, decreased anterior facial height, and lip competence in repose. The cephalogram demonstrates an increased airway profile and a normal dental overjet and overbite.

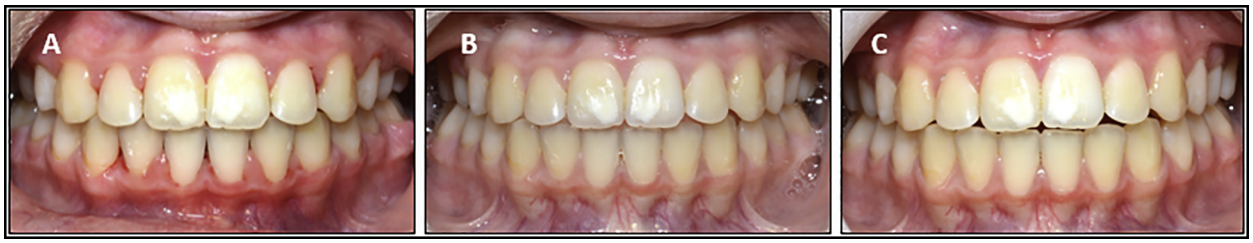


**Fig 21.** Patient 3. **A,** Frontal view at debanding. **B,** Follow-up at 3 months. **C,** Follow-up at 9 months. Note progressive bite opening within 9 months.

the condyle, resulting in anterior disc displacement. According to this hypothesis, the cytokines penetrate through the outer surface of the condyle and cause thinning of the cortical bone leading to breakdown of the subcortical bone. The condyle slowly collapses without clinically apparent destruction of the fibrocartilage. In patients where the pathologic process is in remission, excessive joint loading (ie, parafunctional habits, trauma, orthodontics, orthognathic surgery) can reinitiate the resorption process.

Symptoms of TMJ synovitis include pain during jaw movement, crepitus, and restricted mouth opening. Isolated TMJ synovitis can be a presentation of the oligoarticular subtype of JIA.[18] It remains unknown whether JIA-related TMJ arthrosis and ICR are distinct conditions. Isolated TMJ arthrosis may be the first or only

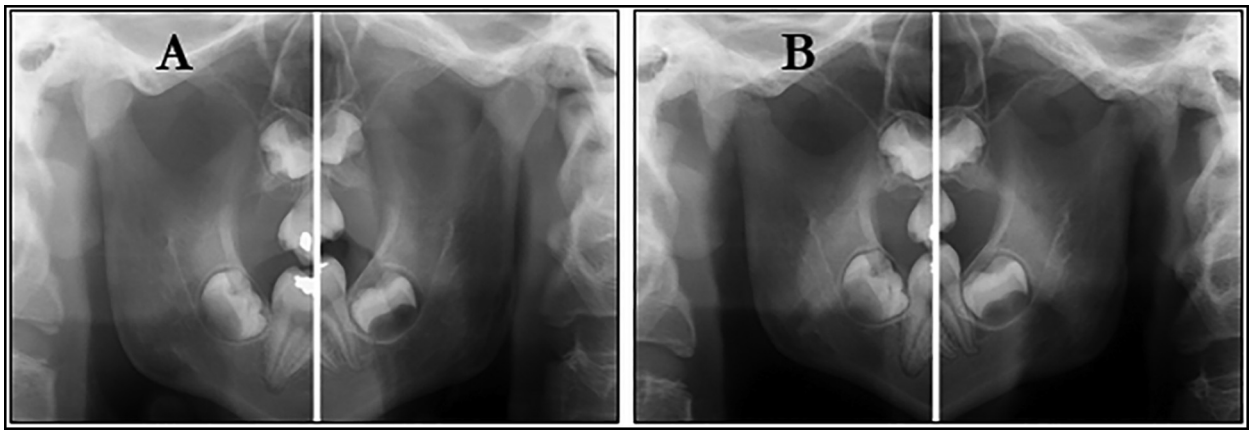


**Fig 22. A,** OPG at debanding. Flattening of both anterosuperior superior surfaces of both condyles is evident. **B,** Follow-up at 9 months after orthodontic treatment.

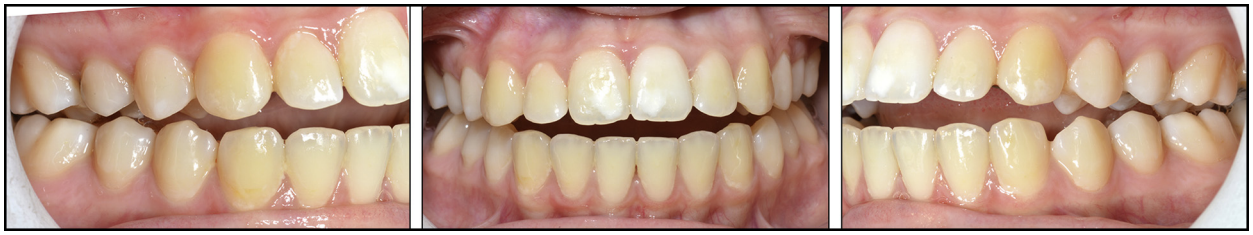

**Fig 23.** Follow-up at 20 months after treatment shows 3 mm anterior open bite.

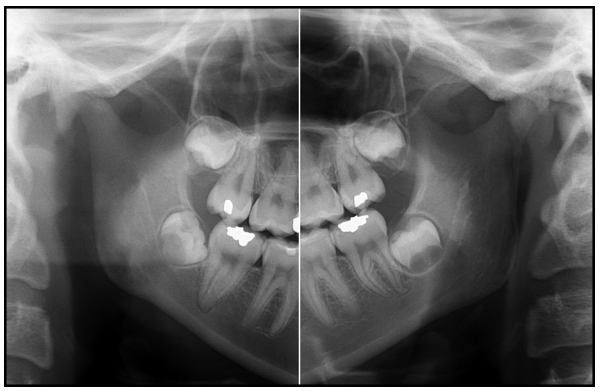

**Fig 24.** OPG at 20 months' follow-up. Both condyles show significant flattening of the anterosuperior aspect, and the condylar neck is inclined posteriorly. The right joint shows more flattening than the left, suggesting more resorption.

manifestation of JIA and may not be as rare as previously reported. Many patients are seen initially by a dentist or an orthodontist who may not be familiar with JIA, so they misdiagnose the patient with either ICR or some other TMJ disorder.[19]

Epidemiologic studies have confirmed the higher prevalence of TMJ disease and pain in women than in men. Estrogen receptors have been identified in TMJs and may regulate the synthesis of proteins involved in articular tissue turnover in the TMJ. Estrogens enhance responses to relaxin, a polypeptide implicated in MMP synthesis and activation. MMPs have been implicated in the degradation of the cartilaginous matrices in degenerative TMJ diseases.[10] There is evidence that relaxin contributes to the degradative remodeling of joint fibrocartilage and that there is an association between relaxin-induced MMPs and matrix loss, suggesting a potential mechanism of action of relaxin in contributing to TMJ diseases in a subset of women with these disorders.[20] These findings show that relaxin, which is found systemically in cycling and pregnant women but not in men, causes the targeted induction of tissue-degrading enzymes of the MMP family in the fibrocartilaginous tissues of the TMJ, potentially predisposing to TMJ disease. Moreover, it has been found that the TMJ disc and pubic symphysis show the greatest induction of MMPs and matrix loss in response to relaxin and 17β-estradiol.[14,21] This helps to explain the role of hormones in the disease of patient 2.

## WHAT TO DO?

Early diagnosis of TMJ degenerative change should include a careful examination of the condyle and condyloid process on the screening OPG. Signs of degenerative bony condylar changes or condyloid process shortening may be present despite absence of clinical symptoms. The suspicion and recognition of these imaging changes, plus awareness of any clinical signs and symptoms, may be an indication for more sophisticated imaging (CBCT, MRI, nuclear medicine scan), blood testing, and consultation. Blood tests should include erythrocyte sedimentation rate and C-reactive protein, antinuclear antibody, rheumatoid factor, anti–cyclic citrullinated peptide, vitamin D, and 17β-estradiol levels.

Antiinflammatory medication such as the NSAIDs (Naproxen, Celebrex, Feldene), as well as vitamin D and calcium supplementation, both of which are known to increase bone density, should be prescribed. Colleague rheumatologists with an understanding of this TMJ pathology should manage medications such as methotrexate or etanercept.[17,22,23]

## CONCLUSION

Cases such as the 3 reported here can be found in any orthodontic practice. If we are at fault, it is likely because we looked at this pathology as a dental problem. Maybe it is time that we look at TMJ arthrosis as a systemic pathology. To avoid the outcome of deleterious skeletal change and unsalvageable TMJ, the orthodontist should be able to make an early diagnosis and early treatment "en amont" (upstream) of the skeletal changes.

The mandible contains teeth as the end-organ for TMJ function. This has led some within the dental profession to embrace the concept that the presence of teeth makes the TMJ a unique articulation. This has in the past resulted in those practitioners focusing their diagnosis and management of TMJ disorders on the occlusion, despite no supporting evidence.[24,25]

All 3 patients were treated to a Class I functional occlusion. The postorthodontic continuation of joint problems does not support the theory that Class I occlusion, canine guidance, incisor guidance, or balanced occlusal contact would avoid or prevent TMJ problems. Given the cycle of TMJ arthrosis that can go from active to inactive, it may mean that using a TMJ splint for pain relief may be a matter of chance that splint use is initiated before the remission period.

In conclusion, it is essential that TMJ arthritic disease be discussed in dental circles as a pathologic entity in the same way our colleagues discuss arthritic disease in orthopedic circles. Not doing this only exacerbates the problem that everyone has with TMJ disorders in genera—patients, clinicians, insurance carriers, etc—because they do not consider TMJ pathology as orthopedic pathology, but as just dental.

Further studies are necessary to determine the true frequency of isolated TMJ arthrosis in JIA and explore other possible causes for isolated TMJ arthrosis as well as the optimal therapy.

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