ELSEVIER
SAUNDERS

Oral Maxillofacial Surg Clin N Am 19 (2007) 223–234

ORAL AND
MAXILLOFACIAL
SURGERY CLINICS
of North America

Condylar Resorption

Maria E. Papadaki, DMD, MD, Fardad Tayebaty,
Leonard B. Kaban, DMD, MD, Maria J. Troulis, DDS, MSc*

Department of Oral and Maxillofacial Surgery, Massachusetts General Hospital, Harvard School of Dental Medicine, 55 Fruit Street, Warren Bldg. 1201, Boston, MA 02114, USA

Various pathologic conditions, such as osteoarthritis, rheumatoid arthritis, condylar hyperplasia, and idiopathic condylar resorption (ICR), affect the temporomandibular joints (TMJ). As a result, the size and morphology of the condyles may be altered. Both men and women are affected; however, more so than the other conditions, ICR is almost exclusively found in women [1]. In this article, the diagnosis and management of ICR are discussed.

ICR is a poorly defined acquired disorder that is characterized by progressive decrease in condylar mass and alteration of condylar shape. It is almost always bilateral and seems to have a high predilection for women in the age range of 15 to 35 years [1]. Other terms for this condition include condylysis, osteoarthrosis, dysfunctional remodeling, avascular necrosis, osteonecrosis, and condylar atrophy. Resorption may progress to include the entire condyle, leaving only a stump at the inferior aspect of the sigmoid notch. The mandibular ramus below the sigmoid notch is spared.

The resorptive process may become quiescent after 1 to 5 years but also can be reactivated; hence, the distinction between active and inactive condylar resorption. After age 40, it is rare for patients to experience further resorption.

Condylar resorption was first reported by Burke in 1961 [2]. He described it as an acquired condylar hypoplasia. By 1977, condylar resorption was distinguished from congenital condylar aplasia or hypoplasia, and the progressive lytic (destructive) nature of the condition was documented [3].

Etiologic theories

General considerations

The exact cause and pathogenesis of condylar resorption remain unclear. It has been associated with rheumatoid arthritis, TMJ internal derangement, trauma, steroid use, systematic lupus erythematosus, scleroderma, other vascular collagen diseases, orthodontic treatment, and orthognathic surgery (secondary condylar resorption) [4,5]. In most cases, however, there is no identifiable precipitating event; hence, the term "idiopathic condylar resorption." De Bont and Stegenga [6] subdivided condylar resorption into primary (idiopathic) and secondary, depending on the presence of predisposing factors [7].

Two theories have been described with regard to the pathogenesis of condylar resorption. Arnett and colleagues [8] correlated it with increased, abnormal joint loading and subsequent pressure resorption, as might occur after orthodontics, orthognathic surgery, occlusal therapy, internal derangement, parafunction, trauma, and unstable occlusion [9]. Chuong and colleagues [10,11] postulated that the mechanism of condylar resorption was similar to that of avascular necrosis of the femoral head. Intraluminal or extraluminal obliteration of small vessels, damage to the arterial or venous walls, and increased intraosseous pressure from enlargement of intramedullary fat cells or osteocytes are factors that contribute to the development of avascular necrosis of the femoral head [12]. Theoretically these conditions could occur in the mandibular condyle. In 1989, Schellhas

* Corresponding author.
E-mail address: mtroulis@partners.org (M.J. Troulis).

doi:10.1016/j.coms.2007.01.002

and colleagues [13] also reported MRI findings of unilateral or bilateral avascular necrosis of the condyle in five patients who had undergone orthognathic surgery, including sagittal split osteotomies, followed by intermaxillary fixation.

Condylar resorption also has been considered to be the result of loss of the normal remodeling capacity of the condyle caused by factors such as age, systemic illness, and hormones [8,9]. Wolford and Cardenas [14] believe that the mediators to promote condylar resorption exist in the bilaminar zone of the meniscus. In a series of 15 patients with ICR, they found hypertrophy and proliferation of bilaminar and synovial tissues around the head of the condyle. They hypothesized that exposure of the condyles to mediators in these tissues creates the resorptive phenomenon [15]. The experience of our group does not support this hypothesis. At the time of operation, we have found no abnormalities in the synovial tissues. Condylectomy and reconstruction with a costochondral graft (CCG) result in recreation and remodeling of the condyle, and there is no further resorption of the "neocondyle" with time, despite contact with the synovial tissue.

Idiopathic and secondary condylar resorption affect men, but they are more frequently seen in women, with a 9:1 ratio [15–17]. The female preponderance may be attributed to the modulation of the bone response by estrogen and prolactin [8,17–19]. Estrogen receptors have been found in the temporomandibular disk of women [20] and in the condyle and disc of female animals [21,22]. In a study of the human TMJ, Abubaker and colleagues [20] reported that estrogen and progesterone receptors were found five times more often in women with symptoms of TMJ internal derangement than in asymptomatic women. Seven TMJ disc specimens obtained from women with TMJ symptoms were analyzed for the presence of these receptors. These specimens were compared with another group of 15 TMJ discs removed from men and women with no TMJ symptoms who were undergoing skull base surgery and who had extirpation of the TMJ for access to the skull base. Detection of hormone receptors was performed with immunohistochemistry. It was concluded that the TMJ disc is potentially a female sex hormone target tissue in some patients and that women may have different responses to estrogen levels, based on the target receptors within the TMJ. More studies should be performed to determine the role of hormones in ICR.

Orthognathic surgery and condylar resorption

Most of the literature on condylar resorption discusses its relationship to orthognathic surgery. In 1978, Phillips and Bell [23] first reported bilateral atrophy of the mandibular condyles after sagittal split osteotomy for mandibular advancement. Although a definite cause could not be established, they hypothesized that a biomechanical phenomenon based on increased muscle tension was the most likely causative mechanism. By 1991, the role of condylar resorption in postoperative relapse and instability of occlusal and skeletal results was elucidated [18].

The incidence of condylar resorption after orthognathic surgery is reported to range from 1% to 31%, depending on various nonsurgical and surgical factors [4,17,24–27]. The first radiologic signs of resorption usually present 6 or more months postoperatively [17]. Mandibular advancement, maxillary impaction, mandibular autorotation, and bimaxillary osteotomies can induce condylar resorption.

Preoperative, nonsurgical, patient-related risk factors include antecedent TMJ dysfunction, condyles with a posterior inclination, small, thin condyles, and a high mandibular plane angle with a low posterior-to-anterior facial height ratio [4,7,8,17–19,25]. Patients who have open bite are more prone to condylar resorption compared with patients who have deep bite deformities [17].

Surgical risk factors that contribute to postoperative condylar resorption are controversial [4,13,17–19,28]. Bimaxillary osteotomies performed for the correction of mandibular retrognathia with open bite present the highest frequency of condylar resorption. Kerstens and colleagues [4] reported on 12 of 206 patients with high-angle mandibular retrognathia and open bite who developed condylar resorption after bimaxillary surgical treatment. In 1994, De Clercq and colleagues [26] found 31% incidence of condylar resorption of more than 2 mm in female patients with high-angle mandibular deficiency who underwent bimaxillary osteotomies (9/29 patients). No correlation between resorption and age, the amount of retrognathism, or the presence of preoperative TMJ dysfunction was found. Hoppenreijs and colleagues [17] reported progressive condylar resorption after bimaxillary osteotomies in 27 of 117 patients who had mandibular hypoplasia and open bite.

The incidence of condylar resorption after Le Fort I osteotomy with maxillary posterior

impaction only and autorotation of the mandible was found to be 9% to 12.5% [17,24]. In these cases, it was hypothesized that biomechanical loading of the condyles caused by autorotation of the mandible was a significant contributing factor.

With regard to the magnitude of mandibular advancement, analysis of 18 orthognathic surgery patients by Huang and colleagues [1] showed that relapse occurred in patients having bimaxillary surgery with mandibular advancements more than 5 mm. On the contrary, Hoppenreijs and colleagues [17] found in their series that the magnitude of mandibular advancement had no effect on the incidence of condylar resorption.

The type of fixation used in orthognathic surgery is also a contributing factor. Bouwman and colleagues [25] investigated the role of intermaxillary fixation in postoperative condylar resorption in a group of 158 patients treated for mandibular deficiency with high mandibular plane angle. In the intermaxillary fixation group ($n = 91$), 24 patients (26.4%) showed signs of condylar resorption. In the group of 67 patients treated without intermaxillary fixation, only 8 (11.9%) of the patients showed signs of reduced volume of the condyle. Avoidance of intermaxillary fixation seems to reduce the incidence of condylar resorption after orthognathic surgery [17,25].

Cutbirth and colleagues [28] evaluated long-term condylar resorption after mandibular advancements stabilized with bicortical screws. One hundred patients with mandibular deficiency who underwent bilateral sagittal split osteotomy (BSSO) and fixation with three bicortical screws per side were followed for a minimum of 1 year. Evaluation showed that 10 of 100 patients had 10% or more vertical change in their condyles unilaterally. Large advancement and preoperative TMJ symptoms were statistically correlated with postoperative condylar resorption in this study.

In a multicenter prospective study, Borstlap and colleagues [29] reported a 2-year follow-up in a group of 200 patients who underwent BSSO and rigid fixation with two miniplates for advancement of the mandible. In 8 patients (4%), resorption of the condyles developed postoperatively. Patients treated at a relative young age (≤ 14 years) seemed to be at risk for the occurrence of condylar alterations, including resorption. A steep mandibular plane angle and a low facial height ratio were also significantly related to the occurrence of condylar change. The occurrence of pain and TMJ sounds in the first few months postoperatively was highly predictive of condylar changes occurring in the ensuing months. Of note is that both of these studies lack information regarding the presence or absence of active resorption before the operation.

Orthodontics and condylar resorption

Orthodontic treatment alone can cause condylar resorption. Kato and colleagues [30] treated a 12-year-old girl who had bilateral impacted maxillary canines and degenerative disease of the TMJ. Two years after completion of active orthodontic treatment (age 17), symptoms in her TMJ intensified. By age 21, the patient had developed an anterior open bite with a long, slender facial appearance. Cephalometric analysis showed shortening of the ramus, backward and downward rotation of the mandible, and severe deformity and resorption of the condyles bilaterally. Whether the condylar resorption was specifically caused by the orthodontic treatment cannot be determined. Peltola and colleagues [31] found 9% condylar flattening after orthodontic treatment in 625 patients.

Rheumatoid arthritis

Evidence indicates that the TMJ/mandibular condyle is affected in two thirds of all cases of the disease. In most of these cases, the condyle exhibits resorption [1,32]. Polyarticular and early-onset arthritis are associated with a significant risk of TMJ involvement and a severe loss of condylar bone [33,34].

Connective tissue or autoimmune diseases and condylar resorption

Scleroderma, systemic lupus erythematosus, Sjögren's syndrome, ankylosing spondylitis, and dermatomyositis can manifest condylar resorption [1,4,35]. Scleroderma is a chronic generalized disease of the small arterial vessels and mesenchymal tissues of unknown origin. The musculoskeletal system is often involved, and the disease may affect the mandible, causing bony erosions, osteolysis, and atrophy of the masticatory muscles. It is believed that these bony lesions are of ischemic origin. The lesions, which are usually bilateral, occur in the condyles, the coronoid processes, and the gonial angles. Ramon and colleagues [36] reported a severe case of condylar resorption in a patient with scleroderma. Mandibular involvement was present in the early stages of the disease, which had a rapid fatal course.

Haers and Sailer [35] reported a 20% to 33% incidence of condylar resorption in patients suffering from scleroderma. Women are especially affected, with a male/female ratio 1:7. Bilateral condylysis caused by scleroderma has been described in seven cases, or 13.7% of the reported cases [35]. Brennan and colleagues [37] reported the first documented case of bilateral condylar resorption in a patient who had dermatomyositis. In 1979, Lanigan and colleagues [38] reported the only case in literature of condylysis as a complication of a mixed collagen vascular disease. A 26-year-old woman who had rheumatoid arthritis, systemic lupus erythematosus, scleroderma, and Sjögren's syndrome was treated with orthognathic surgery for the mandibular retrognathism and open bite caused by the disease.

Fractures and condylar resorption

In 1991, Iizuka and colleagues [39] reported on 13 patients with high condylar fracture treated by open reduction and fixation with miniplates who were monitored for an average of 18 months postoperatively. Radiologically, signs of condylar resorption and osteoarthrosis were diagnosed in all patients. In 4 patients with associated multiple fractures of the facial bones, rapid complete resorption of the condyle was observed.

Diagnosis of condylar resorption

Condylar resorption should be suspected in any patient who presents with an acquired and progressive open bite, increasing overjet or class II

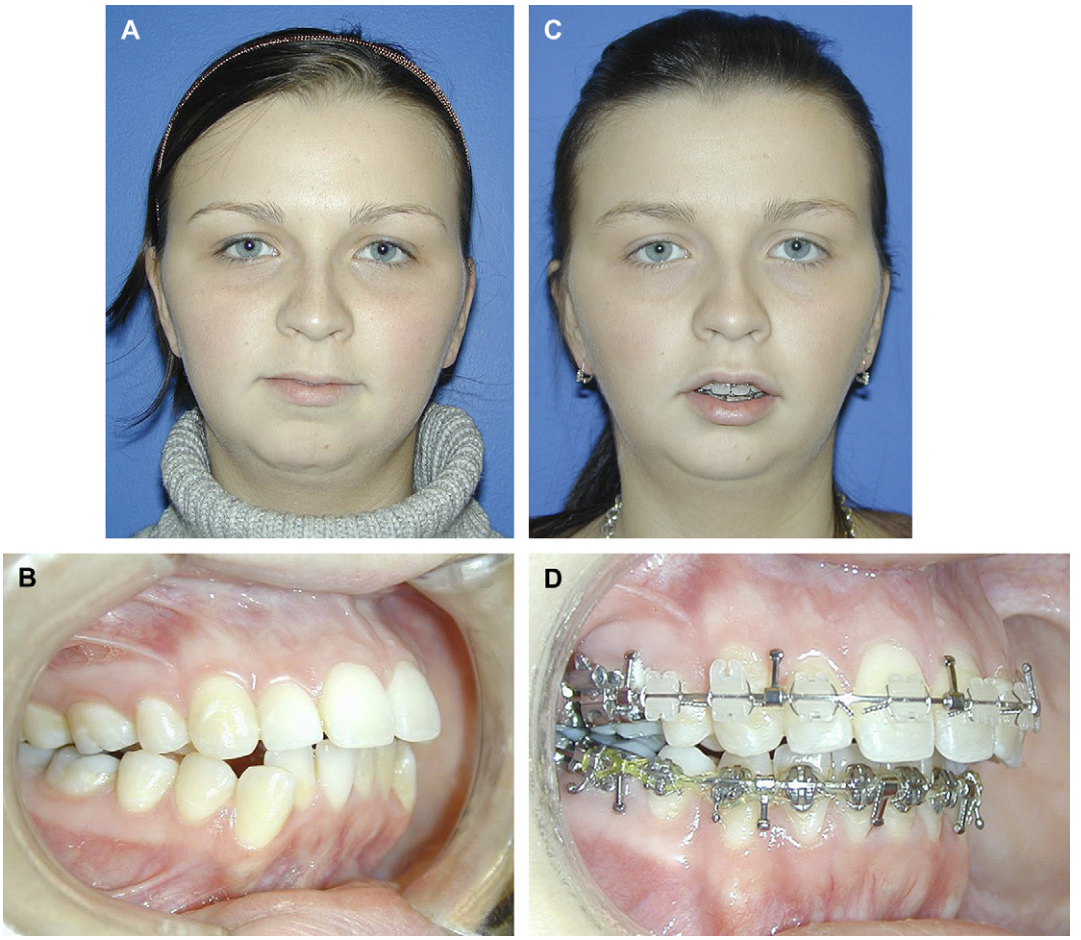

Fig. 1. Frontal (*A*) and intraoral (*B*) photographs of a 22-year-old woman with active ICR. Frontal (*C*) and intraoral (*D*) photographs of the same patient 2 years later. Note the increase in overjet.

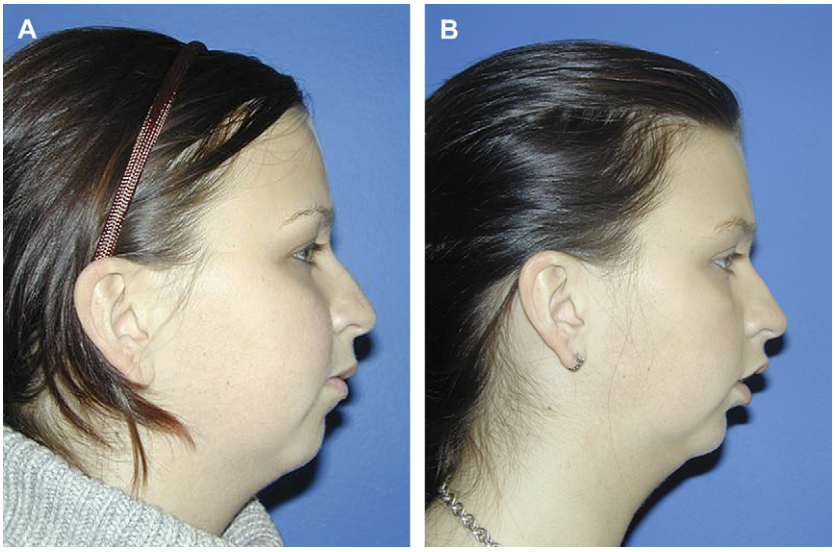

Fig. 2. Lateral photographs of the same patient at age 22 (*A*) and 24 (*B*) show the progressive mandibular retrognathia and the "disappearing chin" phenomenon.

malocclusion, mandibular retrognathia, and a "disappearing chin" (Figs. 1 and 2). Hoppenreijs and colleagues [40] distinguished between "deep bite" and "open bite" condylar resorption. Deep bite condylar resorption corresponds to resorption on the superior site of the condyle. Open bite condylar resorption develops with resorption on the superior and anterior sites of the condyle. Idiopathic condylar resorption tends to present with open bite deformities. As a consequence of condylar resorption, a decreased condylar head volume, ramus height, and growth rate occur. Limited mandibular range of motion, TMJ symptoms and pain may be present [8,9,15].

The clinician should elicit a history of previous orthodontic or orthognathic treatment, orthopedic devices, steroid use, collagen vascular and autoimmune disorders to distinguish idiopathic from secondary condylar resorption [1]. The differential diagnosis includes condylar resorption, condylar remodeling, hypoplasia and aplasia of the condyle. Evaluation of serial photographs is helpful (Fig. 2). The parents and the patient usually can estimate when the chin began to retrude or "disappear." Photographs before and after this period of time are helpful for documenting the progression. Panoramic radiographs, anteroposterior and lateral cephalograms, and dental

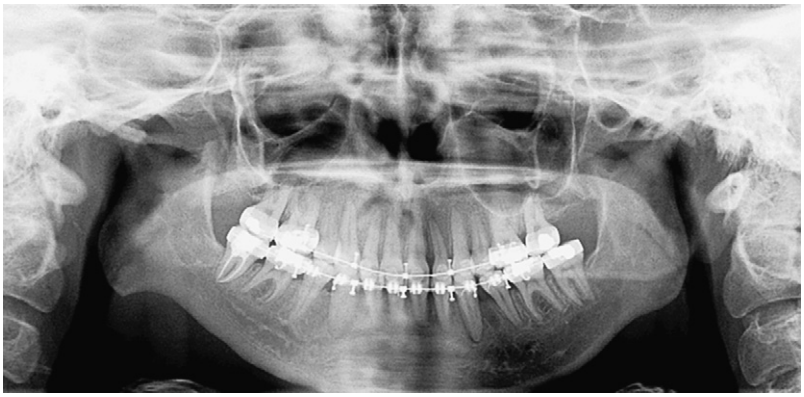

Fig. 3. Panorex of the patient demonstrates small size, short neck, and irregular surface of the condyles, reduced ramus height, and antigonial notching.

casts should be obtained. Condyles commonly exhibit small size with flat-, finger-, or spike-shaped configuration on radiographs (Fig. 3) [17]. The cortical outline of the condyle is lost in active resorption, whereas the condyle tends to become recorticated once the resorption has ceased for at least 1 year [41]. Measurement of the ramus/condyle unit length on the panorex shows shortening. Overbite, overjet, Sella-Nasion-B point (SNB) posterior face height, and mandibular plain angle are measured on the lateral cephalogram.

Technetium isotope bone scan for differentiation of active and inactive ICR is essential for diagnosis (Fig. 4) [1]. Kaban and colleagues [42] developed normal standards of technetium-99m methylene diphosphonate (99mTc-MDP) uptake in the mandible of adults and children [43]. The ratio of uptake of the condyle to the fourth lumbar vertebra (a standard bone for scanning purposes) is calculated and compared with normal standards. In patients with active condylar resorption, uptake in the abnormal condyle is more than two standard deviations greater than normal, which confirms the diagnosis. Serial bone scans are also obtained and compared in order to evaluate whether resorption has ceased. Positron emission tomography MRI is a promising method in evaluating the condyle resorption process [44].

Treatment

Treatment of ICR is controversial. Orthognathic surgery has been attempted to manage ICR. The relapse rate is high, however, if the process is active at the time of operation or if it becomes reactivated during the postoperative period. Even inactive condylar resorption can be reactivated by BSSO and Le Fort I osteotomy [17].

Arnett and Tamborello [16] treated six patients with condylar resorption with orthognathic surgery. Five of the patients had further resorption. Crawford and colleagues [45] reported on seven patients with progressive condylar resorption that developed after BSSO for mandibular advancement. Patients were treated with repeated orthognathic surgery that included BSSO. Five of the seven patients developed continued condylar resorption postoperatively. One of them underwent a third BSSO that resulted in further resorption. Merkx and Van Damme [19] treated 8 patients of a group of 329 who developed condylar resorption after sagittal split osteotomy. Four were managed with

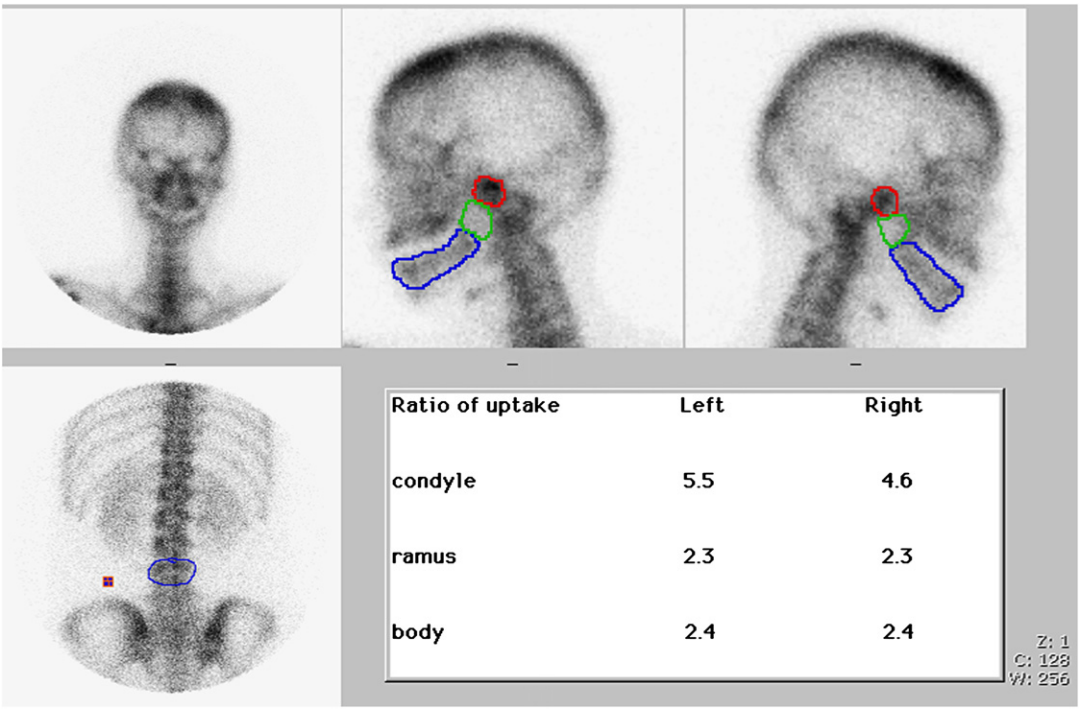

Fig. 4. Technetium scan in active resorption shows increased uptake in the abnormal condyles (more than two standard deviations greater than normal). Normal uptake ratio is less than 0.70 at age >20 years.

orthognathic surgery, and four were managed with occlusal splint, orthodontics, or prosthetic therapy or a combination thereof. Results for orthognathic surgery were unstable.

Huang and colleagues [1] reported 18 patients who underwent orthognathic surgery for the management of what was believed to be inactive condylar resorption. Four patients demonstrated condylar resorption with recurrence of open bite and retrognathism. Four patients had a stable result at the time of the study but developed TMJ symptoms. These patients were at high risk for eventual relapse. Ten patients had a stable result (no change in postoperative occlusion or jaw position) without TMJ symptoms.

Hoppenreijs and colleagues [40] evaluated retrospectively the long-term treatment results of 26 patients who developed progressive condylar resorption after a bilateral sagittal split advancement osteotomy (n = 19) or a bimaxillary osteotomy (n = 7). The patients were divided into two groups: one group received nonsurgical treatment that included splints, orthodontics with or without extractions, and restorative dentistry, and the second

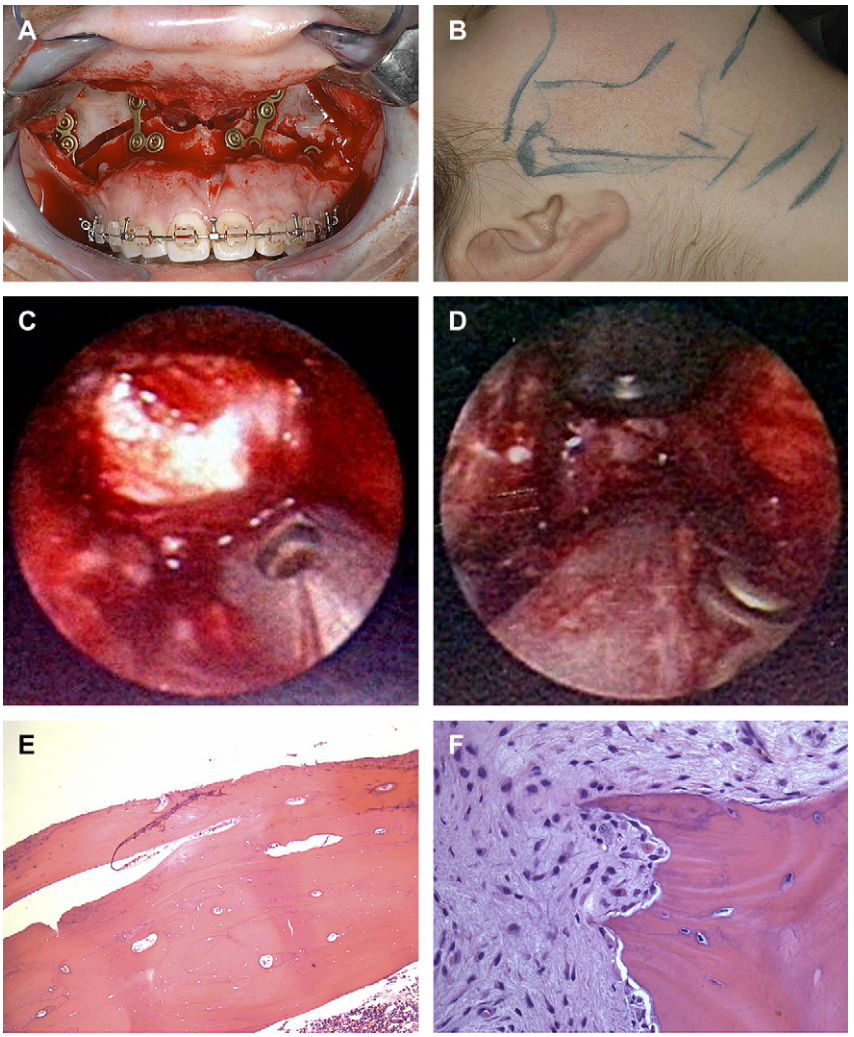

Fig. 5. This patient underwent Le Fort I osteotomy (*A*) for correction of 5° occlusal cant and (*B*) endoscopic condylectomy/reconstruction with CCG for the treatment of active ICR. (*C*) Endoscopic view of the articular disk. (*D*) Endoscopic view of the fixated graft. (*E*) Histology of the resected condyles shows the articular surface with decreased to absent articular cartilage (hematoxylin-eosin, original magnification ×100). (*F*) Focal resorption of the condylar cortical bone is present (hematoxylin-eosin, original magnification ×400). (*Courtesy of* B. Faquin, MD; Boston, MA.)

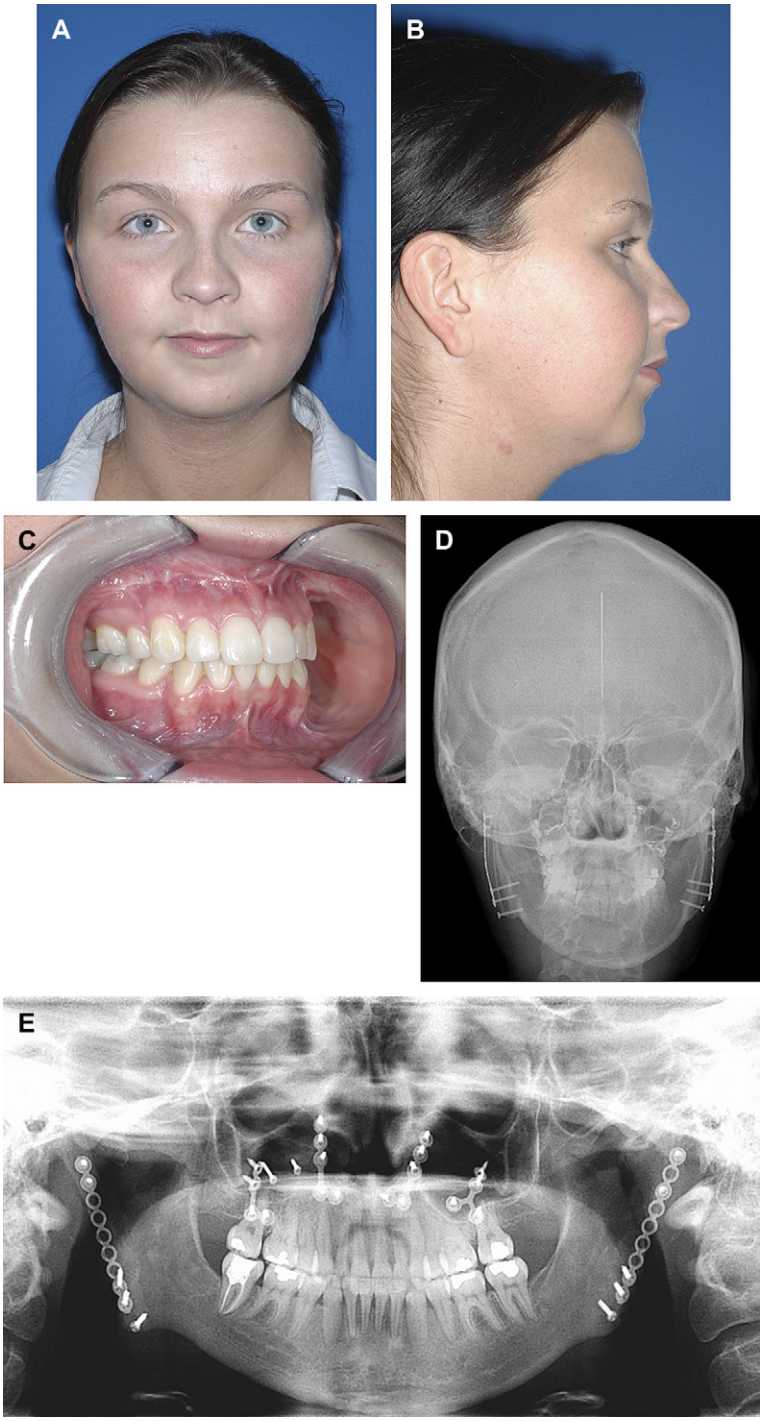

Fig. 6. Frontal (*A*), lateral (*B*), and intraoral (*C*) photographs, anteroposterior cephalogram (*D*), and panorex (*E*) of the same patient 1 year postoperatively.

group underwent repeated surgery to treat skeletal relapse. The first group had satisfactory results from orthodontic dental compensation. In the second group (n = 13), 7 patients had satisfactory occlusal and aesthetic results and were skeletally stable, 4 patients had 40% to 80% relapse, and 2 patients had 120% and 100% relapse, respectively, and needed a third surgical intervention. It seemed that without surgical intervention after condylar resorption, further resorption ceased after approximately 2 years. Second surgery seemed to produce variable results.

It seems that orthognathic surgery alone is not an ideal treatment for ICR. Reported long-term results are not stable, and resorption may proceed or be triggered if quiescent preoperatively.

Wolford and Cardenas [14] suggested an alternative treatment modality based on the hypothesis that the mediators that promote condylar resorption exist in the hypertrophic bilaminar zone of the meniscus. They proposed removal of the hyperplastic synovial tissue, repositioning and stabilization of the disc to the condyle using a mini anchor placed in the posterior aspect of the condylar head with two sutures functioning as artificial ligaments [14,15,46,47]. This procedure was followed by orthognathic surgery to correct the associated jaw and occlusal deformities. Twelve female patients with active condylar resorption were treated with this protocol. The mandible was advanced an average of 10.9 mm, and the occlusal plane angle decreased an average of -7.8°. The mean postsurgical follow-up was 33.2 months. The postsurgical condylar length change was -0.5 to $+1.5$ mm. Five patients younger than 16 years at the time of surgery exhibited a modest amount of postsurgical condylar growth, with an average increase in condylar height of $+0.43$ mm. In all 12 patients, jaw function remained unchanged, with no statistically significant difference between presurgical and postsurgical incisal openings (47 mm) or excursive movements (>7 mm).

Because mandibular orthognathic surgery entails the risk of reactivating the resorptive process, it has been suggested to perform maxillary orthognathic surgery alone. This would theoretically be accompanied by less (but not minimal) incidence of retriggering the condylar resorption [16]. This approach does not always provide favorable aesthetic results, however [40].

Condylectomy and reconstruction with CCG has also been described for the treatment of ICR (Figs. 5–7). Huang and colleagues [1] reported stable functional and aesthetic results in five patients who underwent condylectomy and CCG for the treatment of ICR. A retrospective study performed at our institution showed favorable results with this technique [48]. Fifteen patients with active bilateral idiopathic condylar resorption confirmed by clinical examination, plain radiographs, and technetium-99 bone scan were included in the study. All patients underwent condylectomy and immediate reconstruction with CCG using an endoscopic technique. Preoperative, immediate

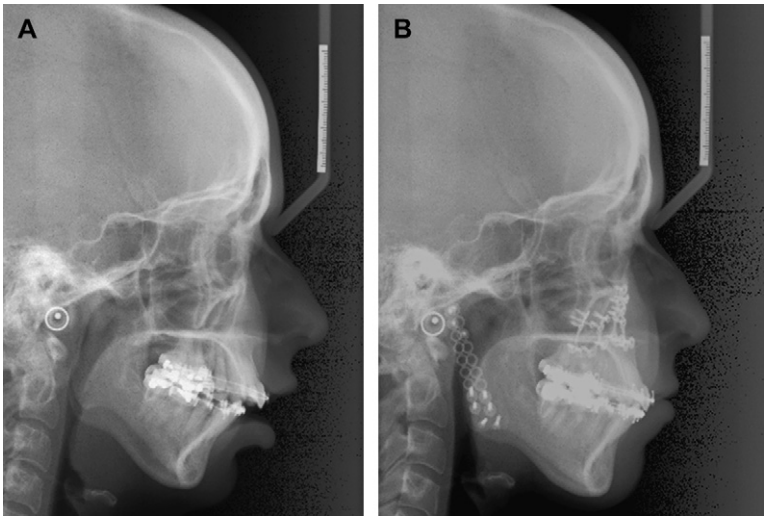

Fig. 7. Preoperative (*A*) and postoperative (*B*) lateral cephalogram of the patient in Fig. 6. Overjet and posterior face height have been corrected.

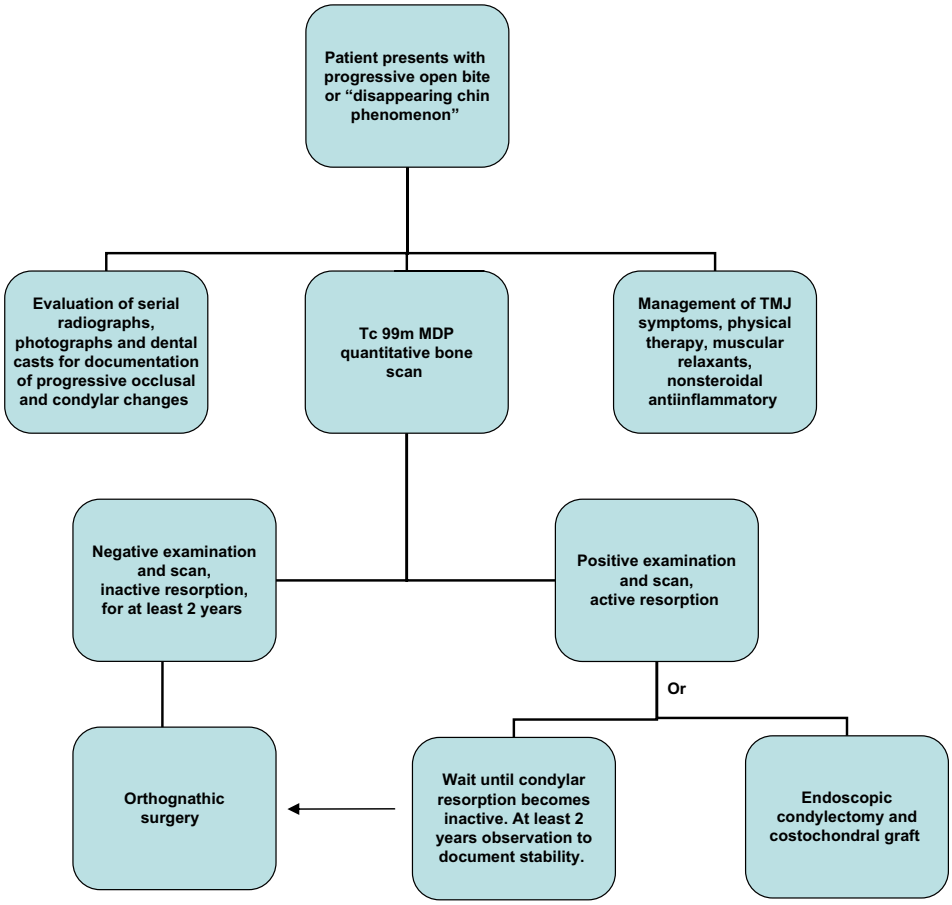

Fig. 8. Algorithm for management of ICR, MGH.

postoperative, 6-month, 1-year, and latest follow-up clinical examinations, lateral cephalograms, and panoramic radiographs were used to evaluate the outcomes. Cephalometric measurements included overbite, overjet, SNB, and mandibular plain angles. Ramus/condyle unit height was measured on panoramic radiographs. Mean follow-up was 24.2 months (range 12–72 months). Preoperatively, all patients presented with class II occlusion and an anterior open bite. Postoperatively, all patients demonstrated class I occlusion with normal overbite. All patients maintained a clinically acceptable, stable, and reproducible occlusion at the latest follow-up and showed good range of motion with no evidence of TMJ dysfunction or neurologic changes. The graft was totally incorporated in the ramus and provided new functional condyles formed in anatomic position.

Reconstruction with CCG is definitive treatment. It restores ramus height and the anterior/posterior height ratio, and the graft is not affected by the resorption.

CCG is considered the ideal donor material for reconstruction of the ramus condyle unit because it provides "a growth center" located at the costochondral junction [49]. It is used successfully for hemifacial microsomia, ankylosis, and condyle reconstruction after tumor resection. Avoidance of linear overgrowth of the graft, especially in growing patients, is achieved by maintaining a cartilaginous cap size on the graft of no more than 1 to 3 mm [50–52]. The graft should be fixed rigidly, and its length should provide a 2- to 3-mm posterior open bite that compensates for loss of vertical height (settling) of the CCG during healing and remodeling. The patient is placed in maxillomandibular fixation for 7 to 10 days with a splint maintaining the posterior open bite, and aggressive physical therapy follows. The position of the graft is regulated by the orthodontic splint

that is adjusted to close the open bite over a period of 3 to 6 months. By using an endoscopic approach to the ramus/condyle unit, condylectomy and CCG fixation are accomplished through a submandibular 1.5-cm incision [53]. This technique is minimally invasive, causes less postoperative swelling, and has a shorter course of recovery.

The Massachusetts General Hospital Protocol for management of patients with ICR is demonstrated in Fig. 8. It is essential to distinguish active from inactive condylar resorption with patient history, serial radiographs, photographs, dental casts, and technetium-99m methylene diphosphonate quantitative bone scan. TMJ symptoms must be controlled preoperatively with physical therapy, muscle relaxants, and nonsteroidal anti-inflammatory drugs. Active, progressive resorption that results in open bite and functional and aesthetic problems is treated immediately with endoscopic condylectomy and CCG. Resorption that remains inactive for at least 2 years may be treated with orthognathic surgery.

References

[1] Huang YL, Pogrel MA, Kaban LB. Diagnosis and management of condylar resorption. J Oral Maxillofac Surg 1997;55(2):114–9.

[2] Burke PH. A case of acquired unilateral mandibular condylar hypoplasia. Proc R Soc Med 1961;54: 507–10.

[3] Rabey GP. Bilateral mandibular condylysis: a morphanalytic diagnosis. Br J Oral Surg 1977;15(2):121–34.

[4] Kerstens HC, Tuinzing DB, Golding RP, et al. Condylar atrophy and osteoarthrosis after bimaxillary surgery. Oral Surg Oral Med Oral Pathol 1990; 69(3):274–80.

[5] Kaban L. Acquired abnormalities of the temporomandibular joint. In: Kaban LB, Troulis MJ, editors. Pediatric oral and maxillofacial surgery. Philadelphia: WB Saunders; 2004. p. 350.

[6] De Bont LGM, Stegenga B. Pathology of temporomandibular joint derangement and osteoarthritis. Int J Oral Maxillofac Surg 1993;22:71–4.

[7] Hwang SJ, Haers PE, Seifert B, et al. Non-surgical risk factors for condylar resorption after orthognathic surgery. J Craniomaxillofac Surg 2004; 32(2):103–11.

[8] Arnett GW, Milam SB, Gottesman L. Progressive mandibular retrusion–idiopathic condylar resorption. Part I. Am J Orthod Dentofacial Orthop 1996;110(1):8–15.

[9] Arnett GW, Milam SB, Gottesman L. Progressive mandibular retrusion–idiopathic condylar resorption. Part II. Am J Orthod Dentofacial Orthop 1996;110(2):117–27.

[10] Chuong R, Piper MA. Avascular necrosis of the mandibular condyle: pathogenesis and concepts of management. Oral Surg Oral Med Oral Pathol 1993;75(4):428–32.

[11] Chuong R, Piper MA, Boland TJ. Osteonecrosis of the mandibular condyle: pathophysiology and core decompression. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79(5):539–45.

[12] Arlet J. Nontraumatic avascular necrosis of the femoral head: past, present, and future. Clin Orthop 1992;277:12–21.

[13] Schellhas KP, Wilkes CH, Fritts HM, et al. MR of osteochondritis dissecans and avascular necrosis of the mandibular condyle. AJR Am J Roentgenol 1989;152(3):551–60.

[14] Wolford LM, Cardenas L. Idiopathic condylar resorption: diagnosis, treatment protocol, and outcomes. Am J Orthod Dentofacial Orthop 1999; 116(6):667–77.

[15] Wolford L. Idiopathic condylar resorption of the temporomandibular joint in teenage girls (cheerleaders syndrome). Proc (Bayl Univ Med Cent) 2001;14(3):246–52.

[16] Arnett GW, Tamborello JA. Progressive class II development: female idiopathic condylar resorption. Oral Maxillofac Surg Clin North Am 1990;2: 699–716.

[17] Hoppenreijs TJ, Freihofer HP, Stoelinga PJ, et al. Condylar remodelling and resorption after Le Fort I and bimaxillary osteotomies in patients with anterior open bite: a clinical and radiological study. Int J Oral Maxillofac Surg 1998;27(2):81–91.

[18] Moore KE, Gooris PJ, Stoelinga PJ. The contributing role of condylar resorption to skeletal relapse following mandibular advancement surgery: report of five cases. J Oral Maxillofac Surg 1991;49(5):448–60.

[19] Merkx MA, Van Damme PA. Condylar resorption after orthognathic surgery: evaluation of treatment in 8 patients. J Craniomaxillofac Surg 1994;22(1): 53–8.

[20] Abubaker AO, Raslan WF, Sotereanos GC. Estrogen and progesterone receptors in temporomandibular joint discs of symptomatic and asymptomatic persons: a preliminary study. J Oral Maxillofac Surg 1993;51(10):1096–100.

[21] Aufdemorte TB, Van Sickels JE, Dolwick MF, et al. Estrogen receptors in the temporomandibular joint of the baboon (*Papio cynocephalus*): an autoradiographic study. Oral Surg Oral Med Oral Pathol 1986;61(4):307–14.

[22] Yamashiro T, Takano-Yamamoto T. Differential responses of mandibular condyle and femur to oestrogen deficiency in young rats. Arch Oral Biol 1998;43(3):191–5.

[23] Phillips RM, Bell WH. Atrophy of mandibular condyles after sagittal ramus split osteotomy: report of case. J Oral Surg 1978;36(1):45–9.

[24] De Mol van Otterloo JJ, Dorenbos J, Tuinzing DB, et al. TMJ performance and behaviour in patients

more than 6 years after Le Fort I osteotomy. Br J Oral Maxillofac Surg 1993;31(2):83–6.
[25] Bouwman JP, Kerstens HC, Tuinzing DB. Condylar resorption in orthognathic surgery: the role of intermaxillary fixation. Oral Surg Oral Med Oral Pathol 1994;78(2):138–41.
[26] De Clercq CA, Neyt LF, Mommaerts MY, et al. Condylar resorption in orthognathic surgery: a retrospective study. Int J Adult Orthodon Orthognath Surg 1994;9(3):233–40.
[27] Hwang SJ, Haers PE, Sailer HF. The role of a posteriorly inclined condylar neck in condylar resorption after orthognathic surgery. J Craniomaxillofac Surg 2000;28(2):85–90.
[28] Cutbirth M, Van Sickels JE, Thrash WJ. Condylar resorption after bicortical screw fixation of mandibular advancement. J Oral Maxillofac Surg 1998; 56(2):178–82.
[29] Borstlap WA, Stoelinga PJ, Hoppenreijs TJ, et al. Stabilisation of sagittal split advancement osteotomies with miniplates: a prospective, multicentre study with two-year follow-up. Part III. Condylar remodelling and resorption. Int J Oral Maxillofac Surg 2004;33(7):649–55.
[30] Kato Y, Hiyama S, Kuroda T, et al. Condylar resorption 2 years following active orthodontic treatment: a case report. Int J Adult Orthodon Orthognath Surg 1999;14(3):243–50.
[31] Peltola JS, Nystrom M, Kononen M, et al. Radiographic structural findings in the mandibular condyles of young individuals receiving orthodontic treatment. Acta Odontol Scand 1995;53(2):85–91.
[32] Ogus H. Rheumatoid arthritis of the temporomandibular joint. Br J Oral Surg 1975;12(3):275–84.
[33] Pedersen TK, Jensen JJ, Melsen b, et al. Resorption of the temporomandibular joint according to subtypes of juvenile chronic arthritis. J Rheumatol 2001;28(9):2109–15.
[34] Svensson B, Adell R, Kopp S. Temporomandibular disorders in juvenile chronic arthritis patients. A clinical study. Swed Dent J 2000;24(3):83–92.
[35] Haers PE, Sailer HF. Mandibular resorption due to systemic sclerosis: case report of surgical correction of a secondary open bite deformity. Int J Oral Maxillofac Surg 1995;24(4):261–7.
[36] Ramon Y, Samra H, Oberman M. Mandibular condylosis and apertognathia as presenting symptoms in progressive systemic sclerosis (scleroderma): pattern of mandibular bony lesions and atrophy of masticatory muscles in PSS, presumably caused by affected muscular arteries. Oral Surg Oral Med Oral Pathol 1987;63(3):269–74.
[37] Brennan MT, Patronas NJ, Brahim JS. Bilateral condylar resorption in dermatomyositis: a case report. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87(4):446–51.
[38] Lanigan DT, Myall RW, West RA, et al. Condylysis in a patient with a mixed collagen vascular disease. Oral Surg Oral Med Oral Pathol 1979;48(3):198–204.
[39] Iizuka T, Lindqvist C, Hallikainen D, et al. Severe bone resorption and osteoarthrosis after miniplate fixation of high condylar fractures. A clinical and radiologic study of thirteen patients. Oral Surg Oral Med Oral Pathol 1991;72(4):400–7.
[40] Hoppenreijs TJ, Stoelinga PJ, Grace KL, et al. Long-term evaluation of patients with progressive condylar resorption following orthognathic surgery. Int J Oral Maxillofac Surg 1999;28(6):411–8.
[41] Pogrel AM, Chigurupati R. Management of idiopathic condylar resorption. In: Laskin DM, Greene CS, Hylander WL, editors. TMDs: an evidence-based approach to diagnosis and treatment. Hanover Park (IL): Quintessence Publishing Co, Inc; 2006. p. 533–40.
[42] Kaban LB, Cisneros GJ, Heyman S, et al. Assessment of mandibular growth by skeletal scintigraphy. J Oral Maxillofac Surg 1982;40(1):18–22.
[43] Cisneros GJ, Kaban LB. Computerized skeletal scintigraphy for assessment of mandibular asymmetry. J Oral Maxillofac Surg 1984;42(8):513–20.
[44] Gaa J, Rummeny EJ, Seemann MD. Whole-body imaging with PET/MRI. Eur J Med Res 2004;9(6): 309–12.
[45] Crawford JG, Stoelinga PJ, Blijdorp PA, et al. Stability after reoperation for progressive condylar resorption after orthognathic surgery: report of seven cases. J Oral Maxillofac Surg 1994;52(5):460–6.
[46] Wolford LM, Cottrell DA, Karras SC. Mitek mini anchor in maxillofacial surgery. In: Pelton AR, Hodgson D, Duerig T, editors. Proceedings of the first international conference on shape memory and superelastic technologies. Monterey (CA): MIAS; 1994. p. 477–82.
[47] Mehra P, Wolford LM. Use of the Mitek anchor in temporomandibular joint disc-repositioning surgery. Proc (Bayl Univ Med Cent) 2001;14:22–6.
[48] Troulis MJ, Tayebaty FT, Papadaki M, et al. Condylectomy and Costochondral Graft Reconstruction for Treatment of Active Idiopathic Condylar Resorption. J Oral Maxillofac Surg 2005;63(8):32–3.
[49] Padwa BL, Mulliken JB, Maghen A, et al. Midfacial growth after costochondral graft construction of the mandibular ramus in hemifacial microsomia. J Oral Maxillofac Surg 1998;56(2):122–7.
[50] Peltomaki T. Growth of a costochondral graft in the rat temporomandibular joint. J Oral Maxillofac Surg 1992;50:851–7.
[51] Perrott DH, Umeda H, Kaban LB. Costochondral graft construction/reconstruction of the ramus/condyle unit: long term follow-up. Int J Oral Maxillofac Surg 1994;23:321–8.
[52] Kaban LB. Temporomandibular joint reconstruction in children using CCG. J Oral Maxillofac Surg 1999;57:799–800.
[53] Troulis MJ, Williams WB, Kaban LB. Endoscopic mandibular condylectomy and reconstruction: early clinical results. J Oral Maxillofac Surg 2004;62(4): 460–5.