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Review

Idiopathic condylar resorption

K. Mitsimponas [a], S. Mehmet [b], R. Kennedy [c], K. Shakib [a,*]

[a] *Royal Free London NHS Foundation Trust*
[b] *Oral & Maxillofacial Surgery, London Deanery*
[c] *Oral Pathology, Kings College London Dental School, Guys Hospital*

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Abstract

Idiopathic condylar resorption is a well-documented but poorly-understood pathological entity that predominantly affects young women, particularly during the pubertal growth spurt. Several theories have been proposed to explain its aetiopathogenesis, the most favoured of which are the hormonally mediated theory, the theory of avascular necrosis, and the dysfunctional remodelling theory. The condition is diagnosed by a combination of clinical and radiological data as well as elements from the patient's history. Treatments such as orthognathic surgery, repositioning and stabilisation of the disc, condylectomy and condylar repair with a costochondral graft, or total prosthetic joint reconstruction, have been suggested, but so far, no method has proved superior. Further research is required to better understand the pathophysiology of the condition and identify the optimal treatment.

Keywords: Idiopathic condylar resorption; condylysis; condylar hypoplasia; inflammatory arthropathy

Introduction

Conditions that result in a small or abnormally-shaped mandibular condyle can be broadly divided into two groups depending on whether the condyle does or does not reach a normal size and shape before the resorptive process begins. The condyle might fail to grow fully because of congenital underdevelopment or as a result of incidents that arrest its development.

Condylar hypoplasia, which can be defined as congenital underdevelopment of the mandibular condyle, is often part of generalised mandibular hypoplasia in syndromes such as hemifacial macrosomia, mandibuloacral dysplasia, and oculoauriculovertebral syndrome. Condylar growth stops most commonly as a result of trauma to the growing mandible, and infection, radiation, and inflammatory arthropathies that affect it during growth can cause mandibular hypoplasia.[1] In 1961, Burke's report of a case of arrested condylar growth that had resulted from trauma at a young age,[2] underlined the importance of the centres of condylar growth for the symmetrical development of the mandible.

Condylar resorption differs from hypoplasia and arrested growth, as the condyle forms normally but then resorbs. Rabey, who first reported this distinction in 1977,[3] talked about "condylysis" as opposed to underdevelopment of the mandibular condyle. The condition is characterised by a loss of bone at the mandibular condyle with a reduction in bony mass and progressive alteration of condylar shape.[2] It often reduces the height of the posterior face, and leads to malocclusion, dysfunction of the temporomandibular joint (TMJ), and pain.[2,4,5]

Progressive condylar resorption, which is a general term to describe a condition that results in loss of condylar height,

* Corresponding author.
E-mail addresses: konstantinos.mitsimponas@nhs.net (K. Mitsimponas), suziye.mehmet1@nhs.net (S. Mehmet), robertkennedy@nhs.net (R. Kennedy), k.shakib@nhs.net (K. Shakib).

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can occur with a variety of underlying factors that are known to be associated with resorption. These include rheumatoid arthritis, systemic lupus erythematosus, use of steroids, trauma, neoplasia, orthodontic treatment, and orthognathic surgery.[2–15] As the name emphasises, it is progressive.

When there is no obvious underlying cause, the term idiopathic condylar resorption is used. This was introduced by Crawford et al[14] to describe resorption in a case series of seven patients. The condition is well known to have a predilection for young women aged between 15 and 35 years, and is most common in teenage girls during the pubertal growth spurt.[16] These patients tend to have high angles of the mandibular plane, Class II skeletal relations, and pre-existing dysfunction of the TMJ.[17,18] The condition is rare in patients with low angles of the mandibular plane or Class III skeletal relations.[4,5,16]

Aetiology

All local conditions (such as osteoarthritis, reactive arthritis, infection, traumatic injury) and systemic disorders (such as rheumatoid arthritis, scleroderma, systemic lupus erythematosus, and psoriatic arthritis) that can cause progressive condylar resorption must have been excluded before a diagnosis of idiopathic condylar resorption can be made.

The exact aetiological mechanism and pathogenesis are not yet fully understood, and there are three main aetiological theories: sex hormone-induced necrosis, avascular necrosis caused by pathological compression of the condyle, and resorption caused by loss of the capacity to remodel.[4,5,16]

Hormonally-mediated theory

Idiopathic condylar resorption has a strong predilection for teenage girls (hence the term "cheerleader syndrome") and tends to occur during the pubertal growth phase. In women, oestrogen mediates the metabolism of cartilage and bone, and oestrogen receptors have been identified in the TMJ of patients with TMJ symptoms such as pain, headache, myofascial pain, clicking, and crepitus.[19–21] An increase in oestrogen receptors is thought to lead to an exaggerated response to loading of the joint, which causes synovial hyperplasia and results in condylar resorption. The breakdown of ligamentous structures allows the disc to become anteriorly displaced.

Avascular necrosis theory

Advocates of this theory suggest that pathological compressive forces on the posterior aspect of the condyle (on the ligamentous retrodiscal soft tissues) lead small vessels to constrict, which impairs the circulation to the condyle and results in aseptic necrosis.[22–24] Some think that a chronically dislocated, non-reducing disc or the presence of malocclusion can trigger this vicious circle of events.[16]

Dysfunctional remodelling theory

Arnett et al,[10,11] who were among the first to describe the condition,[4,25] proposed the concept of "dysfunctional remodelling" of the condyle to explain it. They suggested that this could result from increased mechanical stress or reduced capacity of the host for remodelling. Three categories can affect this process: age, and systemic and hormonal factors.[4,10,11]

Pathogenesis

Bone is a dynamic tissue that is renewed by a process of resorption and formation. Osteoblasts produce osteoid, the organic matrix of bone, and then initiate and regulate its calcification. Osteoclasts, derived from the monocyte/macrophage lineage, are the terminally-differentiated and multinucleated cells that are responsible for bony resorption.

The relative activity of osteoblasts and osteoclasts decides the rate of bony resorption or deposition. This is influenced by hormones, cytokines, mechanical stress, infection, and other factors that activate or inhibit a number of biochemical pathways. The RANK/OPG system is the principal regulator of bony resorption/formation. RANK (receptor activator for nuclear factor kappa beta) is a member of the tumour necrosis factor (TNF) family of receptors that are expressed mainly on cells of macrophage/monotypic lineage, such as preosteoclasts. Binding of the RANK ligand (RANKL) to its receptor RANK induces osteoclastogenesis and bony resorption.[26]

Osteoprotegerin (OPG), which is also a member of the TNF receptor superfamily, acts as a decoy receptor for RANKL, and therefore inhibits bony resorption.[27] These key regulators are implicated in the pathogenesis of bony resorption in arthritis,[26] periodontitis,[28] hyperparathyroidism, and other metabolic and inflammatory diseases, although these findings are largely based on in vitro studies.

As in other systems, RANKL/OPG probably has a key role in the regulation of condylar resorption. However, the mandibular condyle has a number of unique developmental, histological, and functional features that should also be considered. Unlike other synovial joints, it initially develops through intramembranous, rather than endochondreal, ossification. After this, three secondary cartilages develop. The condylar cartilage is the largest and, because of its proliferation and ossification, is thought to serve as a growth centre for the mandible. Intramembranous development of the condyle results in the coverage of its articular cartilage by fibrous tissue rather than by hyaline cartilage, as is the case with the articular surface of bones formed by endochondreal ossification. Beneath the fibrous layer there is a cellular layer (the proliferation of which replenishes the adjacent layer) then another fibrous layer and finally, a thin zone of calcified cartilage that is the remnant of the secondary condylar cartilage.[29] The unique function of the condyle is also likely to contribute to the resorptive processes that affect it. Although idiopathic

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resorption of hard tissues is rare, it is well-known to occur in the roots of teeth[30] (parts of the anatomy that are close to the condyle).

Despite different initial pathways and aetiological factors, in all cases of condylar resorption, a common, cytokine-mediated pathway is thought to be responsible for the loss of bone. Osteoblasts are activated by cytokines that promote the recruitment and activity of osteoclasts. Osteoclasts then secrete matrix metalloproteinases (MMP), which breakdown hydroxyapatite and collagen, resulting in articular bony resorption.[31]

Cytokines

Cytokines are involved in cell signalling and may be produced by several different cell types within the TMJ, including osteoblasts and synoviocytes. Several cytokines have been implicated in the pathogenesis of condylar resorption. These include tumour necrosis factor alpha (TNF-α), interleukin-6 (IL-6), and RANKL.[32–38] Studies have shown correlations between concentrations of these cytokines and severity of disease in humans with diseases of the TMJ.[39–41]

Matrix metalloproteinases (MMP)

MMP are enzymes that require zinc as a cofactor. They can degrade various extracellular matrix molecules such as collagen and elastin, which are present within the articular tissues of the TMJ, and they have an important role in the degradation of bone and cartilage in the joint. Their production by cells, including osteoclasts, may be induced by cytokines.[42–44] The activity of MMP is regulated by tissue inhibitors of metalloproteinases (TIMP), which bind to MMP, and are active and inhibit them. In some cases of condylar resorption, an imbalance between the activities of TIMP and MMP has led to the unregulated degradation of tissue.[45–47]

Predisposing factors[48]

Female sex

The female:male ratio is roughly 9:1.

Young age

Patients are affected between the ages of 10 and 40 years, but the greatest incidence is during pubertal growth.

High-angle facial types

Patients often have high angles of the occlusal plane and mandibular plane (dolichocephalic facial types). The condition is rare in low-angle (brachycephalic) facial types.

Class II skeletal and occlusal relations

Patients may or may not have an associated anterior open bite with their Class II skeletal or occlusal anomalies. Idiopathic condylar resorption is rare in those with Class III skeletal patterns.

Clinical features[4,5,48]

Diagnosis is based on the patient's history, clinical and radiological findings, and after exclusion of local and systemic disorders that can cause progressive disease.

Patients may report a worsening of their occlusion and aesthetics with or without TMJ symptoms and associated pain. The condition can progress and affect the entire condyle, but typically spares the ramus of the mandible below the sigmoid notch.[5] Condylar involvement is usually bilateral and symmetrical, but unilateral cases also occur.

Bilateral condylar involvement

Bilateral condylar involvement usually results in a symmetrical posterior shift of the mandible (worsening Class II relation), condylar flattening, reduction in condylar height, loss of posterior facial height, and development of an anterior open bite. The mandible appears to rotate clockwise, which creates a "bird-face" deformity.

Unilateral condylar involvement

Unilateral condylar involvement usually results in a shift of the dental midline and point of the chin towards the affected side, an ipsilateral Class II malocclusion, cross-bite, and premature occlusal contacts (with resultant lateral open bite on the contralateral side).

Progressive condylar resorption is followed by stabilisation without further loss of condylar height, and function of the TMJ is usually good with minimal or no symptoms. Wolford and Cardenas reported that 25% of patients in their study had no TMJ symptoms (Wolford LM, et al. Evaluation of a new treatment protocol for idiopathic condylar resorption. Presented at AAOMS 77th Annual Meeting and Scientific Sessions. Toronto, Canada, 1995). There is often an absence of clicking and popping because the hyperplastic synovial tissues enlarge the joint space and provide a smooth pathway for translation of the condyle on to the displaced disc.

Imaging[5,6,25,48,49]

Traditionally, diagnosis has depended on conventional two-dimensional images such as orthopantograms and lateral cephalograms that show loss of condylar volume, but magnetic resonance imaging (MRI) is also useful, particularly to

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assess the articular disc. Bone scans can monitor the activity of the disease (technetium-99 m).

Lateral cephalograms[5,6,25,48,49]

Findings depend on whether condylar involvement is bilateral or unilateral, and serial images may show progression. Patients with unilateral condylar involvement may have unilateral skeletal and occlusal Class II relations; differences in vertical height at the inferior border of the mandible, ramus, and occlusal plane; and a lateral open bite on the contralateral side. Those with bilateral involvement often have Class II skeletal and occlusal relations, anterior open bite, high angles of the mandibular plane and mandibular occlusal plane, reduced posterior facial height, increased proclination of the lower incisors and, in severe cases, restriction of the oropharyngeal airway.

Computed tomography (CT) and cone-beam CT

CT and cone-beam CT can be valuable in the study of the condylar morphology.[50–52] CT can reliably diagnose the status of the hard tissue of the joint and help in the identification and study of features such as the joint space, presence of sclerosis, flattening of the margins, erosion of the cortical plate, and the presence of osteophytes.[51] Cone-beam CT can be particularly useful in the study of condylar resorption, as it enables clinicians to identify areas of resorption and to quantify the amount of bone lost. As these methods of imaging enable an accurate estimation of the volume of the condylar head, both can be used to monitor the progress of resorption.[50,52]

MRI[4,5]

MRI can show deformation and degenerative changes in the disc, the extent of which will depend on the duration of its displacement. Other findings include reductions in the size and volume of the condyle, anterior displacement of the disc (with or without a reduction in opening), thinning or loss of continuity of condylar cortical bone, and the presence of thickened soft tissue occupying the space between the condyle and fossa. The residual condyle should be assessed to find out whether it can withstand normal functional loading.

Although imaging findings have been widely reported, a recent systematic review showed that the characteristic radiographic features of this disease entity have not yet been described in detail.[4]

Technetium-99 scans

It can be difficult to interpret bone scans in these patients, as active disease and post-resorptive remodelling increase uptake. To plan treatment, it is necessary to distinguish between the two forms of condylar resorption, and to this end, several attempts have been made to use nuclear medicine studies.[5,50,51]

Kaban et al tried to develop normal standards for ^{99m}Tc-MDP (technetium medronic acid) values in children and adults.[5,53,54] They proposed a comparison of relative uptake between the two condyles (uptake of the fourth lumbar vertebra was used as the baseline value), and stated that more than a two-fold increase in uptake points to a diagnosis of active condylar resorption.

Single proton-emission computed tomography (SPECT) allows for more accurate and easier quantitative analysis of bone compared with planar scans,[55,56] and enables calculation of percentile differences in the uptake of technetium between the two condyles. Further studies have focused on the identification of normal values.[57] New fusion technologies that combine nuclear medicine with other methods of imaging, such as SPECT/CT and PET/MRI, look promising.[5,58,59]

Management[4–6,25,48]

Treatments range from conservative management with occlusal splints to orthodontic or restorative treatment, or both; orthognathic surgery to invasive operations on the disc or condyle; prosthetic joint replacement, or a combination of procedures. Definitive treatments that alter the occlusion should be delayed until there is sufficient evidence that resorption has ceased. Management, however, remains controversial. Successful treatment is defined by optimal aesthetic and functional outcomes together with long-term stability of results.

Occlusal splint therapy

This may be started as soon as idiopathic condylar resorption has been diagnosed, and it aims to "unload" the condyles to relieve discomfort and muscular pain. Splints may help prevent progression (although we have found little evidence to support this) and may be used with or without concurrent orthodontic treatment. A systematic review by Sansare et al,[4] however, included two studies[60,61] (combined total n = 17) that reported no relapses over a period of 24–27 months.

Wolford and Cardenas protocol[48]

If the disc can be salvaged, the authors recommended excision of hyperplastic synovial tissue from the joint, repositioning of the articular disc, and stabilisation with a Mitek mini anchor (DePuy Synthes) in the posterior aspect of the condylar head. This is attached to two 0-Ethibond sutures (Ethicon) that function as artificial ligaments. Associated jaw and occlusal abnormalities can then be corrected with orthognathic surgery, which eliminates the TMJ disease and corrects the functional and aesthetic dentofacial defects. Orthognathic surgery alone, however, is not recommended because of the

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potential for further condylar resorption and redevelopment of these deformities.

When the disc cannot be salvaged but adequate condyle remains, hyperplastic synovial tissue should be removed and the disc replaced with autogenous tissue. In severe cases when the condyle cannot be salvaged, it may have to be replaced with an autogenous graft or total prosthetic replacement.

No relapse was reported over a period of 18 months to 12 years.[48] Success of this protocol does, however, depend on early detection and the presence of adequate condylar bone with which to stabilise the disc.

Condylectomy with costochondral graft

This technique has been reported as successful with no reports of relapse beyond 12 months.[62] It is a valuable treatment when there is extensive resorption (with a considerable reduction in ramus height) and when salvage of the disc is not possible.[4,5] Papadaki et al pointed out that the ability of a costochondral graft to act as a natural growth centre in place of the removed condyle was an additional benefit.[5] However, some authors have reported excessive growth of the graft material with resultant mandibular asymmetry or ankylosis.[63–66]

Several surgeons have been concerned that grafts of autologous tissue to reconstruct the TMJ are subjected to the same pathophysiological mechanisms that caused the initial resorption, increasing the possibility of recurrence. Although this seems logical, it is not backed by evidence.

In their systematic review, Sansare et al recognised the lack of studies with long-term follow up for condylectomy or costochondral grafts. These are essential if we are to ascertain the long-term success of this treatment.[4]

Orthognathic surgery

Orthognathic surgery has a high rate of relapse compared with other treatments, and Sansare et al,[4] found that it had the highest relapse rate (83.3%–100% over a follow-up period of 1–39 months).[59,60] Sometimes it can also exacerbate further condylar resorption.[14,60,66] Huang et al[6] reported that only 10 of 18 patients treated by orthognathic surgery were asymptomatic and stable postoperatively. The remainder had TMJ symptoms (n = 4) or continued condylar resorption (n = 4). Troulis et al[62] attributed part of the increased relapse rate to the poor selection of patients, and highlighted the importance of distinguishing between active and inactive disease. Orthodontic and orthognathic treatment of patients with active disease seems to be more likely to result in relapse, but stabilisation of the TMJ with appliances, and anti-inflammatory medication preoperatively may produce more stable results (as suggested by Arnett and Tamborello in their proposed treatment protocol).[66]

Preoperative condylar stability may be established by clinical examination and series of radiographs or bone scans, as proposed by Cisneros and Kaban.[53,54] Papadaki et al advised the use of orthognathic treatment only when the disease has been inactive for at least two years.[5]

Maxillary surgery alone to alter the occlusal plane may produce stable results in carefully selected cases,[66,67] but the aesthetic results are considered less favourable.[5,61]

Bimaxillary surgery with counter-clockwise rotation of the maxillomandibular complex, which will be stable if the remaining condyle is adequate, is thought to restore posterior facial height and facilitate greater mandibular advancement. However, it may worsen TMJ symptoms and predispose to further resorption because of increased biomechanical loading on the joint. Patients with mandibular movement of more than 8 mm have the highest risk of relapse.[68] Mandibular sagittal split osteotomy may independently be associated with a risk of condylar resorption, and its use alone to treat idiopathic resorption should be considered with extreme caution.[4,5]

The use of distraction osteogenesis is controversial. Schendel et al made the case for its use when extreme advancement of the mandible is required, so a high risk of relapse is to be expected.[69,70] Posnick and Fantuzzo, on the other hand, noted that distraction osteogenesis limited the area of intervention in the mandible and tended to result in aesthetically suboptimal results.[16]

Condylectomy and total prosthetic joint reconstruction

Reconstruction of the TMJ with an alloplastic prosthesis has been widely used for many years to treat a variety of conditions. Mercuri tried to illustrate the logic behind its use by stressing that it can neutralise the dysfunctional remodelling of the joint.[71] Dysfunctional remodelling is one of the key biological factors in Arnett's pathophysiological model of idiopathic condylar resorption, referenced in a paper by Wolford et al.[72]

Alloplastic joint prostheses can overcome the limitations posed by the biological and mechanical adaptability of the TMJ.[4,71] Another advantage of reconstruction is that the maxillomandibular complex can be rotated counter-clockwise with impunity.[73] Counter-clockwise rotation is desired in patients with idiopathic resorption, as it can restore the lost posterior facial height and facilitate advancement of the mandible at point B and pogonion. However, despite having beneficial effects on aesthetics, it can aggravate TMJ disorders, and result in unstable surgical outcomes.[73–76]

Use of an alloplastic joint can have the benefits of counter-clockwise rotation and minimise the risks.[73] We know of few studies that have presented long-term data regarding clinical outcomes for patients fitted with TMJ prostheses for idiopathic resorption.[4] In their study of 21 patients with a mean (range) follow up of 6.2 (5–12) years, Mehra et al[73] reported favourable long-term results with excellent stability of movement, and a reduction in TMJ symptoms. However, despite reports of good results with low relapse rates in this and other studies, only small numbers of cases have been treated.

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Timing of operation

There is no clear consensus about the optimum time to operate.[4]

In conclusion, idiopathic condylar resorption is well documented but poorly understood, particularly as far as its aetiology is concerned. A lack of clear understanding of its pathogenesis does not allow for the development of an aetiological treatment, and further studies of its aetiopathogenesis are therefore required. Sansare et al[4] showed that there is a low level of evidence for current management, and well-planned and well-documented prospective trials are now needed to enable the development of a universally-accepted treatment.

Ethics statement/confirmation of patients' permission

Not applicable.

Conflict of interest

We have no conflicts of interest.

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