Degenerative Disease of the Temporomandibular Joint

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Progression of degenerative joint disease is dependent on the underlying pathologic and/or reactive processes involved that, in general, compromise tissue adaptability. A review of clinical and experimental literature relating to degenerative joint disease is presented. Epidemiology, pathogenesis, diagnosis, treatment, and prognosis are described with particular emphasis given to the temporomandibular joint. This article describes factors affecting the temporomandibular joint remodeling/degeneration parity and presents rationale for approaches to diagnosis and treatment.
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Breakdown of temporomandibular joint (TMJ) tissues, and articular surfaces in general, may occur due to an increased mechanical stress and/or a reduced ability for the tissues to adapt to applied stress. Local and systemic factors have been identified in the etiology, progression, and ultimate quiescence of degenerative joint disease (DJD). The purpose of this article is to describe factors affecting the TMJ remodeling/degeneration parity and, in doing so, present rationale for diagnosis and treatment.

Temporomandibular disorders (TMD) generally represent a small group of separate musculoskeletal disorders[1] that are distinct but related.[2–4] Six types of TMD specific to the TMJ proper have been recognized.[4] The TMJ disorders may be divided into specific articular disorders related to *(1)* deviation in form; *(2)* disc displacement with or without reduction; *(3)* dislocation; *(4)* inflammatory conditions including synovitis and capsulitis; *(5)* arthritides including osteoarthrosis, osteoarthritis, and polyarthritis; and *(6)* ankylosis.[4,5] Specific TMJ disorders can contribute, in varying degrees, to an overall TMD.[6]

Degenerative joint disease is a descriptive term, often used as a diagnosis, that fails to identify a specific etiology. Various musculoskeletal disease and reactive processes[7–13] have been commonly implicated in the progression of DJD. Diagnoses of DJD reflect disease processes of tissue deterioration in which soft tissue, cartilage, and bone are converted into or replaced by tissue of inferior quality.[4] Generally, DJD appears to be the manifestation of an imbalance between an adaptive response (remodeling) and a nonadaptive response (degeneration).

Epidemiology

Literature relating to epidemiology of degenerative disease of the TMJ is descriptive and retrospective. Schiffman et al[14] conclude that approximately 7% of the general population may benefit from treatment for TMJ problems and that most symptomatic subjects can function adequately without treatment. Of patients treated at temporomandibular dysfunction clinics, 8% to 12% receive diagnoses of DJD.[15–18] Autopsy results confirm TMJ degenerative disease prevalence that varies from 22% to greater than 40% of the population,[19–21] with osteoarthritis of the TMJ commonly appearing asymptomatically.[22] Data support the concept that aging women seem to be more prone to DJD than are men.[15,16,19,23]

Pathogenesis

Role of Aging

There is a general association between the incidence of DJD and increasing age. Studies indicate that the frequency of DJD increases in older persons,[19–21,24–27] suggesting that age is a predisposing factor. The incidence of tooth loss and osteoarthritis is associated with increasing age,[4,28–31] but when age is controlled, the association is coincidental.[4,32] Attrition has also, independently of age,[33] been associated with TMJ osteoarthritis despite its questionable etiologic role.[34] Although age may be correlated with observations of temporomandibular DJD, the possible correlation does not elucidate etiology.

Age-related changes in articular tissues have been documented. The fibrous component of the matrix of aged articular tissue of the TMJ consists of a well-organized collagen network occasionally dispersed with elastic fibers.[35] The overall amount of collagen does not significantly decrease with aging; however, infrastructural alterations occur. Proteoglycan content decreases and binding quality is altered. In mice, TMJ cellularity generally decreases with aging, resulting in loss of proliferative zone potential.[36] Such age-related changes in the articular tissues affect their mechanical properties and, in turn, may facilitate the pathogenesis of DJD.

Joint Loading and Stress Distribution

Strong evidence exists to suggest the TMJ is loaded during function.[37–45] Intensity and direction of stresses generated by various occlusal forces have been analyzed utilizing three-dimensional mathematical[43,46,47] and finite element–analysis models.[44,48] Each TMJ is a pressure-bearing, compound, double-synovial joint.[49]

Repetitive cyclic microtrauma has been implicated in the etiology of DJD. Repeated impact loading of cartilage-lined articular surfaces results in an increased rate of osteogenesis in subchondral bone evidenced radiographically as subchondral sclerosis.[50] Affected bone, of increased stiffness, may increase the susceptibility of articular cartilage to trauma caused by impact,[51] with loss or degeneration of cartilage normally resistant to compressive stress. In the TMJ, reducing disc displacements may potentiate repetitive impact loading[52] and consequently lead to a subchondral sclerosing known to occur prior to the onset of DJD.[53] Whether or not DJD can be attributed to the cumulative effect of repetitive minor trauma to normal TMJ articular tissue remains debatable.

Major trauma to synovial joints may progress to DJD.[54] Excessive joint loading may disrupt the normal adaptive capacity of articular tissue, resulting in eventual cartilage breakdown and elevated proteoglycan levels in the synovial fluid.[55,56] Kopp et al[57] conclude, however, that degenerative changes described in their autopsy material are probably mostly due to local factors. While synovial fluid aspirates of joints that show arthroscopic evidence of DJD contain elevated levels of keratin sulfate,[56] histochemical studies of articular surfaces that macroscopically demonstrate DJD have shown a reduction in sulfated glycosaminoglycan.[25]

Arthritic lesions of the TMJ are most often localized in the lateral aspect of the temporal fossa, compared to the medial aspect.[19,24,58] Lesions are markedly fewer in the condyle than in the temporal component. In a study of location of osteoarthritic lesions in patellofemoral compartments of 39 cadaveric knee joints,[59] it was hypothesized that articular cartilage adapts mechanically to transmit, without sustaining damage, the stress to which it is most regularly subjected, and that damage occurs only if cartilage is subjected to less frequent but much higher stress.[60] Articular tissue thickness and distribution probably reflect areas of stress concentration in the condylar and temporal components,[61] with the lateral part of the joint exposed to the largest functional and parafunctional loads.[62] Incongruities between loaded TMJ articular surfaces predispose to stress concentrations that are observed more frequently in the temporal component where degenerative lesions more commonly occur.[63]

Adequate lubrication of the TMJ components is required to facilitate the mechanics of joint mo-

tion. The upper joint compartment is subject to translatory movement while the lower undergoes rotational movement.[64] Differences in the frequency of degenerative lesions between the condylar and temporal components may be due to greater frictional loading between the disc and temporal components than between the condyle and disc. Nitzan and Dolwick[65] suggest that a lack of gliding in the joint can be attributed to disc adherence to the fossa by a reversible effect such as vacuum and/or decreased volume of high-viscosity synovial fluid. Decreased synthesis of the glycoprotein lubricin, normally associated with the lubrication fraction of synovial fluid, may be involved in the etiology of DJD.[55] Lack of joint lubrication may exacerbate articular tissue failure by increasing frictional resistance during loaded joint movements.

Internal Derangement

Internal derangement of the TMJ, particularly with disc deformation, may progress to DJD. Position and configuration of the articular disc has been related to DJD.[66,67] Westesson[68] conducted a radiographic study including arthrograms on 128 patients with internal derangements. Osseous changes were seen in 50% of the patients with anterior disc displacement *without* reduction but seldom in patients with disc displacement *with* reduction. Degenerative changes were consistently found in joints with disc perforation. Anderson and Katzberg[69] obtained comparable findings with a tomographic and arthrographic study of 141 patients with temporomandibular dysfunction. Only 9% of the patients with a reducing displacement showed signs of degeneration, but 39% of the patients with nonreducing disc displacement and 60% of the patients with perforation had degenerative changes. These authors[68,69] conclude that, in many cases, internal derangement of the TMJ precedes degenerative disease.

There is a relationship between disc perforation and degenerative disease of the TMJ.[70–72] Surgical creation of bilateral disc perforation in Macaca fascicularis monkeys produces pathologic alterations consistent with DJD in the majority of experimental joints.[73,74] In humans, observed perforations localized to the posterior attachment of the disc[71] suggest the attachment does not have the same resistance to compressive loading as does the disc itself.[75] Despite the relationship between disc displacement and disc perforation,[23,76,77] the concept that DJD is the result of displacement and perforation of the disc is disputed.[78,79] In a blind study of joints with normally positioned discs, displaced but reducing discs, and displaced nonreducing discs, perforations were found only in cases with DJD.[80] However, 73% of the cases with DJD did not have perforations. These observations suggest it is more likely that DJD is the cause and perforation is the effect.

Schellhas[81] regards internal derangement of the TMJ as an irreversible and generally progressive disorder that may be staged clinically. Stegenga et al[82] hypothesized that TM joints are subject to a continual process of articular surface breakdown and repair. If the degradative response exceeds the reparative response, then a DJD process is initiated. The gliding capacity of the articular disc may become impaired, thus predisposing to internal derangement. Stegenga et al[82] suggested that internal derangement is an accompanying sign of DJD and is capable of causing a rapid progression of the disorder. Stegenga et al[82] concluded that in many cases of temporomandibular dysfunction, DJD is the primary disorder, and that the accompanying muscle pain and internal derangement is secondary.[83]

Inflammatory/Histochemical Considerations

Histologic studies of synovium of degenerative joints show a significant amount of inflammation[84–87] and proteoglycan turnover.[88–90] In appendicular joints with cartilage articular surfaces, early stages of DJD show fibrillation and fissures at the articular surface level that will eventually progress to a complete erosion of the cartilage.[91] Pathologic changes appear to be related to increased levels of metalloproteinases that produce both a breakdown of the collagen network[92–94] and a size reduction of the proteoglycan monomer.[95–98] The TMJ articular surfaces are covered with fibrous connective tissue, as opposed to hyaline cartilage, but research suggests[84,85,95] that inflammatory changes in articular matrix components are *(1)* responsible for impairment of the physicochemical properties of the articular surfaces and *(2)* likely to contribute to the development of DJD.

In addition to mediators of inflammation,[99] other biochemical mediators have been identified with DJD and osteoarthrosis. The cytokines interleukin-1 (IL-1) and interleukin-6 (IL-6) mediate chondrocyte protease production causing cartilage destruction in DJD.[100–105] Proteases such as collagenaselike (CL) peptidase[106–108] and prolyl endopeptidase (PEP)[109–112] increase in serum concentration in mice inbred for osteoarthrosis of the TMJ.[113] Articular cartilage is also an estrogen-sensitive tissue.[114] It has been demonstrated that tamoxifen, an estrogen

antagonist, reduces the development of experimentally induced DJD in rabbits. In contrast, estradiol facilitates the process. Both estradiol and tamoxifen affect proteoglycan, prostaglandin, and proteoglycanase production by cartilage cells.[115]

Limitations of Remodeling

Change in condylar morphology, as a result of remodeling, is a normal biologic adaptation to continual functional demands made on the TMJ. In a study of 96 joint specimens from young adults, localized surface changes were common.[116] Local modifications of both the condylar surface and the overall condylar shape appear to be interrelated adaptive responses to functional stimuli. Remodeling changes, however, present with high variability. Different presentations of remodeling may be observed between the lateral and medial aspects of the joint components within the same joint. Although DJD usually occurs in an articular area formerly subjected to remodeling,[53] the variable presence of adaptive remodeling alone cannot predict a progression to active degenerative disease.

Different functional demands made on the mandibular complex contribute to the variable nature of remodeling changes. Animal studies indicate that changes in mandibular position obtained by intraoral or extraoral devices induce characteristic remodeling changes in the articular surfaces.[117,118] Anterior condyle placement caused by the application of Class II orthodontic forces is accompanied by osteogenesis on the posterior aspect of the condyle and increased periosteal deposition on the post glenoid tubercle. Distal placement of the condyle with Class III orthodontic forces produces regressive remodeling of the posterior condylar surface and anterior surface of the post glenoid tubercle.[119] Internal derangement with anterior disc displacement may lead to flattening of the anterior condylar surface, whereas posterior disc displacement is accompanied by flattening of or concavities in the posterior aspect of the condyle.[120]

Contribution of Occlusion

Functional demands may be imposed on the TMJ, in part, by altered occlusal relationships. Characteristics of the occlusal scheme may depict patterning and intensity of forces transferred to the articular surfaces of the joints.[121] Biomechanical analyses of the mandibular complex predict that joint loading is increased as the point of application of bite force is moved anteriorly.[122] Unilateral loss of tooth support will increase loading in the contralateral joint,[41] as does unilateral chewing.[46,123] Although the etiologic contribution of excessive joint loading to onset of osteoarthrosis is plausible, the direct etiologic effect of occlusion is debatable. The remodeling capacity of the TMJ demonstrates that the joint can accommodate and adapt to various occlusal conditions.[124]

Pullinger et al[125] analyzed occlusal variation in an osteoarthrotic sample of patients and reported that occlusion was neither a unique nor a dominant factor in defining the sample. They concluded that features such as anterior open bite in patients with osteoarthrosis are the consequence of, rather than an etiology for, DJD. Seligman and Pullinger[126] stated that epidemiologic studies may demonstrate associations between occlusal factors and DJD but fail to prove the etiologic contributions of occlusion. It is currently open to speculation whether specific occlusal factors predispose to DJD[4,127–135] or rather result from intracapsular or capsular changes.[136] The specific etiologic role of occlusion, with respect to TMD in general, remains to be proven.[124,137]

Diagnosis

Clinical Considerations

Clinical findings vary with the course of degenerative disease of the TMJ. There is a potentially asymptomatic DJD population segment who, under certain circumstances, may suddenly become symptomatic.[138] Trauma, in general, is a common precipitating factor. Principal clinical findings in osteoarthrosis of the TMJ include pain on movement or biting, reduced range of motion, joint tenderness to palpation, and crepitus.[15,139] Rasmussen[140,141] reported that during the early painful phases, restricted joint mobility and limited excursive movements are accompanied by tenderness of the joint capsule and pain in the masticatory muscles. As the pain ceases, mobility improves and crepitation, if not already present, may appear.

Except for crepitation, the clinical signs and symptoms of patients with TMJ degenerative disease do not differ from those of other patients with mandibular dysfunction.[142] Crepitus is an accurate predictive sign of DJD[140,141] but has low sensitivity as a diagnostic sign. Rohlin et al[20] found that 10 of 12 joints with crepitation had degenerative changes while the remaining two had extensive remodeling. In one study,[69] only one half of joints with confirmed DJD exhibited crepitus. If crepitation is used as the only diagnostic criteria, joints with DJD will not be properly diagnosed.

Imaging Considerations

Radiographic observations characteristic of DJD include reduced joint space, osseous flattening, subchondral sclerosis, erosions or loss of cortical lining, and presence of osteophytes.[143,144] Reduced joint space, particularly in association with crepitation, may represent articular soft tissue destruction. However, Kopp and Rockler[144] concluded that reduced joint space is probably not an indicator of DJD if molar support is present and the radiographs are exposed with the mandible in the intercuspal position. In advanced cases, osteophytes and lipping may be found in the anterior part of the condyle. Apparent osteophytes can be caused by either apposition of bone or simulated apposition due to destruction of adjacent areas. If condylar surface erosions or irregularities are observed, they are predominantly located in the lateral pole.[145]

It is often difficult, if not impossible, to radiographically differentiate between degenerative changes and adaptive remodeling.[143,146] Osseous changes must be pronounced to be detected radiographically;[147] thus, early degenerative changes in the articular soft tissue may occur long before radiographic signs are visible.[147,148] Condylar and eminential flattening are frequently associated with bony condensation or subchondral sclerosis, independent of arthrosis,[19] and may be adaptive responses to increased functional loading.[144] Especially in the absence of degenerative signs such as surface erosions or irregularities, condylar flattening and subchondral sclerosis are usually representative of remodeling. However, the absence of radiographic changes cannot exclude the presence of degenerative lesions.

Osteoarthritic hard and soft tissue abnormalities have been identified using magnetic resonance imaging (MRI).[149–151] Cartilage erosions, visible with MRI, are potentially quantifiable.[152] In rhesus macaques knees, MR relaxation times and proton density values have been shown to vary with the severity of osteoarthritis.[153] In other animal models, MRI is positive for accumulation of synovial fluid and cartilage degradation.[154] Researchers have also shown correlational trends between areas of decreased signal intensity and histologic degenerative changes in cartilage of goat knees.[155] In the human hip, MRI may be sensitive for specific early degenerative change[156,157] but may underestimate cartilage and osseous abnormalities.[158,159] Although MRI of the TMJ may confirm disc displacement,[160–162] it has been shown to underdiagnose osseous changes, adhesions, and perforations.[163,164]

Histochemical Considerations

Histochemical markers of cartilage metabolism have been identified, and they correlate with early soft tissue changes associated with onset of osteoarthritis.[165,166] Results of synovial fluid assays suggest that the enzyme activities of N-acetyl-beta-glucosaminidase and N-acetyl-beta-galactosaminidase reflect the degree of TMJ dysfunction.[167] Fibronectin fragments are detectable in synovial fluid aspirates of patients with osteoarthritis and are known to potentiate release of metalloproteinases resulting in proteoglycan depletion.[168–170] In mice inbred for osteoarthrosis, observed increases in serum collagenaselike (CL) peptidase and prolyl endopeptidase (PEP) occurred at an earlier stage than histologic changes.[112] Histochemically detectable entities may be useful as early biochemical markers of the onset of osteoarthrosis.

Treatment

Palliative Treatment

Palliative care should begin with an explanation of the nature and prognosis of TMJ degenerative disease.[171] Management is primarily symptom directed, based on the understanding that DJD appears to run a clinical course of 1 to 3 years generally followed by a natural regression of symptoms.[15,17,172,173] Modification of parafunctional habits such as clenching, bruxing, and gum chewing should be undertaken. Patients should be advised that the joints may be easily irritated,[174] and unnecessary mechanical stresses on the joint may be avoided by changing the diet to softer, smaller food.[175] Physiotherapy, utilizing various modalities to control inflammation, reduce secondary muscle spasm, and improve joint mobility, should be undertaken and followed by home exercises.[174,176,177] If pain relief is inadequate, short-term analgesic and anti-inflammatory medication may be helpful.[176]

Splint Therapy

Existing concepts of DJD etiology suggest treatment should attempt to reduce loading in the TMJ. Distraction of the TMJ may be an effective means of eliminating joint loading and has been attempted with spring mechanics,[178] splints with increased vertical dimension,[179] and pivoting splints.[180,181] However, the effect of splint therapy on condylar distraction has been shown to be questionable.[42] Rasmussen[182] reported that treat-

ment of DJD with pivotal splints resulted in relief from pain but increased the extent of the radiographically observed degenerative disease.

Occlusal splint therapy remains a common treatment modality. Ito et al[42] investigated joint loading associated with several different splint designs. Splints without posterior tooth support result in increased joint loading during clenching. Likewise, splints with unilateral posterior contact create increased loading in the contralateral joint, with distraction of the ipsilateral joint. Bilateral centric stops on posterior teeth appear important for protecting the joints from excessive loading, particularly during parafunctional activities. Occlusal splint therapy can reduce joint loading indirectly by reducing muscle hyperactivity,[174,183] and it may provide symptom relief by decreasing coexistent neuromuscular symptoms.[172,184,185]

Injection Modalities

Intra-articular injections have been proposed as a possible treatment modality for some types of TMD.[186] Human osteoarthritic synovial membranes experimentally treated with hydrocortisone demonstrate decreased production of alpha and beta IL-1[187] known to be involved in the osteoarthritis pathophysiologic process. Corticosteroid injections have also been shown to suppress enzyme synthesis in experimental osteoarthritis.[188] Clinically, intra-articular corticosteroid injection is known to give short-term symptom relief[189] but is also controversial.[190] Intra-articular corticosteroid injections have been associated with both beneficial and adverse effects.[191-193]

Intracapsular injections of hyaluronic acid have been shown to provide relief from TMJ pain.[194] The sodium salt of hyaluronic acid, sodium hyaluronate, is a high-molecular weight polysaccharide[195] that functions as a lubricant in normal synovial fluid.[196] Short-term results of intra-articular injections into painful shoulders have produced rapid symptom relief from pain.[197] However, Bertolami et al[198] reported that patients with temporomandibular DJD who received intracapsular sodium hyaluronate, compared to placebo injections, show no significant difference in treatment outcome.

Surgical Treatment

Temporomandibular joint surgery is restricted to patients with long-standing, severe pain and restricted range of mandibular movement who show no favorable response to conservative treatment.[199] Arthroscopy has been introduced into standard therapy for TMJ internal derangement and osteoarthritis, and it compares favorably with open surgical techniques.[200] Arthroscopy also has diagnostic merit with the potential to provide highly tissue-specific pathologic information.[201] Short-term outcome of patients treated with arthroscopic surgery, compared with nonsurgery patients, includes subjective reports of increased mobility and pain relief.[202] In experimental models, however, arthroscopic intervention may cause irreversible changes in TMJ articular tissues.[203] Different approaches, using animal and human models, provide continued rationale for open and arthroscopic surgical techniques.[200,204]

Prognosis

Degenerative joint disease appears to have an initial destructive phase, and a subsequent reparative phase, that terminates in healing. Rasmussen[145] estimated that the destructive and reparative phases last 1 to 1.5 years each. In 75% of subjects examined, the entire course is estimated to be less than 18 months, and subjects generally complete the healing phase by 3 years. Despite improvement in subjective symptoms, however, radiographic evidence of healing is minimal.[205,206] Generally, subjective symptoms subside,[199,207] and the joints appear clinically stable. Residual symptoms such as mild restriction of movement and crepitus remain in many patients long after the subjective symptoms subside.[199,208] Long-term studies confirm that the majority of DJD patients with crepitus show no clinical change in crepitation.[205]

Rasmussen[182] compared effects of various treatment modalities on subjective symptoms of patients with DJD. Treatment included flat plane splints, pivotal splints, steroid injection, and no treatment. No statistically significant difference was found in the duration of presence of symptoms with the different treatment modalities in the study population. Pullinger and Seligman[209,210] suggested that there are two distinct populations of patients with DJD. One group is mainly composed of patients under 35 years of age where internal derangement precedes onset of DJD. A second group is composed of an older population where internal derangement is secondary to the DJD process. The concept that although the end result is similar, there is more than one pathogenesis helps to explain many of the apparent conflicts regarding etiology, diagnosis, and management of patients presenting with DJD.

Conclusion

Temporomandibular DJD is a pathologic response to mechanical stress placed upon the articular surfaces of the joint. There is a delicate balance between adaptive response (remodeling) and nonadaptive response (degeneration) to functional demands. Articular surface breakdown can occur because of increased mechanical stress or reduced ability of the tissue to withstand and adapt to the applied stress. The aim of treatment of DJD is to shorten its natural course or at least to make it more tolerable. It is hoped that treatment during the active phases of the disease will relieve pain, preserve function, and prevent or minimize deformity. Once the disease process has stabilized, treatment is aimed at minimizing TMJ loading. These objectives are, most likely, best achieved with a multiprofessional approach.

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Resumen

Enfermedad Degenerativa de la Articulación Temporomandibular

La progresión de la enfermedad degenerativa de la articulación depende de los procesos patológicos subyacentes y/o reactivos envueltos, y que en general, comprometen la adaptabilidad tisular. Se presenta una revisión de literatura clínica y experimental relacionada a la enfermedad degenerativa de la articulación. Se describe la epidemiología, patogénesis, diagnóstico, tratamiento, y pronóstico con un énfasis particular en la articulación temporomandibular (ATM). Este artículo describe los factores que afectan la paridad en la remodelación/degeneración y presenta la razón fundamental de los enfoques de diagnóstico y tratamiento.

Zusammenfassung

Degenerative Erkrankungen des Kiefergelenkes

Das Fortschreiten einer degenerativen Gelenkerkrankung hängt vom ihr zugrundeliegenden pathologischen und/oder reaktiven Prozess ab, der, im allgemeinen, die Adaptationsfähigkeit des Gewebes einschränkt. Es wird eine Übersicht über klinische und experimentelle Literatur zu degenerativen Gelenkerkrankungen vorgestellt. Epidemiologie, Pathogenese, Diagnose, Therapie, und Prognose werden beschrieben, mit speziellem Schwergewicht auf dem Kiefergelenk. Der Artikel behandelt Faktoren, die das Gleichgewicht zwischen Remodeling und Degeneration im Kiefergelenk beeinflussen können und stellt Grundprinzipien zur Diagnose und Therapie vor.