Osteoarthrosis/Osteoathritis in the Temporomandibular Joints

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Temporomandibular disorders (TMDs) can be categorized into 3 groups: muscle disorders, internal derangements, and degenerative diseases. Historically, the latter have been overlooked as a structural basis for TMD.

Osteoarthrosis is a noninflammatory disease characterized by a pattern of reaction of the joints to injury. Deterioration and abrasion of articular cartilage and soft tissue surfaces, thickening and remodeling of the underlying bone, and formation of marginal spurs and subarticular cysts can be observed. These changes are very commons in elderly patients, but are often asymptomatic.

Patients may become symptomatic and severely disabled when osteoarthrosis is associated with synovitis. Secondary inflammatory changes resulting from tissue damage in the joint may play a role in producing pain. The term osteoarthritis best describes this secondary inflammatory arthropathy.

Prevalence

Osteoarthrosis/osteoarthritis (OA) is the most common degenerative disease of temporomandibular joints (TMJs); however, the prevalence is difficult to establish since there is a lack of concordance between structural changes and pain. The method of examination is a significant factor in determining the prevalence of OA (Table 1 and Fig 1). OA is strongly correlated with age, and occurs more often and is more generalized in women than in men.

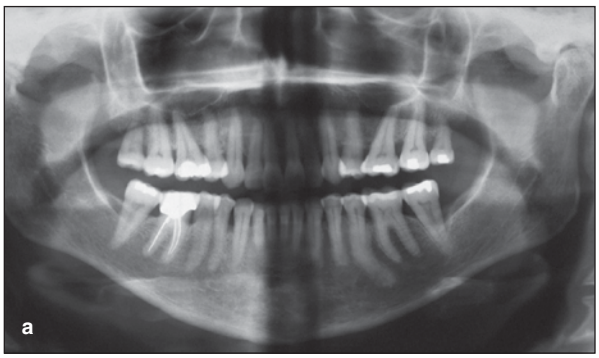
Etiology

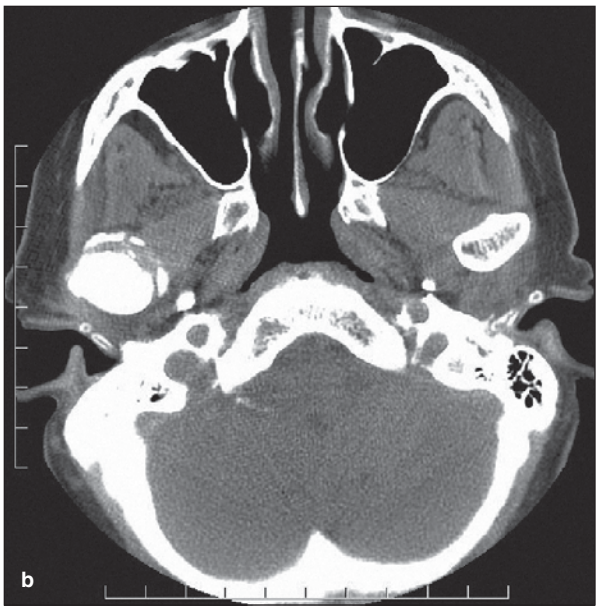
The etiology of TMJ OA is not clearly established but appears to include both systemic and local factors. Joint overload is predominant in the development of OA. Articular cartilage is primarily affected since it shows limited adaptive capacity compared with other connective tissues and subchondral bone. Overloading may exceed mechanical properties of the cartilage and disturb tissue remodeling.

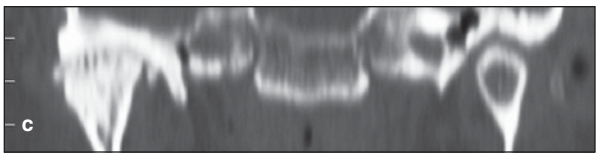
Age is clearly a predisposing factor, although the evidence of age-dependant alterations in joint tissue is unclear. Many autopsy studies suggest a strong correlation between loss of molar support and the occur-

Table 1 Prevalence of OA in TMJs

Method of evaluation	Prevalence (%)
Clinical	8–18
Radiologic	14–44
Macro/Microscopic (from autopsy)	22–84







Figs 1a to 1c (a) Orthopanthogram showing structural changes of the right TMJ and a normal left joint. **(b and c)** Computerized tomography scan (axial cut and coronal reconstruction) showing reduced joint space, severe subchondral sclerosis, and osteophytes in the right TMJ. (Images courtesy of Dr Marie Dagenais.)

rence of OA, especially in individuals over 40 years of age. Because of the high prevalence of partial and complete edentulism in elderly patients, it can be suggested that the role of aging is significant in this context of depleted dentitions and resulting adverse biomechanical loading.

With rare exceptions, OA is associated with disc displacement. It is also believed that joint overload plays a role in the development of internal derangement (ID). The relationship of time onset between ID and OA is not fully understood. It seems more likely that ID precedes OA; however, it is possible that the causative event simultaneously initiates both conditions.

Trauma to the face may also cause or aggravate OA. Repetitive loading associated with parafunction is possibly an etiologic factor, but a clear relationship has not been established. Genetic and metabolic factors that could influence the threshold for tissue damage may also be important factors in the development of OA.

Signs and Symptoms

Patients usually complain of pain and tenderness in the joint, jaw muscle fatigue, stiffness and tiredness, reduced range of motion, and joint noises during mandibular movement. Pain is aggravated by wide opening of the jaw, mastication, and parafunction, and can radiate from the joint to the temple, ears, side of neck, and upper shoulder. A history of changing occlusion and acquired facial skeletal deformity (open bite, asymmetry) may also be signs of OA.

These signs and symptoms are similar to those of other TMDs with subtle but very significant exceptions. Generally, OA is unilateral, although bilateral involvement does occur. In these cases, one side usually shows greater severity. OA is generally characterized by morning jaw stiffness, and the symptoms appear to worsen during the day. Pain over the joint and crepitation are often present. Radiographic evidence is frequently visible.

There is, however, a poor correlation between radiographic findings and clinical symptoms. Because OA affects the articular cartilage first, early changes such as reduced joint space—indicating loss of the articular cartilage and/or perforation of the disc—may not be detected in radiographs. The most common early change in the condyle is subchondral bone sclerosis; however, substantial changes in mineralized tissues are required for detection. As the disease progresses, flattening and marginal lipping of the condyle and flattening of the articular eminence can be observed. In the late stages, the changes are in the form of erosion of the cortical plate and/or osteophyte formation.

Signs and symptoms improve with time in most individuals. The nature of OA suggests variable symptomatic progressions, which usually burnout in 12 to 24 months. On the other hand, pathologic changes in the TMJ progress with time at a rate that varies among patients.

Clinical Management

A detailed history, head and neck evaluation, and general physical examination are essential for diagnosis. Imaging of the TMJ is necessary to establish the presence of pathology and stage of disease, in order to select the appropriate treatment and assist in prognosis.

Because adaptive mechanisms seem to play a major role in the natural course of TMJ degenerative disorders, treatment should seek to promote a joint condition that is most likely to repair any damage. The goal of nonsurgical management is to reestablish the balance between degradative and repair processes within the affected tissues.

Therapeutic strategies include symptomatic treatment, control or reduction of predisposing factors, and treatment of pathologic sequelae. Surgical management should only be considered after reasonable nonsurgical efforts and when the patient's quality of life is significantly affected.

Bibliography

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