ELSEVIER
SAUNDERS

Dent Clin N Am 49 (2005) 327–342

THE DENTAL
CLINICS
OF NORTH AMERICA

Derangement, Osteoarthritis, and Rheumatoid Arthritis of the Temporomandibular Joint: Implications, Diagnosis, and Management

Jack S. Broussard, Jr, DDS

University of Southern California, School of Dentistry, Oral Health Center, 3151 South Hoover Street, Los Angeles, CA 90089-7792, USA

The prevalence of deterioration of both function and form from ''wear and tear'' in the load-bearing joints of the body increases with aging. Wear and tear deterioration of a joint is called osteoarthritis. Sometimes the dysfunction is a discal derangement problem and does not involve deterioration of the surfaces of the joint. Whether the dysfunction is osteoarthritis or derangement, when it is isolated to one joint, the origin is generally traumatic. When the dysfunction is polyarthritic in nature, the agents of crystal deposition, autoimmunity, Lyme disease infection, and idiopathic causation are evoked.

Polyjoint arthritic disease is extremely common in the human population, with the highest prevalence in the elderly. Polyjoint osteoarthritis is by far the most common of the rheumatic diseases affecting an estimated 20.7 million Americans and is responsible for over 7 million patient visits per year [1–3]. When all of the arthritic diseases are added together, over 70 million United States citizens (one in three adults) report a rheumatic disease [4]. In the group of those 75 years or older, a majority reports having arthritis. As might be expected given this high prevalence, the social and economic impact of rheumatic diseases taxes our health care systems. Adults over the age of 65 have more patient visits for these diseases than any other age group [5]. In addition to clear-cut rheumatologic disease, more than 100 medical conditions affect the muscles, tendons, and joints and are classified as musculoskeletal-related diseases. It is hypothesized that approximately 33% of the United States population demonstrates signs and symptoms of

E-mail address: jbroussa@usc.edu

0011-8532/05/$ - see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.cden.2004.10.003 *dental.theclinics.com*

musculoskeletal disease [5,6]. Moreover, $118.5 billion per year has been spent by United States citizens on the care of musculoskeletal diseases [4,6]. Over $86.2 billion is spent annually on rheumatic diseases [4]. The percentage cost of the United States gross national product used to treat musculoskeletal disease has increased each decade since the 1960s. When these diseases are severe, mobility and functional limitations cause increases in work loss, disability, use of nursing care, and premature retirement. Musculoskeletal disorders are second only to heart diseases as a cause of work disability. Work-loss costs associated with rheumatic diseases account for 50% to 76.5% of all indirect costs [4]. As the older population increases in number and longevity, these costs will continue to rise.

The good news is that the temporomandibular joint (TMJ) is less likely to show aging-related deterioration than are other major body joints, perhaps because the TMJ is less of a load-bearing joint than the knee, shoulder, or spine. Moreover, the prevalence of TMJ signs and symptoms decreases with age. In adult nonpatient populations, the prevalence of at least one sign of temporomandibular disorders (TMD) ranges from 40% to 50% [7–10]. Limited jaw opening may occur in 5% [11]. With the exception of limited opening, which has a slightly higher prevalence in the elderly, the prevalence of jaw pain and TMJ clicking appears to be stable across adulthood, then inexplicably decrease in the population aged more than 65 years. Matsuka et al [12] investigated the prevalence of signs and symptoms of TMD in Japan. They examined 672 individuals (304 men and 368 women between ages of 20 and 92) selected randomly in Okayama City, Japan. They reported that, although TMD signs and symptoms were common in all age groups, they were less numerous in the older age group. Notably, they also reported that clicking was higher in the younger adults, whereas crepitation increased significantly in the oldest age group.

It is perplexing that the elderly do not exhibit more clinical symptoms of joint pain when we consider that the TMJ shows increased morphologic changes with aging, suggesting increased deterioration [13,14]. For example, one postmortem study of elderly cadavers found that 91% of joints examined had morphologic changes, including osteoarthritic change [14]. This article reviews normal anatomy and function of the TMJ, derangements, localized osteoarthritis (OA), generalized or polyjoint OA, and rheumatoid arthritis as they relate to the TMJ and the elderly.

Normal and age-related changes in temporomandibular joint anatomy and physiology

The TMJ is a complex synovial joint. It is the only joint in the human body where the condyle slides completely out of its socket yet is not considered dislocated. This joint is unique in that it can undergo not only hinge movement but also extensive translational or sliding movement. It contains a meniscus composed of dense fibrous connective tissue; the

temporal and condylar articular surfaces are also covered with fibrocartilage, rather than the more typical hyaline cartilage seen in other joints [15,16]. Synovial fluid lubricates the joint and the loading of the articular fibrocartilage, and subchondral bone causes chondrocytes in the articular cartilage to synthesize and secrete collagen, proteoglycans, and other proteins necessary for cartilage and subchondral bone repair [17–21]. Proteoglycan molecules consist of a protein core with negatively charged glycosaminoglycan side chains composed of keratin sulfate and chondroitin sulfate. Aggregates of proteoglycans are linked to a core of hyaluronic acid, allowing the binding of large numbers of water molecules inside this complex molecule. Compression of the cartilage releases this fluid, which is recaptured as compression is removed [20]. This fluid movement allows cartilage to undergo reversible deformations.

Why does the TMJ exhibit such a high prevalence of wear-and-tear–related problems with aging? One explanation is that the interposing disk between the condyle and the temporal component of the joint is damaged or displaced. However, many patients develop joint arthritic changes independent of disk displacement. Certainly crystal deposits (eg, gout and pseudogout) inside the joint, Lyme disease, and autoimmunity reactions also explain joint-surface deterioration. The first two conditions (ie, crystal deposition arthritis and Lyme disease) are not discussed in this article; autoimmunity-related arthritis is covered in the section on rheumatoid arthritis. Another, and perhaps the most important, reason joints break down and become arthritic involves changes in the frictional interface between the condyle, disk, and temporal components of the TMJ.

As in all synovial joints, the fibrocartilage and TMJ disk are largely acellular and are maintained in health and repaired and lubricated by the synovial fluid in the joint. High load or loading that occurs with inadequate lubrication results in a surface breakdown, leading to OA [22]. Aging has been reported to induce articular cartilage thinning in synovial joints: the cartilage actually changes color (from white to a dull yellow) [23]. In addition, the fluid that lubricates and protects the joint surface changes with age. Aging is accompanied by reduced accumulation of this synovial fluid and the synthesis of smaller proteoglycans, which hold less water and exhibit reduced compressive ability and more breakdown in the surface of the joint. This process increases keratin sulfate and reduces chondroitin sulfate content in the synovial fluid. These changes, partially caused by the decrease in water content that accompanies aging and a change in cartilage proteoglycan, are considered one of the earliest signs of articular cartilage loss in OA. Nakayama et al [24] examined normal synovial fluid and measured the concentrations of chondroitin 6-sulfate (C6S), chondroitin 4-sulfate (C4S), and hyaluronic acid in healthy subjects of different ages. The subjects were 82 healthy volunteers ranging in age from 20 to 79 years. They found that the concentrations of CS and hyaluronic acid varied with age. Their values were highest between 20 and 30 years of age and thereafter showed

a tendency to decrease. The ratio of C6S to C4S was significantly lower in the group aged 60 to 70 years when compared with the 20- to 30-year-old group. In fact, multiple regression analysis demonstrated that age correlated strongly with the C6S concentration and the C6S to C4S ratio (ratio = −0.521 and −0.617, respectively).

The oddity of the aged TMJ is that, even though the joint shows clear signs of deterioration, it is not highly painful in most of the patients who have arthritic alteration of the joint. It is possible that TMJ deterioration changes are better tolerated (less painful) in the elderly than in younger patients. In fact, research has suggested that the degree of inflammation cartilage damage induces in the elderly is inferior to that caused by the same insult and injury to the joint of a young patient. This concept of reduced degradation is based on the work of DeGroot et al [25], who reported on an age-related decrease in the susceptibility of human articular cartilage to matrix metalloproteinase–mediated degradation. Specifically, they looked at the effect of age-related accumulation of advanced glycation end products (AGEs) on cartilage. They found that, the more AGEs were accumulated, the less susceptible the cartilage was to proteolytic degradation by matrix metalloproteinases present in synovial fluid in both OA and rheumatoid arthritis patients. The joint deterioration changes seen in the elderly, which have developed slowly and progressively across a lifetime, may also be better tolerated than problems due to acute injury in younger patients. A change in nociceptive physiology may even occur in the elderly.

Local temporomandibular joint disk dysfunction in the elderly

Although disk derangement of the TMJ is more common in the 20- to 50-year-old population, the elderly have their share of dysfunction (clicking and locking). When an elderly patient develops new-onset TMJ clicking, the first order of business is to rule out osteoarthritic disease as the underlying cause. If no OA changes are evident on the imaging of the joint, then education about the need to avoid this frictional event during function is essential to reducing the likelihood of progression to a more serious deterioration of the joint. This approach is called avoidance therapy and has three components. First, the patient is shown how to limit the degree of jaw opening and protrusion, thus stopping translation and TMJ clicking. Second, the patient is informed that chewing on the same side as the click usually helps to avoid it and is provided with a chewable substance (eg, gum) so they can attest, under the physician's observation, that this is so. Third, the patient is informed that a dietary change to small bites and soft food will be necessary to avoid the click. Strict avoidance of all clicking and all gum chewing is required. It should be clearly understood that this approach will not stop clicking, only reduce the number of times the disk is jammed between the condyle and eminence, thus reducing wear and tear on these tissues.

In the elderly patient who presents with a jaw locked open, manipulation of the jaw is necessary to achieve a closure of the opening. Depending on how long the jaw has been locked, the manipulation of the jaw can usually be performed without adjunctive medications (antispasmodics). Assuming the mandible is locking in a wide-open position, this manipulation involves holding the mandible with both hands, placing the thumb on the back molars of the lower jaw and the fingers under the mandible on each side. The manipulation is performed by pushing down on the mandible with the thumbs while pulling up on the anterior portion of the mandible with the fingers. When successful, this manipulation allows the condyle to move down and then back, so that it can then move posteriorly to the TMJ eminence. If manual manipulation alone is not successful, the next step is to sedate the patient with a short-acting benzodiazepine. This medication can be delivered orally (if the patient can swallow) or intravenously (if he or she cannot swallow or if haste is needed in the reduction process). Once reasonable sedation is achieved, the previously described manipulation is usually easier to perform.

In those cases where the open locking problem has been reduced but has become a recurrent event, a thorough history and examination of the TMJ with tomographic or MR imaging should be completed. If these films show no osseous abnormality or substantial disk derangement, the primary therapy is to avoid wide opening to prevent more recurrences of the open locking. If, however, a substantial articulator surface or discal disease is present, the primary treatment for a mechanical derangement of the TMJ is arthroscopic surgery to achieve a thorough lavage and stretching of the TMJ.

Local temporomandibular joint arthritis in the elderly

Localized OA is characterized by focal degeneration of joint cartilage with osseous erosion and sclerosis and sometimes osteophyte formation occurring at the joint margins [26]. It is considered a disease of the bone, cartilage, and supporting tissues and results from both mechanical and biologic events that destabilize the normal coupling of degradation and synthesis of articular cartilage and subchondral bone [22]. Localized OA is usually thought to be traumatic in nature (either macrotrauma or repetitive microtrauma) but may also be due to a rare infective arthritic disease. When an elderly patient comes to a dentist's office with a complaint of jaw pain, the most likely diagnosis is localized arthritis (assuming the patient does not exhibit polyjoint arthritic disease). The arthritis can usually be discovered with palpation, auscultation, and radiographic examination of the joint. Occasionally, the jaw joint pain is related to a disk derangement of the jaw (clicking, locking, or dislocation), but OA is the more prevalent problem among the elderly. Dulcic et al [27] examined the frequency of internal derangement of the TMJ in elderly individuals. They examined the TMJ of 96 elderly subjects (mean age 76) and reported that a clinical diagnosis of

internal derangement of the TMJ was present in only 9.3% of the subjects. By contrast, about 20% of a younger population (ages 20–50) typically exhibit noticeable symptoms of internal derangement.

As mentioned earlier, clicking and locking do not increase among the elderly, but localized OA does. In a 2003 study based on a European population, Gillette and Tarricone [28] reported that the prevalence of OA is approximately 12% for subjects between 25 and 50 years of age but reaches as high as 95% in the subset of patients over 60 years of age. Fortunately, osteoarthritic changes in the TMJs of the elderly population are much less prevalent than the above data for all body sites might suggest. Specifically, Hiltunen et al [29] reported on a random sample (N = 88) of elderly subjects between the ages of 76 and 86 years living in Helsinki. The most frequent radiographic finding in the TMJ was flattening of the articular surface, indicating OA, which occurred in 17% of the population.

Still, assuming that 17% of the population over 65 have TMJ osteoarthritic change and 9.3% have internal derangement, this is a large group of patients. It is likely that as many as 50% of those with radiographic change have at least a mild-to-moderate degree of pain and dysfunction in their jaws. Aging in and of itself is not thought to cause OA, but if a combination of several age-related changes occurs in the same individual, OA will result. Specifically, forceful repetitive function (eg, bruxism) or disk displacement, along with synovial fluid alterations of the TMJ, will predispose the elderly individual to OA.

Treatment of localized osteoarthritis and derangement in the elderly

When joint pain symptoms occur and management is required, the principles governing treatment of the TMJ are no different from those used to treat other body joints with painful OA. Treatment always begins with nonpharmacologic therapy, which includes patient education, rest, and physical therapy, including the use of occlusal appliances (when tooth clenching or an unstable bite is evident). Education should be directed toward reducing stressful jaw function (eg, chewing hard foods) and reducing or eliminating aggravating factors, such as teeth clenching, opening wide, and gum chewing. One consistent feature of arthritic disease management that applies to both TMJ derangement and TMJ arthritis is that the patient must be taught about the chronic nature of the disease. It is sometimes difficult for patients to accept that the displaced disk in their jaw joint cannot be put back in place or that the damaged joint tissues cannot be repaired or replaced. Depending on the medical propaganda to which patients have been exposed, they may have unrealistic treatment expectations, which can lead to frustration and depression.

Physical therapy for TMJ OA usually consists of teaching the patients how to perform jaw exercises and apply heat or ice packs to the jaw. Two primary jaw/tongue posture exercises are recommended. First and foremost

is the ''N'' position exercise, which involves placing the jaw and tongue in the position achieved when saying the letter ''N'' and holding it for a count of 10. The patient is instructed to perform this exercise hourly each day (or 12 times a day). The goal is to put and hold the jaw in the most relaxed jaw position where the teeth are apart and the lips not touching. Once patients' initial pain symptoms are shown to be reduced, they can proceed to the next exercise, called the jaw hinge exercise. The patient is instructed (with use of mirror feedback) to move the jaw carefully in a strict hinge motion to a point about 15 mm open and then close it again. This motion promotes synovial fluid movement without any translation of the condyle. The motion is usually performed 15 times on a 2- to 3-hour schedule, or six times a day.

Although the N-position and hinge exercises help reduce strain on the joint, it is also advisable to apply 20 minutes of heat therapy (hot towels or a moist heating pad) to the sorest muscles. Heat helps to reduce pain and stiffness by relaxing aching muscles and increasing circulation to the area. Finally, nonopioid analgesics and nonsteroidal anti-inflammatory drugs can be helpful. (For more information see article on prescriptions and over-the-counter medications for arthritis in the elderly elsewhere in this issue.)

Corticosteroid injections help with any inflammatory TMJ pain problem that is unresponsive to the usual treatments. This approach may be an early treatment intervention in patients with gastritis, gastro-esophageal reflux disorder, or other indications for not using a nonsteroidal anti-inflammatory medication. When indicated, a corticosteroid intra-articular injection using one of several different corticosteroids is appropriate; a common one is triamcinelone [30,31]. The usual amount of medication injected in a single jaw joint is 10 to 20 mg. The injection is targeted to the superior joint space, and the corticosteroid is usually mixed with an equal amount of local anesthetic to make the joint injection more comfortable. After the injection, it is wise to recommend ice packs as needed and a completely soft diet for 48 hours until the injection has an effect on the inflammation. The general guideline is that the TMJ should not be injected more than twice in a 12-month period.

Vallon et al [32] examined the possibility of long-term adverse effects of repeated corticosteroid injections on the TMJ in patients with rheumatoid arthritis (RA). They performed a long-term (12-year) follow-up of 21 patients with RA and symptomatic TMJs who received either an intra-articular injection of a steroid (n = 11) or a local anesthetic agent (n = 10). Fourteen patients who were available for long-term follow-up reported no pain from the TMJ. Radiographic follow-up examination was performed on 12 patients, and all but 4 of the 24 joints had structural bone changes. Interestingly, the magnitude and prevalence of change was no different in the two groups. The investigators concluded that the chances of long-term progression of joint destruction for the steroid-injected and non–steroid-injected joints were equivalent in this patient group with RA. Presumably these results can be generalized to osteoarthritic disease in the TMJ.

For patients who have only a transient response to the corticosteroids, hyaluronic acid injections have been used with moderate success for new-onset OA with crepitation. Intra-articular hyaluronic acid injection can provide symptomatic relief for several months. Two approved drugs currently exist: Synvisic and Hyalgan. These medications are given in a series of three injections, 1 month apart, and are currently approved for knee joints [33,34]. They have been approved for OA of the knee and are also helpful for TMJ pain and dysfunction. One of the first studies of the use of hyaluronate in the TMJ was performed over a decade ago by Bertolami et al [35], who studied 121 patients at three test sites using a randomized, double-blind, placebo-controlled experimental design. Patients were selected on the basis of a confirmed diagnosis of degenerative joint disease (DJD), reducing displaced disc (DDR), or nonreducing displaced disc (DDN), along with nonresponsiveness to nonsurgical therapies and a severe jaw dysfunction according to several measures. Subjects received either a unilateral upper joint space injection of 1% sodium hyaluronate (HA) in physiologic saline or a United States Pharmacopia physiologic saline injection. The researchers reported no differences for DJD and only minor difference for DDN. However, for DDR they found a statistically significant within-group and between-group improvement throughout the 6-month test period. More recently, Shi et al [36] have evaluated the effect of HA on TMJ degenerative and derangement disorders using a prospective, randomized, controlled clinical trial in 67 subjects (12 men, 51 women). They provided HA injections in the upper compartments of the involved TMJs, with 35 patients receiving 6 mg of 1% HA and 28 receiving 12.5 mg of prednisolone. The protocol in this study included three to four injections over a 2-month treatment period. The investigators concluded that the intra-articular injection of HA is effective and safe for the treatment of TMJ degenerative disorders, with mild adverse reactions.

Topical creams (eg, capsaicin or nonsteroidal anti-inflammatory drugs [NSAIDs]) have been described as being helpful if the patient does not tolerate oral medications and does not want an injection-based therapy. Many products are sold over the counter to help patients with arthritis pain. However, the experimental data for transdermal therapy in arthritis are weak. For example, Winocur et al [37] examined the effect of topical application of capsaicin on localized pain in the TMJ area. They conducted a randomized, double-blind, placebo-controlled study in 30 patients suffering from unilateral pain in the TMJ area. Patients received either 0.025% capsaicin cream or its vehicle and were instructed to apply the cream to the painful TMJ area four times daily for 4 weeks. Capsaicin cream produced no statistically significant difference in the outcome measures when compared with placebo. This general result was supported by another, more recent study by Myrer et al [38], who examined the effects of a topical analgesic and placebo in treatment of chronic knee pain. They conducted a double-blind, randomized, placebo-controlled clinical trial in 46 men and

women with chronic knee pain. Testing took place before treatment and after 21 and 35 days of treatment. Although both groups experienced improved pain scores, there were no differences between groups over the treatment period for any of the dependent variables.

Another topical medication (transdermal lidocaine patch) has become available for neuropathic pain. The effect of this new patch on OA was recently evaluated by Burch et al [39]. Specifically, they looked at the effectiveness of the lidocaine patch 5% on pain, stiffness, and function in OA pain patients in a prospective, multicenter, open-label effectiveness trial. The authors concluded that the lidocaine patch 5% appears effective as an add-on therapy for OA pain. They recommended the use of up to four patches, changed every 24 hours, to provide effective analgesia without anesthesia and to reduce stiffness and disability and improve quality of life in polyjoint arthritis patients, particularly those who have responded incompletely to prior medication therapy. This approach has the advantage of offering an effective topical analgesic option for OA with a minimal risk of systemic toxicity or drug interactions. Obviously, controlled clinical trials are needed to confirm the efficacy and safety of lidocaine patch 5% therapy.

Finally, arthrocentesis-based lavage is useful for TMJs with limited mobility but is controversial as a treatment for joint pain without joint hypomobility. The primary goal of this procedure is to mobilize the TMJ; it involves washing the joint with saline solution and conducting manual manipulation of the jaw when it is anesthetized. It plays a role primarily in those patients who do not respond to pharmacologic treatment and present with limited opening. The long-term benefits of arthrocentesis lavage are unknown; only limited good-quality comparative therapy studies have been done. Moreover, there are no data on the efficacy of arthrocentesis in the elderly TMJ. Nitzan and Price [40] looked at the use of arthrocentesis for the treatment of osteoarthritic TMJs. Specifically, they examined 36 patient records in a retrospective fashion. The patients were 29 women and 7 men (aged 16 to 54 years) presenting with 38 dysfunctional joints that exhibited OA and had not responded to conservative treatment. The authors reassessed the patients after arthrocentesis, at a time point varying from 6 to 62 months. They reported that, of the 38 OA TMJs treated with arthrocentesis, 26 joints reacted favorably to the treatment. The authors even stated that in many instances the osteoarthritic TMJs returned to a healthy functional state. Emshoff and Rudisch [41] looked more closely at who benefited from an arthrocentesis procedure. They evaluated 29 TMJ pain patients with a diagnosis of disc displacement without reduction and various degrees of OA. They used a multiple logistic regression analysis and reported a significant increase in risk for an unsuccessful outcome in patients with a pretreatment report of chronic TMJ pain. Finally, Gu et al [42] reported on the effect of intra-articular irrigation injection therapy on OA of the TMJ. They treated 37 patients (the test group) with an

intra-articular irrigation (arthrocentesis) and 26 with an intra-articular injection of steroid only. The percentages of patients rated as excellent or good in the two groups were 86% (arthrocentesis group) and 65% (steroid group), and the authors claimed this difference was statistically significant.

One possible reason why arthrocentesis is seldom used in the elderly is that their OA of the TMJ appears to be self-limiting, results in mild-to-moderate dysfunction, and is usually managed well with traditional nonsurgical therapy. The data discussed previously suggest that arthrocentesis-based lavage of the TMJ can be effective in increasing range of motion and decreasing pain in cases not responding to medical management, but that it should be restricted to cases with recent-onset hypomobility.

Polyjoint osteoarthritis and the temporomandibular joint in the elderly

Polyjoint or generalized OA is generally classified as either primary or secondary. The most common sign of a primary polyjoint OA is when the patient demonstrates the formation of Heberden's nodes on his or her distal interphalangeal joint of the hand. The proximal interphalangeal joint, the first carpometacarpal joint, and the spine, knee, and hip joints are also common OA sites. Primary polyjoint OA is more or less considered idiopathic, although genetic defects are suspected strongly in this disease, especially when a familial pattern of OA is present [22,26,43,44]. Secondary polyjoint OA is defined as joint damage or cartilage changes characteristic of OA caused by other disorders [43,45]. Secondary polyjoint OA may present in congenital and developmental disorders. Prior trauma, surgery, inflammatory disease, bone disease, blood dyscrasias, neuropathic joint diseases, excessively frequent intra-articular steroid injections, endocrinopathies, and metabolic disorders may damage joint surfaces and cartilage [22,44,45]. Finally, in cases of severe and aggressive polyjoint OA, a negative serologic test for rheumatoid factors should be obtained before the diagnosis of polyjoint or generalized OA is made.

The genetic defects most likely to be discovered involve the type-2 cartilage collagen binding proteins. This molecule forms a three-dimensional cross-linked fiber network with proteoglycan, creating compressibility and elasticity of the joint surface [22]. In addition to overt genetic defects, aging appears to decrease the ability of articular cartilage to withstand loading pressures and thus to promote cartilage degradation. In a recent review, Jordan et al [46] state that a formal study of the genetics of OA has only recently been undertaken. They discuss the findings from twin studies, segregation analyses, linkage analyses, and candidate gene association studies and summarize important information about inheritance patterns and the location of potentially causative mutations in the genome. Unfortunately, the various studies have not always agreed on the genetic factors to blame. This discordance probably results from variations in study populations, disease definitions, evaluation of

control subjects, and statistical analysis. Nevertheless, most genetic researchers interested in OA believe that a complex of genetic defects will be identified in the near future.

Rheumatic arthritis and the temporomandibular joint in the elderly

RA is a chronic, systemic, autoimmune inflammatory disorder that is characterized by joint inflammation, erosive properties, and symmetric multiple joint involvement. RA can involve other body organs and is an aggressive disease causing joint damage within 2 years, decreased function, and increased impairment. It shortens life spans by 5 to 7 years [47–49] and, if severe, significantly alters the quality of life. The main serologic marker, rheumatoid factor (RF), an IgM autoantibody against constant fragment portion of an IgG molecule, is found in 75% to 80% of patients. Although the cause is unknown, certain genetic markers, namely HLA-DR4 and DR1, are found in approximately 30% of patients with RA. Familial studies offer strong evidence for genetic factors. Some evidence suggests that an infectious agent (eg, virus, bacteria) may trigger the disease in genetically predisposed individuals. Edema, hyperplasia of synovial lining, and inflammatory infiltrate are early components of RA onset. Chronic RA is characterized by hyperplasia of type-A synovial cells and subintimal mononuclear cell infiltration, resulting in massive damage to cartilage, bone, and tendons by the pannus, an infiltrating inflammatory synovial tissue mass [47–50].

Although RA is found in the TMJ, it appears to be one of the last joints attacked by RA. The TMJ is affected in more than 50% of adults and children with RA [51]. Clinical findings include dull aching pain associated with function, joint edema, and limited mandibular range of motion. Anterior open bites are common. Morning stiffness or stiffness at rest lasting longer than 1 hour is common. Radiographic findings range from flattening of the condylar head to severe, irregular deformity. Multiple joints are typically involved. Symmetric polyarthritis of at least three joints in 14 areas is found in 50% of patients. Laboratory tests for serum RF are positive [47,51].

Pharmacologic treatment of localized or polyjoint arthritis affecting the temporomandibular joint

Pharmacologic treatment of all types of arthritis includes nonnarcotic oral analgesics, such as acetaminophen, in patients with pain complaints. When NSAIDs are used, efficacy, cost, adverse reactions, and comorbidity should be considered; complications of peptic ulcer and bleeding can be serious in the geriatric patient. Renal function should be monitored. Cyclooxygenase–2 (COX-2) inhibitor NSAIDs, such as Celecoxib, cause less gastrointestinal injury because they selectively inhibit the isoenzyme COX-2. They also have a reduced effect on platelet aggregation and bleeding time.

Acetic acids (eg, Sulindac) are said to have a lower rate of renal adverse reactions than other NSAIDs. Indomethicin is poorly tolerated by the geriatric patient. Ibuprofen and naproxen, propionic acids, may be useful. Piroxicam can be taken once a day. Salicylates are less useful in the elderly because of gastrointestinal and central nervous system effects. Salsalate, a nonacetylated salicylate, does not affect platelet function and has minimal gastrointestinal toxicity. Oral corticosteroids have little use in the treatment of OA [51,52], but intra-articular corticosteroid injections may be useful in inflamed joints.

For the more aggressive RA cases that medications such as NSAIDs are not strong enough to manage, the current standard is to use one or more disease-modifying antirheumatic drugs (DMARDs) and a short course of oral or intra-articular steroid. DMARDs act slowly over 1 to 3 months. They appear to alter RA by causing erosive healing, controlling inflammation, and improving function. In practice, the effectiveness of DMARDs varies by patient. DMARDs include antimalarial drug, sulfazalazine, intramuscular gold, methotrexate, hydroxychloroquine, cyclosporine, azathioprine, leflunomide, and cyclophosphamide. Treatment goals in RA include control of immunologic and inflammatory disease process, prevention of joint damage, normalization of function and life span, complete relief of symptoms, return to normal daily activities, avoidance of complications of the disease and its treatments, education, counseling, and physical and occupational therapy [47,52].

Development of new and combined medication regimens is leading to better management. Unique and progressive regimens, such as the use of tumor necrosis factor alpha antagonists in refractory RA, are giving hope to patients with severe disease. Recent work by Alhadaq and Moa [53] with tissue engineering is providing an exciting look into the future. These researchers harvested three tissue-engineered TMJ condyles from host mice. The harvested condyles formed from adult stem cells stimulated to form either bow or cartilage. Although this is early research, someday it may be possible to grow ''new'' condyles to replace diseased ones.

When they understand the influence of genetics, function, and trauma in the initiation of rheumatoid disorders, practitioners can establish clear, reasonable, and attainable treatment goals. We must take time to empower our patients to manage their disease through education, understanding, and reasonable expectations.

Summary and recommendations

The preponderance of data on TMJ is based on patients between the ages of 20 and 50. Very few studies have examined the efficacy of treatment methods for monoarticular or polyarticular disease involving the TMJ. With this caveat, the author offers the following conclusions and recommendations based on the available literature and his own clinical experience:

Conclusions

In most studies where multimodal physical medicine therapy (eg, occlusal appliances, exercises, short-term use of NSAIDs, plus heat/ice and general advice about reduced jaw function) was used, this method produced a positive response in approximately 75% of patients.

The aforementioned treatment approach does not cure the derangement or arthritis; at present, this is not possible.

Judicious use of corticosteroid injections into the superior joint space of the painful TMJ is also a safe and logical therapy for many patients, with a low risk of morbidity and a high likelihood of symptom reduction.

In some patients, this approach will not be adequate, but the percentage of patients who state that they require further treatment is approximately 2%.

When the symptoms linger and do not respond to the above methods, it is likely that some causative factor is still present (eg, strong bruxism or tooth clenching habit, a generalized stress reaction, or an autoimmune disease process).

Therapeutic recommendations

For the patient with all forms of TMJ pain or disk–condyle incoordination (clicking), joint noises are usually managed with strict click avoidance instructions and hinge exercises. This self-directed treatment has the potential to stop and slow the progression of a dysfunctional clicking joint. A new and potentially viable therapy for the most noxious, disturbing joint clicks is the infusion of 1 mL of hyaluronan into the joint space as a method of reducing friction and promoting healing adaptation. This infusion can be performed up to four times (every 3 weeks) in a 1-year period.

For the patient with recent-onset TMJ closed locking, both manual manipulation (with or without anesthetic block of the TMJ) and arthrocentesis-assisted manipulation are logical first-line methods to manage most of the acute cases. Following the manipulation, the physician should institute a series of passive-stretching sessions of the jaw closers.

Open locking of the TMJ is largely avoidable with proper instruction once the locking has been manually reduced. For the patient with recurrent TMJ open locking, a series of hyaluronan injections is the first line of therapy; the second is referral to an oral surgeon for consideration of an arthroscopic surgery to stretch the joint capsule ligament structures or remove the bony abnormality that is inducing the locking.

For TMJ pain due to a polyarthritic diseases process (eg, RA) more specific disease-modifying drugs (eg, immunosuppressive medications) are in order.

The use of occlusal appliances for TMJ pain is indicated in the patient with a clear-cut jaw-muscle clenching or grinding behavior or an unstable occlusion.

Regarding efficacy, it should be noted that most of the individual modalities and methods described here have not been tested in randomized, controlled clinical trials on the specific TMJ disorder with which they appear logically matched. However, multiple clinical case series experiments show that the success rate for physical medicine methods in the management of unspecified TMJ disorder patients is around 75%, depending on the definition of success.

References

[1] Lawrence RC, Helmick CG, Arnett FC, et al. Estimates of the prevalence of arthritis and selected musculoskeletal disorders in the United States. Arthritis Rheum 1998;41:778–99.
[2] Felson DT. An update on the pathogenesis and epidemiology of osteoarthritis. Radiol Clin North Am 2004;42(1):1–9, v.
[3] Felson DT, Lawrence RC, Dieppe PA, et al. Osteoarthritis: new insights. Part 1: The disease and its risk factors. Ann Intern Med 2000;133(8):635–46.
[4] National Institute of Arthritis and Musculoskeletal and Skin Diseases. Arthritis prevalence rising as baby boomers grow older. 1998 NIH Press Release. Available at: www.niams.nih.gov/ne/press/1998/05_05.htm. Accessed June 2004.
[5] Lobbezzo-Scholte AM, De Leeuw JR, Steenks MH, et al. Diagnostic subgroups of craniomandibular disorders. Part I: Self-report data and clinical findings. J Orofac Pain 1995;9:24–36.
[6] Arthritis Foundation. The facts about arthritis. Available at: www.arthritis.org/resources/gettingstarted/default.asp. Accessed June 2004.
[7] Rugh JD, Solberg WK. Oral health status in the United States. Temporomandibular disorders. J Dent Educ 1985;49:389–404.
[8] Schiffman E, Fricton JR. Epidemiology of TMJ and craniofacial pain. In: Fricton JR, Kroening RJ, Hathaway KM, editors. TMJ and craniofacial pain: diagnosis and management. St. Louis (MO): Ishiaku Euro American; 1988. p. 1–10.
[9] DeKanter RJAM, Truin GJ, Burgersdijk RCW, et al. Prevalence in the Dutch adult population and a meta-analysis of signs and symptoms of temporomandibular disorders. J Dent Res 1993;72:1509–18.
[10] Dworkin SF, Huggins KH, Le Resche L, et al. Epidemiology of signs and symptoms in temporomandibular disorders: clinical signs in cases and controls. J Am Dent Assoc 1990;120:273–81.
[11] Huber NU, Hall EH. A comparison of the signs of temporomandibular dysfunction and occlusal discrepancies in symptom free populations of men and women. Oral Surg Oral Med Oral Pathol 1990;70:180–3.
[12] Matsuka Y, Yatani H, Kuboki T, et al. Temporomandibular disorders in the adult population of Okayama City, Japan. Cranio 1996;14(2):158–62.
[13] Widmalm SE, Westesson PL, Kim IK, et al. Temporomandibular joint pathology related to age, sex, and dentition in autopsy material. Oral Surg Oral Med Oral Pathol 1994;78:416–25.
[14] Peirera FJ Jr, Lundh H, Westesson PL. Morphologic changes in the temporomandibular joint in different age groups: an autopsy investigation. Oral Surg Oral Med Oral Pathol 1994;78:279–87.
[15] Bell WE. Temporomandibular disorders: classification, diagnosis, management. 3rd edition. Chicago: Year Book Medical Publishers; 1990.

[16] Boering G. Temporomandibular joint osteoarthritis: an analysis of 400 cases—a clinical and radiographic investigation [thesis]. Groningen (Netherlands): Department of Oral and Maxillofacial Surgery, University of Groningen; 1966 [reprinted 1994].
[17] De Leeuw R, Boering G, Stegenga B, et al. Symptoms of temporomandibular joint osteoarthritis and internal derangement 30 years after non-surgical treatment. J Craniomandibular Pract 1995;13:81–8.
[18] Kaminishi R. Metabolic factors of the TMJ. In: McNeill C., editor. Current controversies in temporomandibular joint disorders. Proceedings of the Craniomandibular Institute's 10th Annual Squaw Valley Winter Seminar. Chicago(IL): Quintessence Publishing; 1991. p. 57–9.
[19] Byers PD, Maroudas A, Oztop F, et al. Histological and biochemical studies on cartilage from osteoarthrotic femoral heads with special reference to surface characteristics. Connect Tissue Res 1977;5:41–9.
[20] Mankin HJ, Thrassher AZ. Water content and binding in normal and osteoarthritic human cartilage. J Bone Joint Surg 1975;557(A):76–80.
[21] Blaustein DI, Scapino RP. Remodeling of the TMJ disc and posterior attachment in disc displacement specimens in relation to glycosaminoglycan content. Plast Reconstr Surg 1986; 78:756–64.
[22] Sah RL, Chen AC, Chen SS, et al. Articular cartilage repair. In: Koopman W, editor. Arthritis and allied conditions—a textbook of rheumatology. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 2264–73.
[23] Nannmark U, Sennerby L, Haraldson T. Macroscopic, microscopic and radiologic assessment of the condylar part of the TMJ in elderly subjects. An autopsy study. Swed Dent J 1990;14(4):163–9.
[24] Nakayama Y, Narita T, Mori A, et al. The effects of age and sex on chondroitin sulfates in normal synovial fluid. Arthritis Rheum 2002;46(8):2105–8.
[25] DeGroot J, Verzijl N, Wenting-Van Wijk MJ, et al. Age-related decrease in susceptibility of human articular cartilage to matrix metalloproteinase–mediated degradation: the role of advanced glycation end products. Arthritis Rheum 2001;44(11):2562–71.
[26] Felson DT, Lawrence RC, Dieppe PA, et al. Osteoarthritis: new insights. Part 2: The disease and its risk factors. Ann Intern Med 2000;133(9):726–37.
[27] Dulcic N, Panduric J, Kraljevic S, et al. Frequency of internal derangement of the temporomandibular joint in elderly individuals. Eur J Med Res 2003;8(10):465–71.
[28] Gillette JA, Tarricone R. Economic evaluation of osteoarthritis treatment in Europe. Expert Opin Pharmacother 2003;4(3):327–41.
[29] Hiltunen K, Vehkalahti MM, Peltola JS, et al. A 5-year follow-up of occlusal status and radiographic findings in mandibular condyles of the elderly. Int J Prosthodont 2002;15(6): 539–43.
[30] Kopp S, Carlsson GE, Haraldson T, et al. Long-term effect of intra-articular injections of sodium hyaluronate and corticosteroid on temporomandibular joint arthritis. J Oral Maxillofac Surg 1987;45(11):929–35.
[31] Alstergren P, Appelgren A, Appelgren B, et al. The effect on joint fluid concentration of neuropeptide Y by intra-articular injection of glucocorticoid in temporomandibular joint arthritis. Acta Odontol Scand 1996;54(1):1–7.
[32] Vallon D, Akerman S, Nilner M, et al. Long-term follow-up of intra-articular injections into the temporomandibular joint in patients with rheumatoid arthritis. Swed Dent J 2002;26(4): 149–58.
[33] Huskisson EC, Donnelly S. Hyaluronic acid in the treatment of osteoarthritis of the knee. Rheumatology (Oxford) 1999;38(7):602–7.
[34] Wen DY. Intra-articular hyaluronic acid injections for knee osteoarthritis. Am Fam Physician 2000;62:565–70, 572.
[35] Bertolami CN, Gay T, Clark GT, et al. Use of sodium hyaluronate in treating temporomandibular joint disorders: a randomized, double-blind, placebo-controlled clinical trial. J Oral Maxillofac Surg 1993;51(3):232–42.

[36] Shi ZD, Yang F, Zhang JY, et al. [Randomized controlled trial of sodium hyaluronate for degenerative disorders of the temporomandibular joint.] Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi 2002;16(1):11–5 [in Chinese].
[37] Winocur E, Gavish A, Halachmi M, et al. Topical application of capsaicin for the treatment of localized pain in the temporomandibular joint area. J Orofac Pain 2000;14(1):31–6.
[38] Myrer JW, Feland JB, Fellingham GW. The effects of a topical analgesic and placebo in treatment of chronic knee pain. J Aging Phys Act 2004;12(2):199–213.
[39] Burch F, Codding C, Patel N, et al. Lidocaine patch 5% improves pain, stiffness, and physical function in osteoarthritis pain patients: a prospective, multicenter, open-label effectiveness trial. Osteoarthritis Cartilage 2004;12(3):253–5.
[40] Nitzan DW, Price A. The use of arthrocentesis for the treatment of osteoarthritic temporomandibular joints. J Oral Maxillofac Surg 2001;59(10):1154–9 [discussion: 1160].
[41] Emshoff R, Rudisch A. Determining predictor variables for treatment outcomes of arthrocentesis and hydraulic distention of the temporomandibular joint. J Oral Maxillofac Surg 2004;62(7):816–23.
[42] Gu ZY, Wu HL, Wu QL, et al. The effect of intra-articular irrigation injection therapy on osteoarthrosis of the temporomandibular joint. Chin J Dent Res 1998;1(3):44–8.
[43] Malemud CJ, Islam N, Haqqi M. Pathophysiological mechanisms in osteoarthritis lead to novel therapeutic strategies. Cells Tissues Organs 2003;174(1–2):34–48.
[44] Lozada C, Altman RD. Management of osteoarthritis. In: Koopman W, editor. Arthritis and allied conditions—a textbook of rheumatology. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 2246–58.
[45] Beary JF, Luggen ME. Osteoarthritis. In: Paget SA, Gibofsky A, Beary JF, editors. Manual of rheumatological and outpatient orthopedic disorders. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2000. p. 337–48.
[46] Jordan JM, Kraus VB, Hochberg MC. Genetics of osteoarthritis. Curr Rheumatol Rep 2004;6(1):7–13.
[47] Mevorach D, Paget SA. Rheumatoid arthritis. In: Paget SA, Gibofsky A, Beary JF, editors. Manual of rheumatological and outpatient orthopedic disorders. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2000. p. 193–228.
[48] Fox DA. Etiology and pathogenesis of rheumatoid arthritis. In: Koopman W, editor. Arthritis and allied conditions—a textbook of rheumatology. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 55.
[49] Hale LP, Haynes BF. Pathology of rheumatoid arthritis and associated disorders. In: Koopman W, editor. Arthritis and allied conditions—a textbook of rheumatology. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 56.
[50] Jasin H. Mechanisms of tissue damage in rheumatoid arthritis. In: Koopman W, editor. Arthritis and allied conditions—a textbook of rheumatology. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 57.
[51] Dolwick FM. Temporomandibular disorders. In: Koopman W, editor. Arthritis and allied conditions—a textbook of rheumatology. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 2019–25.
[52] Furst DE, Hillson J. Therapeutic approaches in the rheumatic diseases. In: Koopman W, editor. Arthritis and allied conditions—a textbook of rheumatology. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 671–703.
[53] Alhadaq A, Mao JJ. Tissue-engineered neogenesis of human-shaped mandibular condyle from rat mesenchymal stem cells. Dent Res 2003;82(12):951–6.