Magnetic Resonance Imaging Findings of the Temporomandibular Joint in Rheumatoid Arthritic and Asymptomatic Egyptian Population

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An accurate diagnosis of the temporomandibular joint status requires a thorough knowledge of the normal and abnormal anatomy of the joint as well as the mechanics of its movement (Al-Balkhi et al. 1992).

The temporomandibular joint (TMJ) is a complex articulation that is composed of a series of complicated osseous and soft tissue structures, each of which may be implicated in clinical syndromes. As such, these joints are responsible for a significant level of morbidity (Wessely and Young 2008).

The TMJ is a synovial joint consisting of mobile condyloid process of the mandible articulating with the squamous portion of the temporal bone (Hedge 2005). Both surfaces are covered by dense articular fibrocartilage. Each condyle articulates with a large surface area of temporal bone consisting of the articular fossa, articular eminence, and preglenoid plane. The TMJ functions uniquely in that the condyle both rotates within the fossa and translates anteriorly along the articular eminence. Because of the condyle’s ability to translate, the mandible can have a much higher maximal incisal opening than would be possible with rotation alone. The joint is thus referred to as “ginglymodiarthrodi”al”: combination of the terms ginglymoid (rotation) and arthroidial (translation) (Fletcher et al. 2004) (Fig.1).
Fig. (1): Sagittal section through the temporomandibular joint (Wessely and Young 2008).

The osseous structures centre around the mandibular condyle and its complementary articulating surface on the glenoid fossa of the temporal bone. The soft tissue elements include the articular disc and its various attachments, a multitude of delicate ligaments, muscular insertions points as well as synovial and capsular components (Wessely and Young 2008).

As mentioned before, the TMJ is a ginglymodiarthrodial (hinge and glide) articulation with some degree of diathrosis (free motion) formed by the mandibular condyle and glenoid fossa of the temporal bone (Fig.1). Within this synovial joint is a fibrous disk or meniscus that divides the joint into superior and inferior compartments that do not communicate unless disk integrity is compromised. The biconcave disk has three functional segments: a thick posterior band that is separated from the anterior band by the thin intermediate zone (Roth et al 2005).
Multiple ligamentous attachments provide disk stability. Posteriorly, the bilaminar zone attaches the disk and capsule to the condyle and temporal bone. Laterally, the disk is continuous with an unnamed ligament attaching to the neck of the condyle. The superior belly of the lateral pterygoid muscle inserts into the anterior portion of the disk (Hayt et al. 2000 and Roth et al. 2005). The bilaminar zone is also known as the retrodiscal tissue. The bilaminar zone is a vascular innervated tissue that plays an important role in allowing the condyle to move forward (Hedge 2005). The superior lamina of the retrodiscal tissue limits extreme translation, whereas the inferior lamina limits extreme rotation (Herb et al. 2006).

The disc completely divides the TMJ into two distinct compartments superior and inferior and, ordinarily, there is no connection between them. However, in the older population there can be a significant degree of degeneration in the region of the disc. This may result in partial or complete perforation of the disc, thus allowing access between the superior and inferior joint spaces. The disc has a lozenge or elongated oval shape and has a superior and inferior surface. The superior surface is concavo-convex from anterior to posterior to conform to the contours of the articular fossa and condyle. The inferior surface is moulded to the condyle of the mandible and is, therefore, concave (Fig.1) (Wessely and Young 2008).

The disc is believed to have several roles, such as, cushioning and distributing joint loads, promoting joint stability during chewing, facilitating lubrication and nourishment of the joint surfaces, preventing gross degenerative changes in the condyle and fossa, and promoting normal growth of the mandible (Hedge 2005).
The surrounding supporting ligamentous structures are the temporomandibular, sphenomandibular, and stylomandibular ligaments. The temporomandibular ligament provides lateral support extending from the zygomatic process of the temporal bone to the condylar neck. The sphenomandibular and stylomandibular ligaments provide medial support coursing from the spine of the sphenoid bone to the lingual of the mandibular foramen and from the styloid process to the mandibular ramus, respectively. No discrete ligaments are normally observed anteriorly or posteriorly around the joint (Fig. 2) (Roth et al 2005).

The masseter, medial pterygoid, lateral pterygoid, and temporalis muscles are the muscles of mastication. The masseter, medial pterygoid, and temporalis are primarily responsible for mandibular closure and bite force, whereas the lateral pterygoid and infrahyoid muscles are responsible for mandibular opening (Herb et al 2006).

The lateral pterygoid muscle (LPM) controls the opening of the mandible. The superior segment of this muscle attaches to the anterior portion of the disc, and the inferior segment attaches inferior to the condyle. As both segments contract, the condyle translates anteriorly along the articular eminence, and the disc remains interposed between the condyle and the temporal bone at all points of translation (Herb et al 2006).

The TMJ receives its vascular supply from the superficial temporal, maxillary and masseteric arteries. Innervation of the joint is provided mainly by the auriculotemporal nerve and, to a lesser extent, the masseteric and posterior deep temporal nerves. The production of synovial fluid is also under a certain amount of neuronal control (Herb et al 2006).
Fig.(2): Diagram illustrating the anatomy of the ligaments related to TMJ (Bixby 2008).
Temporomandibular Joint Disorders (TMD)

Temporomandibular joint disorders are defined by the American Association of Orofacial Pain (AAOP) as: A collective term embracing a number of clinical problems that involve the masticatory musculature, the temporomandibular joint and associated structures, or both (McNeill 1990).

Signs and symptoms of Temporomandibular Disorders were first recognized by Costen in 1934 and since then a plethora of terms have been used, somewhat interchangeably, to describe TMD. These include Costen’s syndrome, temporomandibular joint dysfunction syndrome (Shore 1959), pain dysfunction syndrome (Gray et al 1994) and facial arthromyalgia (Madland et al 2000).

A temporomandibular joint disorder is a common disorder presenting with multiple symptoms of clinical joint dysfunction (Landes et al 2007). Signs and symptoms of TMD have a higher incidence in the general population {20–75%} than the proportion of the population who present for treatment {2–4%} (Gray et al 1994).

The main symptoms of TMD are: limited mandibular range of motion, pain on muscle palpation or mandibular movements or both, and joint sounds. These problems may occur simultaneously, separately, intermittently, or may become chronic (Schmitter et al 2004).
The etiology of temporomandibular joint disorders is considered to be multifactorial, including habits and parafunction (Godard et al 1999).

Biomechanical factors such as occlusal and masticatory dysfunction, loss of posterior teeth, unilateral chewing patterns, and bruxism have been proposed to be involved in the initiation and progression of degenerative TMJ disease through absolute or relative overloading of joint structures (Haskin et al 1995).

There are several variations from normal mastication. Among them, unilateral mastication is chewing predominantly on a preferred side of the dentition and hardly on the non preferred side. Continual unilateral mastication may alter the coordination of masticatory muscles. It produces strain in a single muscle unlike bilateral mastication and contributes to severe damage to the stomatognathic system (Abekura et al 1995).

Okeson in 1996 reported that some non-functional movements of the mandible (bruxing) and tooth-clenching habits are clinically associated with a variety of jaw muscle symptoms and are associated less clearly with internal joint disk derangements. The causes of TMD range from traumatic injury to immune-mediated systemic disease to neoplastic growths to incompletely understood neurobiologic mechanisms (Milam and Schmitz 1998 and Kopp 1998).

Less common but better recognized causes of TMD include; wide range of direct injuries, such as fractures of the mandibular condyle, systemic diseases, such as immune-mediated arthritis, growth disturbances, and tumors (Goldstein 1999).
Parafunctional habits are very common and have been considered as possible causes for temporomandibular joint disorders (*Rugh and Ohrbach 1988; Okeson 1993 and Gavish et al 2000*). Parafunctional activities are usually harmless, until the exerted forces exceed the structural tolerance (*Okeson 1993*).

The most common oral habit in adolescent girls was gum chewing in a study done in 2000. Intensive gum chewing (more than three hours per day) had a potential detrimental effect on the masticatory muscles, as well as on the temporomandibular joint (*Gavish et al 2000*).

TMD and bruxism patients may present many other additional oral jaw habits which may combine to increase masticatory muscle activity, thus leading to TMD signs and symptoms (*Molina et al 2001*).

A study was performed in 2002 and concluded that statistically significant risk factors for precipitating TMD symptoms include; lip biting (for TMJ pain) and trauma (for limitation of mouth opening) while statistically significant risk factors for perpetuating TMD symptoms were female sex for TMJ pain and noise (*Yatani et al 2002*).

Patients with unilateral chewing and bruxism tend to have more complicated symptoms. In contrast, among the patients without unilateral chewing and bruxism, the number with multiple symptoms decreases (*Fujita et al 2003*).

TMJ may be affected by inflammatory, traumatic, infectious, congenital, developmental, and neoplastic diseases, as seen in other joints. However, the most common affliction of the TMJ and masticatory apparatus is a group of functional disorders with associated pain that occurs predominantly in women and was previously known as the TMJ
pain dysfunction syndrome. Since 1978, there have been substantial changes in the study of etiologic factors, pathophysiology, diagnosis, and management of what are now called temporomandibular disorders (Laskin 2008).

Advances in the understanding of joint biomechanics, neuromuscular physiology, autoimmune and musculoskeletal disorders, and pain mechanisms have led to changes in our understanding of the cause of temporomandibular disorders. The cause is now considered multifactorial, with biologic, behavioral, environmental, social, emotional, and cognitive factors, alone or in combination, contributing to the development of signs and symptoms of temporomandibular disorders (de Leeuw 2008).

Suvinen et al in 2005 found that there is a great deal of inter-individual variability in the signs and symptoms of TMD but they can be divided into six broad groups: (1) joint noises -clicking, crepitus (grinding); (2) locking - open (inability to close fully), closed (inability to open fully); (3) pain - in head, neck and shoulders; (4) muscular tenderness - in face, neck and shoulders; (5) ear complaints - otalgia, tinnitus; (6) psychosocial effects.

Temporomandibular joint disorders are most commonly reported in young to middle-aged adults (20 to 50 years of age). The female-to-male ratio of patients seeking care has been reported as ranging from 3:1 to as high as 9:1 (Huber and Hall 1990).
Despite the high prevalence of signs and symptoms of temporomandibular joint disorders, only 5 to 10% of those with symptoms require treatment, because about 40% of patient’s symptoms are resolved spontaneously (Solberg 1983 and Levitt and McKinney 1994).

**Classification of Temporomandibular Joint Disorders (de Leeuw 2008):**

TMDs were classified according to Leeuw 2008 into the following disorders:

1. **Articular disorders:**

   1. **Congenital or developmental disorders:**

      These disorders include; first and second branchial arch disorders: hemi-facial microsomia, Treacher Collins syndrome, bilateral facial microsomia, Condylar hyperplasia and Idiopathic condylar resorption (condylysis).

   2. **Disk-derangement disorders:**

      These disorders include; disk displacement with reduction, disc displacement without reduction (closed lock) and perforation.

   3. **Degenerative joint disorders:**

      These disorders include; inflammatory disorders as capsulitis, synovitis, polyarthritides (rheumatoid arthritis, psoriatic arthritis, ankylosing spondylitis, Reiter’s syndrome, gout). In addition, it also includes non inflammatory as osteoarthritis.
4- **Traumatic disorders:**

These disorders include; contusions, intracapsular hemorrhage, and fractures.

5- **TMJ hypermobility disorders:**

These disorders include; joint laxity, subluxation and dislocation.

6- **TMJ hypomobility disorders:**

These disorders include; trismus, postradiation therapy fibrosis, ankylosis: true ankylosis (bony or fibro-osseous), pseudoankylosis, infection and neoplasia.

**II-Masticatory muscle disorders:**

Myofascial pain disorder, local myalgia, myositis, myospasm, myofibrotic contracture, Neoplasia. The most common forms of temporomandibular disorders seen by the primary care physician are the myofascial pain disorder, intra-articular disk derangement disorders and osteoarthritis (*Scrivani et al 2008*).

- **Myofascial Pain Disorder (MFPD)**

Myofascial pain disorder of the masticatory muscle system is the most common of all temporomandibular disorders. The vast majority of patients present with facial pain, limitation of jaw motion, muscle tenderness and stiffness, along with any number of associated symptoms in the head, face, and neck region (*Scrivaniet al 2008*).
Myofascial pain of the masticatory muscles is more frequently induced by stress-related parafunctional habits (i.e., clenching and grinding) and rarely by mechanical causes such as occlusal prematurities or high dental restorations. Myofascial pain disorder, although considered to be muscular disorders, are thought to possibly play a causative role in degenerative disease of the TMJ (Herb et al 2006).

One must differentiate muscular from joint conditions in order to appropriately treat the patient. At the same time, the clinician must understand the role of myofascial pain disorder within the spectrum of TMDs. It has been reported that approximately 50% of all TMDs are masticatory myalgias or painful masticatory muscle disorders (Stohler 2000 and Herb et al 2006).

Imaging studies of the TMJ usually show no evidence of anatomic pathology. Patients with myofascial pain disorder generally respond to the simple, noninvasive treatments (Scrivani et al., 2008).

❖ Intra-articular Disk Derangement Disorder

Disk derangement disorder is defined as a temporomandibular joint disorder resulting from displacement of the TMJ disk from its normal position or from deformation of the disk. This may lead to synovitis, pain, and limitation of motion (de Leeuw 2008).
Internal Derangements (ID) may include anterior displacement of the disk, with reduction (ADDR) or without reduction (ADDWR) \textit{(de Leeuw 2008)}. Anterior displacement with reduction is defined as disk displacement in the closed-mouth position that reduces (with a click) to the normal relationship at some time during opening. Reduction implies that to some extent the disk is gliding normally, with opening and translational movement. In these circumstances, the patient reports a click with a variable amount of pain on opening. Often, patients have no pain with this condition. The mandible deviates to the affected side on opening until the click occurs and then returns to the midline. This situation may worsen, and there may be intermittent locking of the disk \textit{(Scrivani et al 2008)}.

Intermittent locking may progress over time to anterior disk displacement without reduction (closed lock) \textit{(de Leeuw 2008)}. This implies that the dislocated disk acts as a mechanical obstruction to the opening and translation of the condyle. These patients have a marked decrease in mandibular opening on the affected side and a variable amount of pain. It feels to them as if there is a mechanical obstruction to opening in the joint. Maximal opening may be limited to 20 to 25 mm (the normal range of maximal interincisal opening ranges from 35 to 55 mm, with a mean of 40 to 43 mm), with restricted movement to the contralateral side. There may also be a history of clicking with intermittent locking \textit{(Dimitroulis 2005)}.
The adaptive capacity of the TMJ is not infinite; some individuals are capable of mounting an adaptive response to an articular disc displacement; other individuals may not adapt to these structural derangements, and a progressive Degenerative Joint Disease (DJD) may result. Factors considered to compromise the adaptive response include age, sex, stress, and illness (Milam 2000).

The incidence of Anterior Disc Displacement (ADD) is unknown. Numerous radiographic, clinical, and cadaveric studies of asymptomatic subjects have shown rates up to 30% (Westesson et al 1989). The clinical significance of this finding remains uncertain. Therapy is indicated if pain and significant limitation in range of motion are present (Herb et al 2006).

**Osteoarthritis**

It is a degenerative disease of movable joints (Altman 1991). Osteoarthritis (OA), also known as “Degenerative Joint Disease”, it has a multifactorial pathogenesis including biomechanical, biochemical, inflammatory, and immunologic insults. Excessive and repetitive mechanical stress has been implicated (Israel et al 2006).

Osteoarthritis is classified as primary (no known predisposing factors) or secondary (associated with known abnormalities or injuries). Primary OA symptoms begin in older age than secondary OA (Herb et al 2006).
The patient reports pain on moving the mandible, limited motion, deviation of the jaw to the affected side and may be acute tenderness to palpation of joint. Joint sounds are described as grinding, or crunching, but not as clicking or popping (Limchaichana et al 2006).

There is a strong predilection for the disease among women in their third or fourth decade. Only a few patients have generalized osteoarthritis. The natural course of the disease suggests that the pain and limitation may “burn themselves out” after as little as several months in some patients (Israel et al 1998; Dimitroulis 2005 and de Leeuw 2008). The majority can be kept comfortable with the use of noninvasive techniques until remission. In the acute phase, patients may require intra-articular injection of a long-acting corticosteroid such as beclomethasone or hyaluronic acid (Bjørnland et al 2007).

**Clinical Diagnosis of Temporomandibular Joint Disorders**

Establishment of an accurate diagnosis is necessary for effective management of TMDs. The difficulty lies not in creating a distinction between articular and muscular disorders, but in the interrelation of the two entities; in which joint disorders may lead to muscle dysfunction, and muscle disorders may lead to joint dysfunction (Herb et al 2006).
A detailed history, head and neck evaluation, and general physical examination when indicated, are essential (Murphy et al 2000).

Data are better collected by printed questionnaire (Ozan et al 2007). Due to the need of simpler assessment procedures that could be widely applicable and standardize research samples involving TMD patients, questionnaires have been created to address the main clinical TMD findings and assign clinical indexes for patient classification in terms of severity levels (Bevilaqua-Grossi et al 2006).

The following information should be part of a comprehensive history: chief complaints (presence of pain in temporomandibular joint, head, back, and while chewing, parafunctional habits, movement limitations and joint clicking), history of present illness, past medical and dental history, review of the systems (systemic conditions that can enhance or cause the pain sensation), family history (the patient should report if some relative presents the same conditions because some disorders are genetically predisposed), presence of parafunctional habits (the habits most frequently found in TMD patients are clenching and grinding, nail biting and poor posture due to occupational activities should also be recorded) and psychosocial history (Bevilaqua-Grossi et al 2006 and Conti et al 2007).