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Concepts of TMD Etiology: Effects on Diagnosis and Treatment

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Abstract: *Temporomandibular disorders is a collective term used to describe a number of related disorders affecting the temporomandibular joints, masticatory muscles, and associated structures, all of which have common symptoms such as pain and limited mouth opening. 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. The purpose of this article is to review the basic anatomy, function and disturbances of the temporomandibular joint, its diagnosis, differential diagnosis and clinical management.*
Key words: *TMD, Temporomandibular disorders, headache, facial pain, diagnosis*

I. Introduction

Temporomandibular disorders is a collective term used to describe a number of related disorders affecting the temporomandibular joints, masticatory muscles, and associated structures, all of which have common symptoms such as pain and limited mouth opening. [1]General practitioners will sometimes see patients who present with either persistent or recurrent chronic facial pain. Having eliminated the possibility of headache or ear or sinus problems, the next step is to consider the possibility of temporomandibular joint pain and dysfunction, particularly if the pain is accompanied by clicking jaw joints and limited mouth opening.[1]

The term "TMJ pain" varies greatly in meaning among clinicians, patients, and the general population. Historically, symptom-based classification of the disorder has been problematic. As stated by Laskin [2], the difficulty began with the introduction of a "TMJ syndrome."[2]

Then clinicians erroneously grouped a "variety of etiologically unrelated conditions into one diagnostic category based on the fact that they produced similar signs and symptoms," and this led to "one diagnosis equals one treatment." Only later was it recognized that many of these patients suffered from musclerelated conditions. The terms myofascial pain (MFP) and myofascial pain and dysfunction (MPD) evolved 5 and "TMJ disorders" became "TMDs."[3]

According to the American Academy of Orofacial Pain temporomandibular disorders (TMD) are defined as a collective term embracing a number of clinical problems that involve the masticatory muscles, the temporomandibular joint and associated structures, or both".[4] They are considered to be a subclassification of musculoskeletal disorders and typically run a recurrent or chronic course, with a substantial fluctuation of TMD signs and symptoms over time.[5] Common signs and symptoms of TMD are clicking noises in the temporomandibular joint (TMJ), limited jaw opening capacity, deviations in the movement patterns of the mandible and masticatory muscle and/or TMJ pain in the face.[5,6]

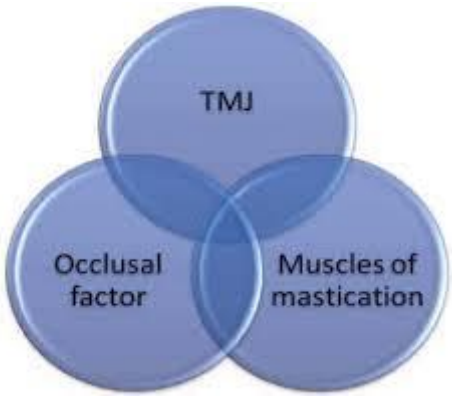

Figure 1: Theories related to physical factors can be summarized by this model

After initial studies in 1934, Costen proposed that patients suffering from auricular pain, pressure and fullness in the ear and swallowing problems (Costen syndrome) improve by occlusion correction.[7] In the 1960s, the quality of clinical examinations and scientific studies improved; the importance of occlusion in TMD etiology in 1970 was studied. Methods including tomography, arthrography, computed tomography (CT) scan and magnetic resonance imaging (MRI) lead to improvements in examination of intracapsular structures.[6]

Today the information in this field show that patients with orofacial pains may suffer from disorders such as systemic, neuromuscular, vascular, and mental or a combination of disorders associated with TMD; some headway in pain mechanism, neurology, physiology and neuoropharmacology have been made. Different studies demonstrated that TMD treatmenthas changed based on the diagnosis of the etiology and stage of the disorder. [1,2,5,6] **[Figure**1]

The concept of TMDs as part of the constellation of musculoskeletal disorders, rather than some special kind of dental condition, is relatively recent. In 1918, Prentiss initiated interest in the dental community when he suggested that the development of "TMJ problems" was due to the following process: "When the teeth are extracted, the condyle is pulled upward by the powerful musculature and pressure on the meniscus results in atrophy." This was soon followed by several articles from other dentists, who emphasized missing teeth and lost vertical dimension leading to displacement of the mandible as the cause of the signs and symptoms displayed by patients with TMD.[5,6,7]

Patients with temporomandibular disorders most frequently present with pain, limited or asymmetric mandibular motion, and TMJ sounds.[3,4]The pain or discomfort is often localized to the jaw, TMJ, and muscles of mastication. Common associated symptoms include ear pain and stuffiness, tinnitus, dizziness, neck pain, and headache. In some cases, the onset is acute and symptoms are mild and self-limiting. In other patients, a chronic temporomandibular disorder develops, with persistent pain and physical, behavioral, psychological, and psychosocial symptoms similar to those of patients with chronic pain syndromes in other areas of the body5-7 (e.g., arthritis, low back pain, chronic headache, fibromyalgia, and chronic regional pain syndrome), all requiring a coordinated interdisciplinary diagnostic and treatment approach.[8]

Recently, distinctions have been made between subgroups of TMD patients. The subclassification of TMD consists of two primary diagnostic categories: arthrogenous and myogenous. These classifications are not always clear, and there can be a considerable overlap or progression from one so-called syndrome to another. [9]In 1990, the American Academy of Orofacial Pain (AAOP) established the first well defined diagnostic classification for TMD, which was revised in 1993.[10] Further, the AAOP published an updated diagnostic classification in 1996 The myogenous classification is often further subdivided into muscular hyperarousal due to stress and muscular abnormality associated with parafunctional oral habits (e.g., bruxism), and the arthrogenous category is subdivided on the basis of specific structural abnormalities (e.g. internal derangement of the temporomandibular joint or degenerative disease).[5,6]

The research diagnostic criteria (RDC) developed by Dworkin and LeResche (1992), established a dual diagnosis that recognizes not only the physical conditions (axis I), including muscle disorders, disc displacements and other types of joint conditions that may contribute to the pain disorder, but also the psychosocial issues (axis II) that contribute to the suffering, pain behavior, and disability associated with the patient's pain experience. [11,12]This dual-axis classification approach has recently been incorporated in a diagnostic scheme not only for TMD but for all orofacial pain disorders .[5] The RDC have been shown to be reliable for diagnosing TMD in U.S. and Swedish populations . In a recent study, however, the RDC have been shown to provide insufficient reliability for the determination of arthrogenous TMD . [13]

This article will discuss the signs and symptoms, diagnosis, and management of TMD.

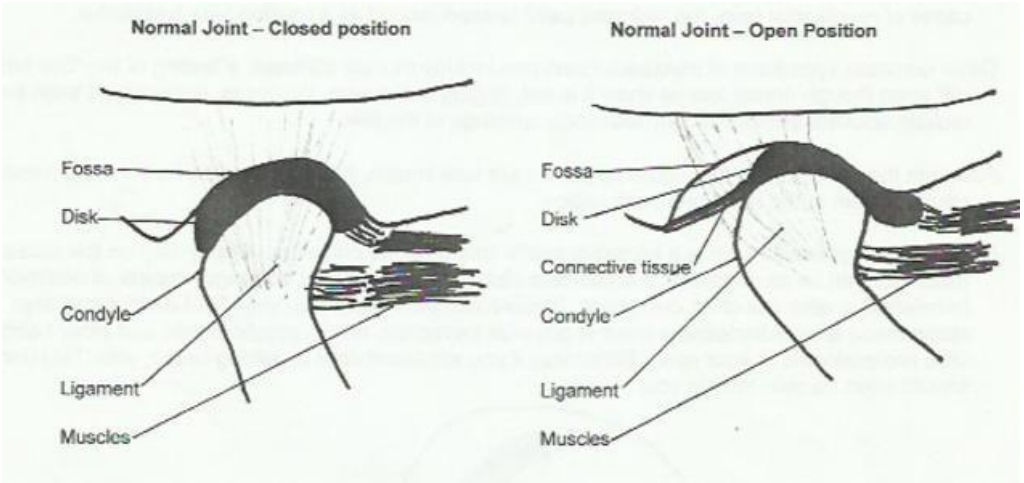

Figure 2; Anatomy of the TMJ

II. TMJ Anatomy

Temporomandibular joint is the junction site of the mandibular condyle to skull base or glenoid fossa of the temporal bone. A disc separates the two bones. The part of the disc which is in contact with mandibular condyle bone consists of fibrous connective tissue without any nerve or vessel.[14] This joint is a compound

one. The disc is divided into three parts, in sagittal view: anterior, posterior and middle. The middle zone is the thinnest part. The disc becomes thicker in the anterior and posterior parts. In coronal view, the medial part of the disc is thicker than the lateral part. [1]

Disc shape is determined by condyle morphology and mandibular fossa. The disc may become displaced or destroyed via degenerative forces. In the posterior part, the disc is attached to a loose connective tissue of nerve and vessels named retrodiscal tissue. In the superior posterior part, it is attached to a connective tissue full of elastic bands named superior retrodiscal layer or bilaminary zone.[15] This tissue connects the disc to the tympanic bone posteriorly. Below this, there is the inferior retrodiscal layer which connects the inferior border of the posterior edge of the disc to the posterior part of condyle joint surface. Inferior disc layer and superior retrodiscal tissue are made of collagen and elastic fibers, respectively.**[Figure**2] Anteriorly to the disc, superior and inferior adhesions of it connect to the capsular ligament. Both of these adhesions are made of collagen fibers. Between the capsular ligaments, the disc is adherent to fibers of the superior lateral pterygoid muscle. The disc adheres to the capsular ligament, not only anteroposteriorly, but also mediolaterally. The joint is divided into two separate and distinct spaces. The superior space is located between the glenoid fossa and superior part of the disc; the inferior disc space lies between the

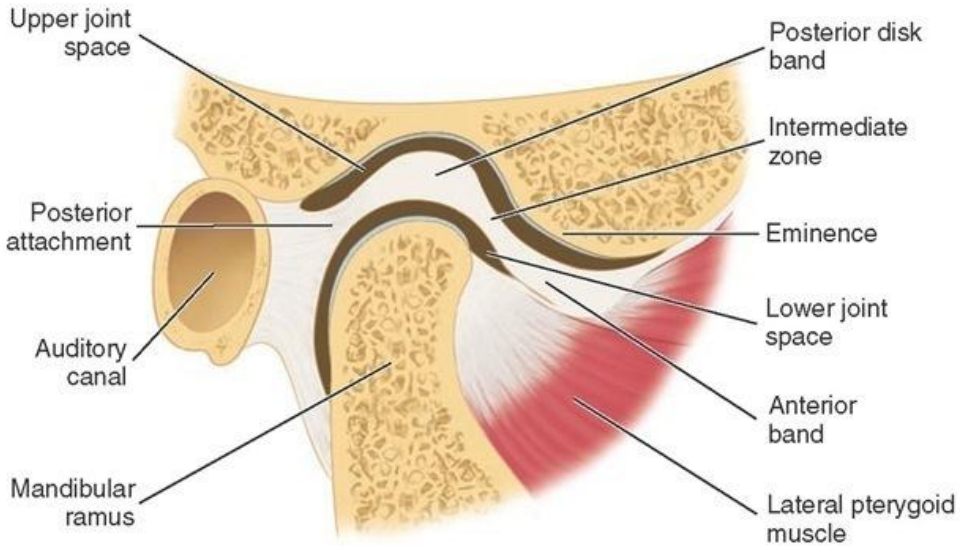

Figure 3; Articular disk and associated structures

Disc and condyle. Internal surfaces of superior and inferior spaces are lined with special endothelial cells which secrete synovial fluid.[14,15,16,17] **This fluid has two functions: 1-** Molecular transport and metabolism and **2-**Lubrication of joint surfaces; the fluid is secreted on the joint surfaces under pressure and results in friction reduction. During function, forces entering to the joint surfaces lead to movement of this fluid into intrajoint tissues. In coronal view, the condyle has a medial and lateral pole; the medial pole is thicker than the lateral one. **[Figure**3]

The TMJ is supported by three major and two minor ligaments. [16,17]

Major ligaments are:
1. Collateral ligaments
2. Capsular ligament
3. Temporomandibular ligament

Minor ligaments are:
1. Sphenomandibular ligament
2. Stylomandibular ligament

III. Epidimiology

The incidence and prevalence of TMDs in different population groups has been the subject of a large number of epidemiologic studies. The results revealed that about 40-75% of cases in general adult population (non-patients) show at least one sign of articular dysfunction (noise, disturbances of mandibular movements, etc.) and in about 33% of cases of those subjects there is at least one symptom of dysfunction (facial pain,

articular pain, etc.) [18]. Some signs are relatively prevalent in healthy general population. Namely, noise or asymmetric mouth opening is present in about 50% of individuals [19]. Other symptoms are very rare, such as difficulty in mouth opening which is present in only about 5% of the cases [17]. In general population, articular disorders are present in 19% of individuals, muscular forms of disorders in 23% and the combination of the two in 27% of individuals [18]. TMDs affect all age groups [19]. Signs and symptoms of TMDs have been noted in young individuals and adolescents but the prevalence is lower than in adults [20,21]. Nevertheless, in general population the symptoms are most commonly pronounced between the ages of 17 and 30, while in patients the symptoms are more pronounced between the ages of 20 and 40 [20]. Children and adolescents rarely complain of any symptoms although during their lifetime they exhibit an increase in the number of signs of TMDs. Patients over the age of 60 also rarely complain of the symptoms of TMDs [21,22].

The prevalence of symptoms is evenly distributed between men and women [20,21], although some authors report that the prevalence of TMDs in women is four times higher than in men [20]. When the symptoms are separately and independently evaluated, it has been noted that women more frequently suffer from headaches, clicking in temporomandibular joints, and sensitivity of joints and muscles. Women more frequently (3 to 9 times) ask for medical assistance which is difficult to substantiate by clinical findings since women and men do not differ in sensitivity and reaction to pain [20]. A possible explanation lies in the fact that women are more health conscious and quicker in seeking medical assistance. [21]

TMDs in general population, the prevalence of complaints is very low [22]. Only about 3.6-7% of the population needs treatment [14,15]. Prevalence of these disorders cannot be entirely and correctly evaluated due to a lack of generally accepted classification norms and diagnostic criteria. However, different researchers used a combination of signs and symptoms to indirectly determine the prevalence of TMDs thus reporting that approximately 26-31% of patients with articular changes and about 30-33% of patients with muscular changes need treatment [20,21].

IV. Etiology Of Tmds

The aetiology of the most common types of temporomandibular disorders is complex and is still largely unresolved. Malocclusion and trauma—whether acute, such as after an assault, or chronic and repetitive, such as tooth grinding or clenching—are often cited as possible causes.However, there is a clear lack of substantial evidence. Psychogenic factors have also been implicated, but, like trauma and malocclusion, these are often considered as exacerbating factors rather than the primary cause of temporomandibular disorders.[1,2,23] It is well established that very few patients with malocclusion, mandibular trauma, or psychogenic related illnesses actually go on to develop temporomandibular pain and dysfunction.8 Hence, there is speculation that only some patients who are vulnerable to temporomandibular disorders will develop pain and dysfunction after an exacerbating event such as trauma.[23]

However, the inherent features that may help identify those patients who are especially susceptible to tempo- romandibular disorders remain unknown.[23,24,25]

4.1. General aspects

De Boever (1979) reported five different etiologic theories of TMD, i.e. mechanical displacement theory, neuromuscular theory, psychophysiological theory, muscular theory and psychological theory.[26] At present, TMD are considered not as a single entity but as comprising several diseases of varying etiology and pathology, and controversy still exists because of limited knowledge regarding the etiology and natural history of the course of TMD.[10,11] General factors, such as impaired health, general joint and muscle diseases, psychological and psychosocial factors, and local influences such as occlusal disturbances, parafunctional activities, i.e. bruxism, and traumas, can affect the condition of the stomatognathic system [5,6]At present, the role of different factors in TMD is still unclear. In addition to studies with most commonly used patient samples, where biases such as access to providers and care-seeking behavior may have an effect on the results, additional population-based studies are needed to clarify the heterogenous factors related to TMD.[26,27,28,29]

4.2. TMD and generalized pain

Several studies with patient samples have found a significant overlap between TMD and pain conditions in other parts of the body .[30] Especially patients with masticatory muscle problems have complaints beyond the masticatory system, mostly in the head, neck and back areas.[31] In addition, high rates of comorbidity between myogenous facial pain and fibromyalgia have been noted in several studies.[32] In addition to these studies with patient samples, however, population-based studies are needed to describe the association between TMD and other pain conditions.

It has been stated that women are more likely to have multiple symptoms than men , and that muscular TMDs can be part of a generalized pain syndrome more commonly among women than among men . [33] This

generalized pain condition has been suggested to associate with psychological disturbances, especially with depression and somatization. [34]

4.3. Hereditary factors

Michalowicz et al. evaluated the hypothesis that signs and symptoms of TMD may be hereditary. They collected information through questionnaires from 494 monozygotic and dizygotic twins. Monozygotic twins showed no significant similarities with dizygotic twins, and monozygotic twins who grew up together showed similar characteristics when compared to each other. The authors conclude that genetic factors and the family environment exert no relevant effect upon the presence of symptoms and signs of the TMJ.[35]

4. 4.Hormonal factors

Signs and symptoms of TMD are four times more common among women, who seek specialized treatment for this disease three times more frequently than men. Despite the fact that the low prevalence of TMD in men has not been completely elucidated yet, the presence of higher testosterone levels may be a plausible explanation [36].

There is the hypothesis that the presence of estrogen receptors in women's TMJ changes metabolic functions increasing ligament laxity. Estrogen also increases susceptibility to painful stimuli by modulating the limbic system. Although researchers do not share the same opinion, studies in humans have shown that painful symptoms increase by 30% among patients on menopause treatment with estrogen replacement therapy and by 20% in women using oral contraceptives. [37]

Polymorphism in the estrogen receptor has been shown to be correlated to the intensity of pain, facial axis angle and mandibular body length in patients who suffer from TMJ osteoarthritis. However, despite these studies, until recently no direct evidence has been found that links female reproductive hormones to TMJ disease or that defines the mechanisms by which these hormones may cause TMJ disease. A recent study has shown that estrogens and relaxin could contribute to the degeneration of cartilage homeostasis by disrupting TMJ and inducing activation of metalloproteinases (MMP) that degrade cartilage matrix macromolecules (collagen and proteoglycans). [38,39,40]

4.5 Occlusal factors

Occlusion is the first and probably the most controversial etiologic factor of TMD. Costen was the one who first established with certainty the involvement of occlusion in the development of TMD. Nowadays, most researchers include occlusion among all the factors related to TMD, having a possible role in both susceptibility and onset or perpetuation of TMD.[4,5,7,40]

Pullinger and Seligman applied multiple factor analysis, which indicated the low correlation of occlusion to temporomandibular disorders. In this research, they compared occlusal characteristics in patients with symptoms of TMD with a group of patients without symptoms of TMD. The authors concluded that malocclusion ccould act as a co-factor in the etiology of TMD and some occlusal features might be consequences of the disease rather than initiating factors. They estimated that occlusal factors contributed about 10-20% to the total spectrum of etiological factors in TMD [41]. On the other hand, Rammelsberg offered a review of the etiopathogenic model of TMD development wherein high abrasion and insufficient restorative procedure on posterior teeth are risk factors causing occlusal instability [42].

According to the literature, the existing data cannot determine the exact role of occlusal factors in temporomandibular joint disorders. Conversely, in the meta-analysis conducted by Koh et al. [43], the authors concluded that there was insufficient data on the treatment or prophylaxis of TMD by occlusal rebalancing.

With regard to the distribution of occlusal contacts, the symmetry of their intensity rather than the symmetry of their number in the posterior occlusion is more important for temporomandibular function. [44]

Improper occlusions due to dental malpositions, untreated or improperly treated edentulism are pathological states of temporomandibular complex, but they are not considered the main etiological factors of TMD [45]. Badel et al. [46] have identified a low incidence of certain variables of malocclusion (unilateral open bite, negative overjet, unilateral cross bite in men, and edge-to-edge bite in women) with signs or symptoms of TMD. In the same study, they found a significantly higher prevalence of hyperbalance and interference contacts in asymptomatic patients compared to TMD patients. No difference was found between Angle's classes in patients with TMD and asymptomatic individuals.[44,43,46]

The importance of occlusal interferences is perceived differently based on the etiopathogenesis of TMD. Le Bell et al. [47] found that artificial interferences do not stimulate the development of dysfunctional symptoms in healthy subjects, who adapt successfully to them. In patients with medical histories of TMD, artificial interferences enhance clinical symptoms.[25]

Posterior cross-bite is a cause of asymmetric muscle functioning, but no certain correlation with TMD has been determined as yet [44,45].

The presence of mediotrusive interferences is considered by some authors to be a predisposing factor for disk displacement [46,47], whereas others suggest that they can exert a protective action [18].The presence of an anterior open-bite can be considered a consequence of articular remodeling [19] rather than the cause [48]. Condylar position may also play a significant role in the etiopathogenesis of TMJ disorders [49,50]. A recent study conducted by Padala et al. [51] evaluated the relationship between condylar position and centric occlusion-centric relation discrepancy and presence of signs and symptoms of TMD. The findings in this study indicate that recording and evaluating the centric relation-centric occlusion discrepancy in individuals with TMD may reveal significant dental inter-arch discrepancies and condylar displacements of significant magnitude. [51]

Debates over occlusal characteristics have influenced and limited therapeutic options for TMD, but recent research works have highlighted the multifactorial etiology of the disease and reduced the importance of occlusion as a major etiological factor of TMD [49.49,50].

4.6.Traumas

TMD have a history of head or cervical trauma. Patients who have undergone surgical treatment of TMD have reported extrinsic traumas, i.e. car accidents and blows in the face etc., preceding their TMD symptoms . It has also been found that patients with trauma history present with signs and symptoms of TMD .[52]

The association between traumas and TMD has also been shown epidemiologically

In contrast, a population-based study of Locker & Slade (1988) found no association between trauma and signs or symptoms of TMD. Additionally, a critical review by Ferrari & Leonard (1998) revealed no substantial theory of mechanical TMJ injury to be connected with TMD.[54,55]

4.7.Bruxism

Bruxism is a psychophysiological disorder that can be defined as diurnal or nocturnal tooth contact parafunctional activity, such as clenching and grinding . [56]Sleep bruxism has been defined by the American Sleep Disorders Association (ASDA) in its International Classification as a "stereotyped movement disorder characterized by grinding or clenching of the teeth during sleep" .[57] The prevalence of bruxism in the general population ranges from 8 to 21 % when assessed by a questionnaire, and from 48 to 58 % when clinical oral examination is employed. There are studies in which bruxism and other parafunctional habits are associated with head and facial pain .[58,59]

The etiology of bruxism is unclear. Bruxism has been suggested to be a multifactorial psychosomatic phenomenon . Bruxers are suggested to have increased levels of stress and tension, disturbed sleep, and depression . At present, bruxism is considered a phenomenon of centrally mediated neurologic activity related to sleep disorders , and it has also been suggested to be a link between autonomic regulation of circulation and rhythmic activation of masticatory muscles, especially when associated with body movements during sleep .[60,61]

4.8 .Psychological factors

The importance of psychological factors has also been emphasized in TMD.[62] They are thought to have a role in the cause or maintenance of TMD and may predispose the condition to chronicity On the other hand, it has been stated that psychological disturbances may be a direct consequence of pain-related life events in TMD patients.[63] Subcategorization of the patients into diagnostic subgroups of TMD suggests that myogenous patients may have more psychological difficulties than patients with arthrogenous TMD.[64]

4.8 .1.Somatization; Somatization is defined as a tendency to experience and communicate somatic distress in response to psychosocial stress and to seek medical help for it.

Somatoform disorder is a condition in which the patient reports somatic complaints, yet no physical evidence of organic disease is present Somatization has been connected with frequent use of health services and about 20 % of frequent attenders have been classified as chronically somatizing patients Somatizing patients have a negative perception of their health and often have psychosocial difficulties substantial distress and show enhanced sensitivity to normal physical sensations In addition, they are characterized by abnormal illness behavior.[65]

TMD patients have been found to have increased scores in somatization. It has been suggested that especially patients with masticatory muscle pain may be more prone to report symptoms as compared to normal controls and are likely to be more sensitive to painful stimuli[66], although this is argued by the data of Carlson et al. (1998)[20].

4.8.2. Alexithymia;The concept of alexithymia was first introduced by Sifneos in 1972 to describe psychological characteristics of patients with psychosomatic diseases Alexithymia (from the Greek a for lack, lexis for word, and thymos for feeling) means literally "no words for feelings". It denotes a deficit in the ability to differentiate emotional states from physical ones and to identify and describe one's feelings, as well as a preference for externally oriented, utilitarian thinking rather than fantasy or introspection.[67]

Alexithymia Scale (TAS) and its modified 20-item version, the TAS-20 .[68,69] In Finnish population, the prevalence of alexithymia has been reported to be 10% among women and 17 % among men. [70]
Many different theories have been presented concerning the etiology of alexithymia, dealing e.g. with traumatic stress psychological deficit dysfunctional communication between hemispheres , cultural aspects genetic origin disturbance of the mechanism of pain experience involving the limbic system and the endogenous opiates , and central catecholamines .[68,69,70] Alexithymia has also been linked with childhood family environment Further, in addition to primary alexithymia, the term secondary alexithymia has also been conceptualized, resulting from psychological traumatic events, With the development of neuroimaging techniques, alexithymia has been found to be associated with certain neurobiologic processes in the central nervous system.[20,67]

Numerous reports have been published linking alexithymia with various somatic and psychosomatic diseases . It has been found that alexithymia associates positively with somatization.[67] Further, a connection between chronic pain and alexithymia has been observed in several studies with patient samples . However, there are no studies with modern methods, using the Toronto Alexithymia Scale, concerning the association between facial pain and alexithymia.[67,68,69]

Personality features. Several studies have found no evidence of any separate personality profile to be connected with TMD [32] , while others have shown that facial pain correlates with some personality characteristics similar to those of other chronic pain patients Dworkin (1995) identified patients with chronic pain and noted distinct personality styles among them. The first personality style was characterized by excessive avoidance in dealing with everyday responsibilities, including management of pain. In contrast, the individual whose personality style was marked by "approach excess" typically overrelies on external sources, such as medical providers, friends and family, to manage his or her life, including the pain.[19]

4.8.3. Depression;. Depression is a disorder that can be defined as a collection of symptoms such as depressed mood, loss of interest or pleasure, weight loss or weight gain, insomnia or hypersomnia, feelings of worthlessness, and diminished ability to think or concentrate . [71] According to the DSM-III-R criteria a major depression episode includes a minimum of five symptoms that have lasted for a period of at least two weeks. Major depression can be diagnosed on the basis of structured clinical interview. However, reliable and valid self-report questionnaires will yield a numeral rating of depressive symptoms .[72,73]Cut-off points have been applied to these questionnaires to provide an indication of the extent of depression.

Epidemiological studies have shown that depression is the most common mental disorder in man. The point prevalence of a clinically significant depressive disorder is 4 % among men and 8 % among women, whereas depressive symptoms are much more common Depression affects at least 20 percent of women and 10 percent of men during their lifetimes .[74]

There is evidence of greater prevalence of depressive symptoms in subjects with chronic pain in community and patient samples than in controls. Numerous studies have also shown a high rate of depression in patients with facial pain and TMD while the number of population-based studies concerning the connection between depression and TMD) is lower than the number of studies with patient samples.[73,74] In contrast, McGregor (1996) found no differences between depression rates in orofacial pain patients and normal controls.[66]

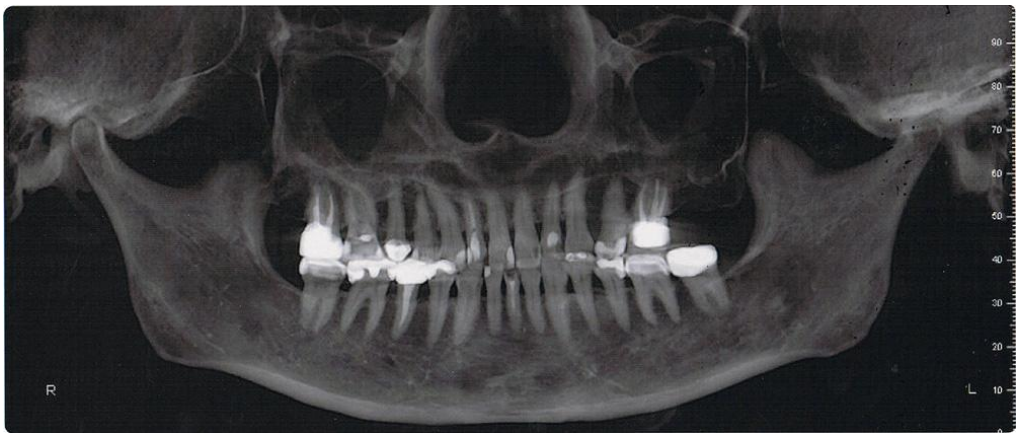

Figure 4;Panoramic film of a patient with significant clicking and mild pain in the right TMJ and a moderate pain and grating noises in the left TMJ.

V. Clinical Evaluation

Temporomandibular disorders are a common cause of craniofacial pain, it is imperative for the health care provider to obtain a comprehensive history, perform a careful physical examination, and obtain appropriate diagnostic studies to exclude other potentially serious disorders. The differential diagnosis should include odontogenic (caries, periodontal disease) and nonodontogenic causes of facial pain, primary or metastatic jaw tumors, intracranial tumors and tumors of the base of the skull, disorders of other facial structures structures (including the salivary glands), primary and secondary headache syndromes, trigeminal neuropathic pain disorders, and systemic disease (cardiac, viral, and autoimmune disease, diabetes, and temporal arteritis).[1,2,3,6,9,10,75] **[Figure 4**] The most common symptom reported by patients with temporomandibular disorders is unilateral facial pain. The pain may radiate into the ears, to the temporal and periorbital regions, to the angle of the mandible, and frequently to the posterior neck. The pain is usually reported as a dull, constant ache that is worse at certain times of the day. There can be bouts of more severe, sharp pain typically triggered by movements of the mandible. The pain may be present daily or intermittently, but many patients have pain-free intervals. Mandibular motion is usually limited, and attempts at active motion, such as chewing, talking, or yawning, increase the pain. Patients frequently describe "locking" of the jaw, either in the closed-mouth position, with inability to open (most common), or in the open-mouth position, with inability to close the jaw. These symptoms are often worse in the morning, particularly in patients who clench or grind their teeth during sleep. Clenching, grinding of the teeth, and other nonfunctional, involuntary mandibular compensatory movements (so-called oral parafunctional habits) are common.[8,9,17,18,22,27,53,56,57,59,75]

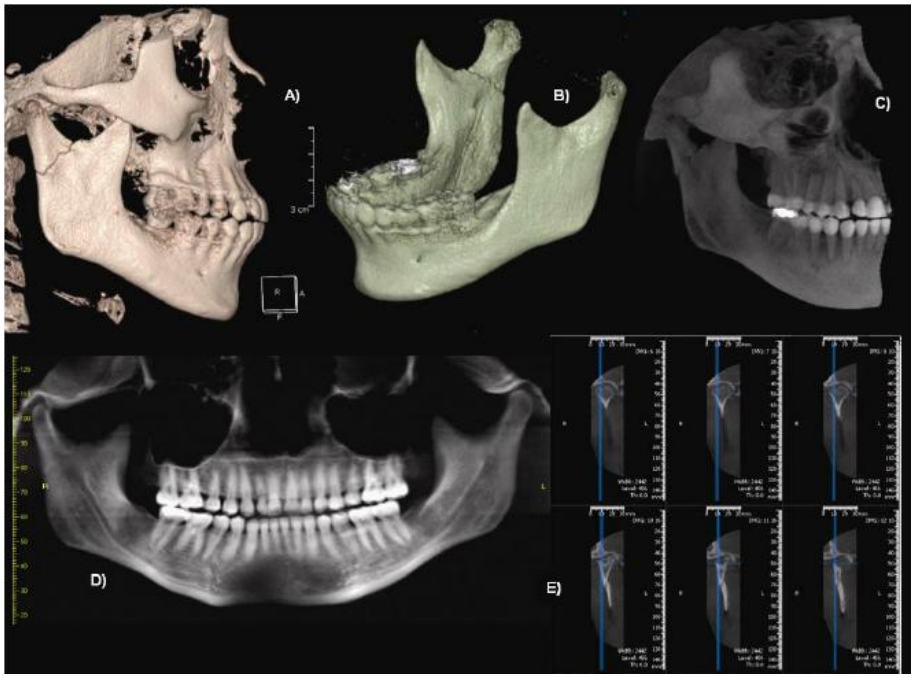

Figure 5;TMJ complex by using CBCT

A basic working knowledge of normal anatomy and function of the stomatognathic complex (mouth, jaws, and closely associated structures) is necessary before conducting a physical examination. Figure 2 shows the normal anatomy of the craniofacial region and the gross anatomy of the TMJ. Normal jaw opening begins with contraction of the suprahyoid muscles and inferior head of the lateral pterygoid muscle, causing a rotation of the head of the condyle in the glenoid fossa with the articular disk between the condyle and the articular eminence of the glenoid fossa (mouth opening of up to 20 mm). With further contraction of the inferior and superior heads of the lateral pterygoid muscles (the major jaw-opening muscles), the condyle continues rotational and translational movement (along an arc of motion) down and forward along the articular eminence of the glenoid fossa, with the articular disk moving in a posterior direction over the anteriorly moving condyle to ensure smooth movement (normal mouth opening up to 35 to 55 mm)**[Figure 5**].Physical examination should include observation and measurement of mandibular motion (maximal interincisal opening, lateral movements,and protrusion), palpation of the muscles of mastication (masseter, temporalis, medial and lateral pterygoid muscles) and the cervical musculature, palpation or auscultation of the TMJ, and examination of the oral cavity, dentition, occlusion, and salivary glands and inspection and palpation of the anterior and posterior neck. Auscultation of the carotid arteries and examination of the cranial nerves, with special attention to the trigeminal system, should also be part of the physical examination.[1,4,24,25,26,36,38,39,55,56,61,75]

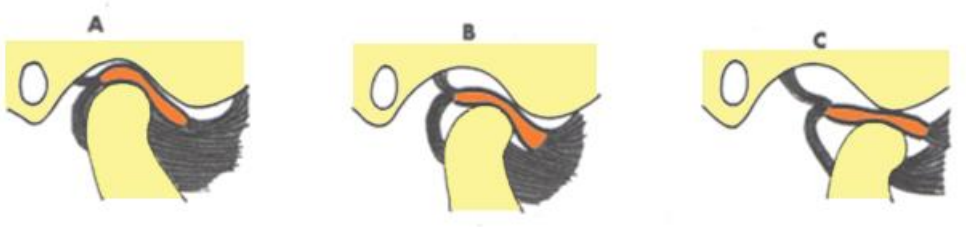

Figure 6. Normal relationship between condyle and disc; they move together

TMJ on mandibular movement is frequently present in patients with temporomandibular disorders. However, noise alone is also a very common finding in people who are completely asymptomatic and may represent a range of normal rather than intra-articular dysfunction. Muscle tenderness, producing pain or discomfort, is generally found on bothextraoral and intraoral palpation of the masticatory muscles. Tenderness may also be present in the anterior neck muscles (suprahyoid muscles and sternocleidomastoid muscles), posterior cervical paraspinal muscles (semispinalis capitus, splenius capitus, and suboccipital muscles), and the upper shoulder muscles (trapezius and levator scapulae).[9,11] There may be mandibular hypomobility and deviation on opening. Finally, the neurologic examination is typically normal, without any objective neurosensory or motor deficits of the trigeminal nerve or other focal cranial-nerve abnormalities.[24,25,75] **[Figure 6]**

Internal joint derangement, however, presents with continuous pain that is localised to the temporomandibular joint and is exacerbated by jaw movement.

Mechanical interferences in the joint, such as clicking and locking, will often result in restricted mandibular opening or deviation of mandibular movements during opening and closing.[24,25,26,36,38,39] **[Figure 7**]

Crepitus or grating sounds emanating from the joint(s) during mandibular function is pathognomonic of temporomandibular joint osteoarthrosis in the elderly. Where the condition is painful, it is referred to as osteoarthritis. Computed tomograms of the

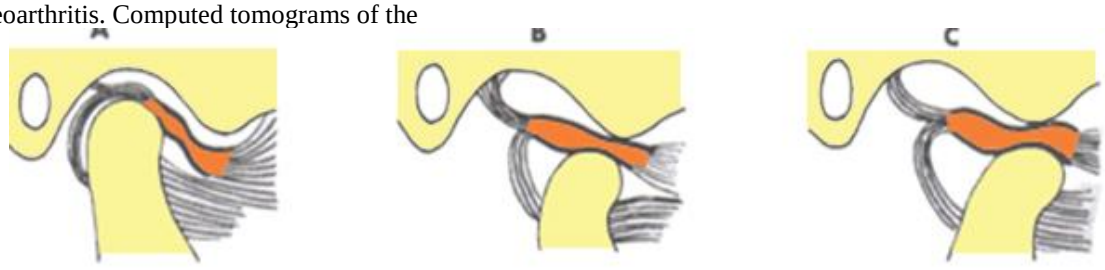

Figure 7. Disc displacement with reduction

temporomandibular joint will often show degeneration and flattening of the condylar head. Most patients seem to cope well with osteoarthrosis as it rarely leads to limited mandibular function. Where computed tomograms show similar condylar changes in younger patients, other arthridities, such as rheumatoid arthritis, should be considered and investigated further. [25,26,36,38,39] [

Diagnostic studies are designed to rule out other disorders. They may include the use of blood and serum inflammatory markers (to rule out autoimmune disorders and vasculitides), imaging diagnostic nerve blocks, muscle trigger-point injections, and dental models for maxillomandibular analysis.[24,25,55,56,61,75]

5.1.Imaging of Temporomandibular Joint

Many diagnostic means have been indicated for the TMDs, including electro diagnostic tests such as jaw tracking devices, electromyography, thermography and vibration analysis. Radiographs have limited use in the identification of temporomandibular disorders7 still various imaging techniques can be used to gain additional insight regarding the health and function of the joint.[75,76] Four basic radiographs can be used for the evaluation of TMJ including; Panoramic view, Lateral Transcranial view, Transpharyngeal view and Transmaxillary AP view. These screening films do not provide enough information therefore more sophisticated techniques are necessary to establish a diagnosis. Lateral tomography, joint arthrography, computed tomography scanning and magnetic resonance imaging are often being used to confirm the diagnosis.[75,76,77] In recent years MRI has become the gold standard for evaluating the soft tissue and the positional abnormalities of the joint disk. The main advantage of MRI is its 3D imaging and no introduction of radiation that might produce soft tissue damage. But it cannot be carried out in patients with pacemakers and who are claustrophobics. Its use is limited by its cost and the time it takes. While computerized tomography has to be reserved to the pre-surgical phase of treatment planning.8

The use of ultrasonography for the diagnosis of TMDs is uncommon, although several reports have been found in the literature suggesting evident advantages of this procedure in depicting effusion in larger joints.[75,76,77,78]

5.2.Laboratory Studies

Lab work may include calcium, phosphate or alkaline phosphatase to check for possible bone disease. An elevated erythrocyte sedimentation rate (ESR) and positive Rheumatoid Factor may indicate rheumatoid arthritis. If gout is suspected, a serum uric acid level should be ordered. An elevated serumcreatinine level may indicate muscle disease .

5.3.Differential diagnosis

When examining patients with suspected temporo- mandibular disorders, the practitioner must bear in mind the possibility of other common disorders such as dental pain; disorders of the ears, nose, and sinuses; neuralgias; headaches; and diseases of the major salivary glands all of which may mimic the symptoms of temporomandibular pain and dysfunction. What distinguishes temporomandibular disorders from other possible diseases is the pain, which is specifically centred in and around the preauricular region and may be accompanied by clicking or grating sounds with mandibular function and restricted mouth opening.[25,75]

VI. Management

Management of all patients with temporomandibular disorders aims to: (1) reduce or eliminate pain, (2) restore normal jaw function, (3) reduce the need for future health care, and (4) restore normal lifestyle functioning.[6] Specific interventions and their sequencing parallel treatment of musculoskeletal disorders in general. A key determinant of success in chronic pain management is the success in educating the patient about the disorder to enhance adherence to the self-care aspects of management, including jaw exercises, habit change, and proper use of the jaw. The treatments included here are supported by randomized controlled trials (RCTs).[1,4,5,6] **[Figure 8**]

6.1.Physical Medicine

Physical medicine interventions can be efficacious for patients with TMD pain and restricted motion.[14,25] Jaw exercise is the primary and often the only physical medicine treatment required. Jaw exercises include relaxation, rotation, stretching (range of motion), isometric exercise, and postural exercise. Stretching exercises, together with cold or heat, are effective in reducing pain and improving range of motion. Their benefit is enhanced when they are incorporated into the patient's daily routine in conjunction with relaxation techniques and a relaxed posture to reduce strain from sustained jaw contraction[16,25].

If exercises are ineffective or worsen pain, other physical modalities can be considered: ultrasound, short-wave diathermy, low-intensity laser, pulsed diathermy, iontophoresis, phonophoresis, superficial heat, cryotherapy (cold), and massage all have demonstrated efficacy. In the short term, such modalities can reduce jaw pain and increase range of motion, thereby allowing jaw exercises to proceed. When range of motion of the jaw is restricted by a TMJ disk displacement without reduction, shortterm manipulation of the jaw by a physical therapist or selfmobilization by the patient can help in remodeling the disk to improve joint translation, range of motion, and pain.

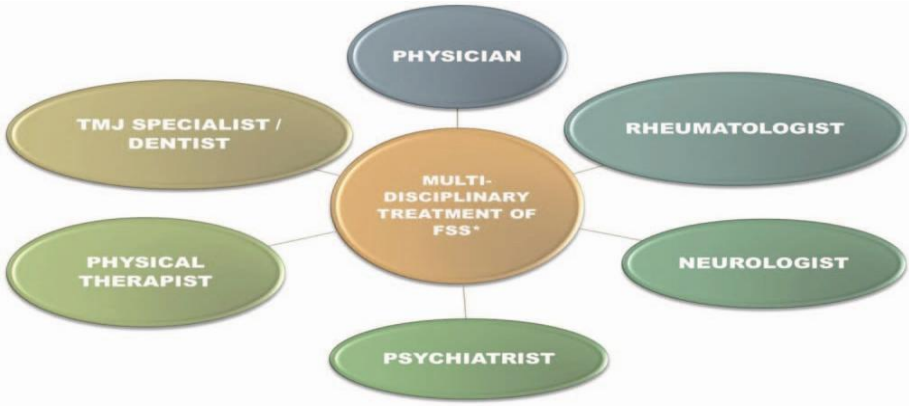

Figure 8: Multidisciplinary approach for treatment of functional somatic syndromes

6.2.Self-Care

Most acute TMD symptoms are self-limited and resolve with minimal intervention. Therefore, initial treatment for masticatory myalgia and arthralgia should be a self-care program to reduce repetitive strain of the masticatory system and encourage relaxation and healing of the muscles and joints. Most patients respond well to self-care in 4–6 weeks; if not, further assessment and treatment are indicated.[22,25,34,37]

6.3.Occlusal appliances

Occlusal appliances are often recommended as initial phase of therapy in the treatment of TMD patients. Occlusal appliances have several uses, one of which is to temporarily provide a more orthopedically stable position. They can also be used to introduce an optimum occlusal condition that reorganizes the neuromuscular reflex activity; this in turns reduces abnormal muscle activity, while encouraging more normal muscle function. [59,60]. Oral appliances also protect the teeth and supportive structures from abnormal forces that may create breakdown or tooth wear (bruxism). The occlusal appliance should be individually made from hard acrylic, preferably by the use of articulato.[25,79] **[Figure 9**]

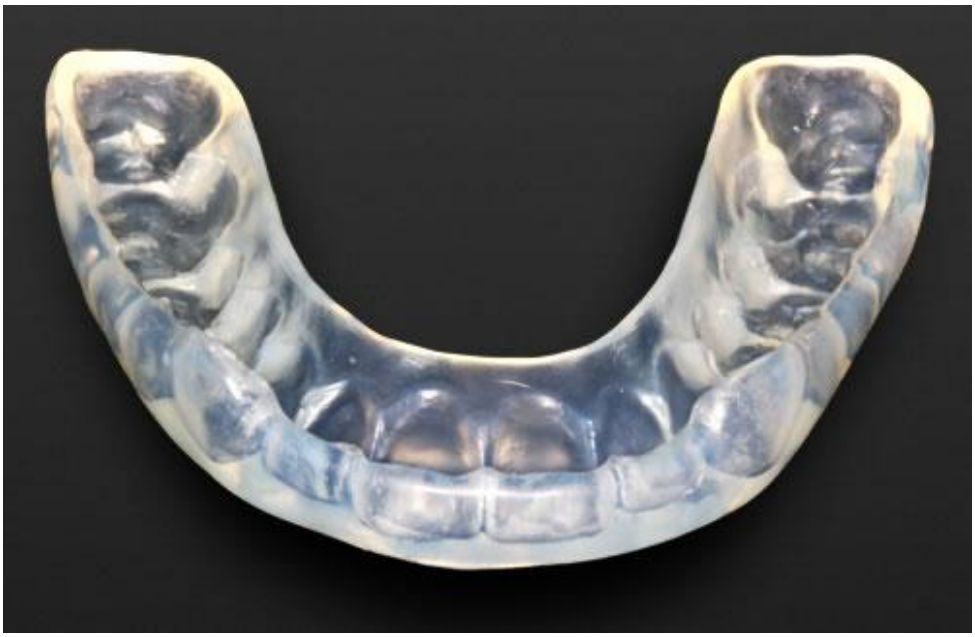

Figure 9;Occlusal Splint

6.4.Pharmacotherapy

Common medications used for TMD pain are classified as nonsteroidal anti inflammatory drugs (NSAIDs), corticosteroids, opioids, muscle relaxants, anxiolytics, hypnotics, and antidepressants.[15,25] Analgesics are used to allay pain; muscle relaxants and anxiolytics for anxiety, fear, and muscle tension; hypnotics for enhancing sleep; and antidepressants for pain, depression, and with certain agents, insomnia.[1,2,7,16] RCTs of NSAIDs for TMD suggest adopting a low threshold for their use as a supplement to self-care. Long-term NSAID use is best approached with caution due to their systemic and gastrointestinal effects, although the safety

of selective COX-2 inhibitors may be greater than that of nonselective NSAIDs. For more severe joint inflammatory symptoms, corticosteroids are efficacious in TMJ synovitis, either as brief, tapering oral doses, injected, or given via iontophoresis.[14,17,18] Injection of hyaluronic acid is just as efficacious as corticosteroids without being associated with any risk of degenerative joint disease.19 Repeated injections of corticosteroids can lead to chondrocyte apoptosis and acceleration of the degenerative process. [20] For myalgia, especially with limited opening, NSAIDs and benzodiazepines are effective.21,22 Cyclobenzaprine has also been shown, in clinical trials of muscle pain, to be efficacious in reducing pain and improving sleep and can be considered when benzodiazepines cause daytime sedation or other side effects.[23] In patients with chronic TMD pain, tricyclic antidepressants such as amitriptyline and nortriptyline significantly ameliorate insomnia, anxiety, and pain. These medications can be used chronically. Selective serotonin reuptake inhibitors (SSRIs) should be used with caution with TMD patients because these agents may increase masticatory parafunctional muscle tension and aggravate muscle pain.[24]

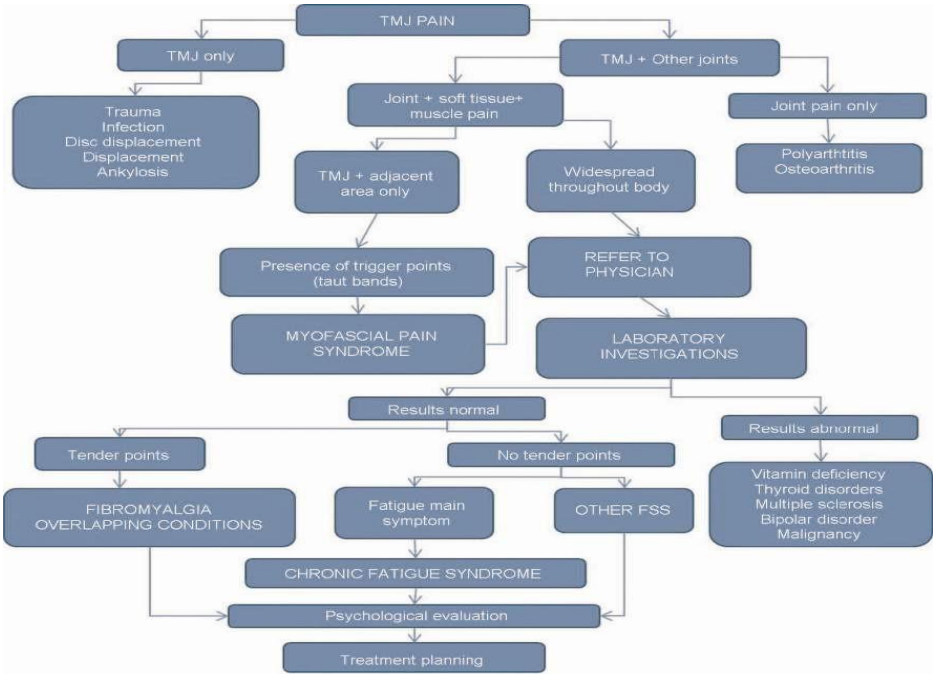

Figure 10: Clues to diagnosis of functional somatic syndromes presenting with temporomandibular disorders

6.5.Cognitive-Behavioral Therapy

Approaches to changing maladaptive habits and behaviors such as jaw tensing and clenching and grinding of the teeth are important in treating painful tissues. Cognitive-behavioral therapies such as habit reversal,biofeedback, relaxation techniques, and stress management can be effective alone or in conjunction with other treatments.[14] Behavior modification strategies such as habit reversal and overcorrection are the most common techniques used to change these habits. Although many simple habits are easily abandoned when the patient becomes aware of them, changing persistent habits requires a structured program that is facilitated by a clinician trained in behavioral strategies. Patients should be aware that habits do not change themselves and that they are responsible for initiating and maintaining behavior changes.

Habit correction can be accomplished by (1) becoming more aware of the habit, (2) knowing how to correct it (i.e., what to do with the teeth and tongue), and (3) knowing why to correct it. When this knowledge is combined with a commitment to conscientious self-monitoring and a focus upon the goal, most habits will change. Supplemental behavioral strategies such as biofeedback may also be helpful.

Even when clenching is unconscious or nocturnal, correcting it during the day will help reduce it at night. Splints may also increase patients' awareness of oral habits. If muscle tensing is the inciting factor, biofeedback and relaxation techniques
may be indicated. Another major issue to address is pacing or hurrying related to a hectic day. For triggers such as depression and anxiety, psychological therapy can helpful. If the problem is a sleep disorder, sleep hygiene self-care can be instituted by the psychologist for nonpathological sleep disturbances, or the patient can be referred to a sleep laboratory for more detailed evaluation.[12,16,19,25]

6.6.Orthopedic Intra-oral Splints

The two most common splints include the anterior positioning splint and the stabilization splint. The anterior repositioning splint places a patient's mandible and TMJ into an anterior position so as to reduce a TMJ click that occurs on opening and closing of the jaw. The anterior repositioning splint is typically placed on the maxillary arch with an anterior ramp that first engages mandibular teeth on initial closure and shifts the jaw forward into final closure, when all mandibular teeth contact the splint. The stabilization splint provides a flat passive occlusal surface that is adjusted with contact on all posterior teeth to allow passive protection of the jaw and reduction of oral habits. Although both splints can reduce TMD symptoms, the indications for each differ somewhat.[26]

Anterior repositioning splints can be efficacious for intermittent jaw locking with limited range of motion, especially upon awakening, or for persistent TMJ arthralgia not responsive to other therapy (including a stabilization splint). They are recommended only for short-term, part-time use, primarily during sleep, because they can cause occlusal changes if worn continuously or chronically.[16,22,33,45]

The stabilization splint is most efficacious for masticatory myalgia and TMJ arthralgia, especially if the pain is worse upon awakening. This type of splint can also be used during the day for oral habit management. Such splints are designed to provide postural stabilization and to protect the TMJ, muscles, and teeth.

Partial coverage splints may cause occlusal changes in some patients. All splints should cover all of the mandibular or maxillary teeth to prevent movement of uncovered teeth, with malocclusion. The splint's occlusal surface can be adjusted to provide a stable occlusal posture by creating single contacts in all posterior teeth in the habitual closure position.[4,8,9,12,14,18,46]

6.7.TMJ Surgery

If persistent pain is localized to the TMJ and is associated with specific structural changes in the joint, surgical intervention can be considered if comprehensive nonsurgical care is unsuccessful.6,14 Muscle pain and associated contributing factors should be addressed and controlled prior to TMJ surgery.[1,4,8,10,25]

In general, the less invasive surgeries are as efficacious as those that are more invasive, so the health care provider should consider an arthrocentesis or arthroscopic procedure before more invasive interventions such as diskectomy or disk repair. Postoperative management includes appropriate medications, physical therapy, splint therapy when indicated, and continued psychological treatment as appropriate. Recommended surgical procedures for the treatment of temporomandibular disorders are:

1. Arthroscopy
2. Arthrocentesis
3. Condylotomy (indirect arthroplasty)
4. Joint replacement (partial or complete)
5. Other Procedures
• Coronoidotomy/coronoidectomy
• Recurrent/chronic dislocation
• Styloidectomy (Eagle's Syndrome)

Arthroscopy of the TMJ was first introduced by Ohnishi (1975). Initially, it involved merely lavage of the joint and the use of a probe to break up adhesions. However, with the introduction of improved instruments, arthroscopic operations for various intra-articular disorders are made possible. Arthroscopic lysis and lavage can be used to treat patients with painful clicking or popping, to release intra-articular adhesions and anteriorly displaced non-reducing discs, and to confirm other diagnostic findings that could warrant surgical intervention.[80]

Arthrocentesis involves irrigation of the joint with lactated Ringer's solution or saline. In certain acutely inflammatory joint conditions, steroid injection may follow arthrocentesis. This procedure is often followed by mandibular manipulation and is recommended for patients who have unresolving joint restrictions and for those individuals who have developed an acute or chronic closed lock25. It is recommended that the patient have a stabilization or repositioning splint ready to be delivered immediately following the procedure. The procedure may need to be repeated if the lock recurs, and the patient must be reminded to avoid activities that cause locking.[80,81] Arthroscopy is direct visualization of a joint with an endoscope. It is performed by an oral and maxillofacial surgeon mainly in the upper joint space and is recommended primarily for lysis and lavage and also for ablation of adhesions and biopsy, An MRI of the joint is needed prior to the arthroscopic procedure.[81]TMJ arthrotomy is an open surgical intervention performed by an oral surgeon. It is recommended for severe osseous pathology involving the TMJ, such as ankylosis and severe osteoarthritis that has not responded to conservative treatments. It is crucial to work closely with an experienced TMJ surgeon to assess the necessity of this procedure. Open surgical procedures include disk repair (discoplasty), disk removal (discectomy) with or without replacement and disk repositioning, and arthroplastic procedures, such as condylar repair and removal (condylectomy).[82,83]

6.8. Acupuncture

Acupuncture is reported to be an adjunctive treatment, producing a short-term analgesic effect in patients with painful TMJ symptoms. Again, accessing these treatments under the NHS is often difficult for patients ,although, private facilities do exist.[84]

6.9. Hyaluronate

Hyaluronate is a gel-like glycosaminoglycan that takes in water to ensure the resilience of cartilage. Hyaluronate injections are sometimes used for osteoarthritis of the knees or hips, as it acts to cushion the joint and reduces pain. However, reviews of the evidence for its application in TMJ problems found that there is currently insufficient evidence to decide whether it was beneficial for these patients .There are several systematic reviews of studies on hyaluronic acid for treating TMJ disorders.Only one of the systematic reviews limited its inclusion criteria to randomized controlled trials and pooled study findings. This was a Cochrane review by Shi and colleagues, published in 2003. The Shi review included RCTs comparing the effect of at least one hyaluronic acid injection alone or in combination with other active treatments to placebo or glucocorticoid injections alone or in combination with the same active treatment group. A total of 7 studies met inclusion criteria; 3 studies compared hyaluronic acid and placebo, 3 studies compared hyaluronic acid and glucocorticoids and 2 studies compared hyaluronic acid plus arthroscopy or arthrocentesis to arthroscopy or arthrocentesis alone. [85]

A 2012 study by Manfredini and colleagues in Italy randomized 72 patients with TMJ dysfunction to one of 6 treatment groups: 1) single-session arthrocentesis alone; 2) single-session arthrocentesis plus corticosteroid; 3) single-session arthrocentesis plus low-molecular weight hyaluronic acid; 4) single-session arthrocentesis plus high-molecular weight hyaluronic acid; 5) 5 weekly 2-needle arthrocenteses plus low-molecular weight hyaluronic acid; or 6) 5 weekly single-needle arthrocenteses plus low-molecular weight hyaluronic acid. (16) A total of 60 out of 72 (83%) participants completed the study, between 9 and 12 patients per treatment group. In a per protocol analysis, there were no significant differences among groups on any of the outcome variables at the 3-month follow-up. For example, the percentage change in pain at rest ranged from -29.1% (standard deviation [SD]: 62.9%) in the group receiving 5 weekly single-needle arthrocenteses plus low-molecular weight hyaluronic acid to -38.4% (SD: 56.5%) in the group receiving a single-session of arthrocentesis alone. Limitations of the study include the small number of patients in each treatment group and the substantial number of dropouts in absence of an intention-to-treat analysis.[86,87]

A 2007 study by Bjorland and colleagues in Norway published a double-blind RCT that included 40 patients with osteoarthritis of the TMJ. Patients received 2 injections, 14 days apart, of either sodium hyaluronate or corticosteroids. Pain was assessed using a visual analogue scale (VAS) from 0 to 100. Patients were followed for 6 months (assessed at 14 days, 1 month and 6 months). There was a statistically significant reduction in pain within each group at all of the follow-up points. At the 6 month follow-up, pain intensity (mean VAS score) was 14 (SD: 16) in the hyaluronic acid group and 31 (SD: 32) in the corticosteroid group; the difference was statistically significant (p=0.0012). The number of patients who were pain-free at 6 months was 7 of 20 (35%) in the hyaluronic acid group and 6 of 20 (30%) in the corticosteroid group (p value not reported).[88]

Bertolami and colleagues published a double-blind placebo-controlled trial in 1993 which included 121 TMJ patients. Patients needed to have a confirmed diagnosis of degenerative joint disease (DJD), reducing displaced disc (DDR) or non-reducing displaced disc (DDN), to have failed other non-surgical treatments, and to have severe dysfunction. Patients received a single injection of sodium hyaluronate or saline and were followed for 6 months. A total of 80 patients were randomized to the hyaluronate group and 41 to the placebo group. This included a total of 57 patients in the DJD group, 50 patients in the DDR group, and 14 patients in the DDN group. Fourteen of 121 patients (12%) were excluded from the analysis because they were found not to meet eligibility criteria. No significant differences in outcomes were seen for the DJD group. In the DDN group, there were significant between-group differences through 1 month, favoring the hyaluronic acid group. The number of patients in the DDN group who completed follow-up after 1 month was insufficient to draw meaningful conclusions about efficacy. In the DDR group, there were no statistically significant differences between groups in any outcome at 1 or 2 months. At 3 and 6 months, 2 out of 7 reported outcomes were significantly better in the hyaluronic acid compared to the placebo group. At 5 months, 5 out of 7 reported outcomes were significantly better in the hyaluronic acid group. The 7 outcomes included 3 measures of dysfunction, 2 measures of patient perception of improvement, 2 measures of change in noise. The most consistent between-group differences in the DDR group were for the 2 measures of patient perception of improvement and one of the noise variables. There were fewer between-group differences on dysfunction measures.[89]

6.10.Botulinum toxin

In recent years, botulinum toxin or Botox® has been used to treat TMJ pain arising from myofacial pain dysfunction syndrome. This involves the injection of Botox into masseter or temporalis muscles and has been shown to increase mouth opening and reduce pain.[90]

6.11.Dental Treatment

There is no consistent evidence from RCTs that altering the occlusion through occlusal adjustment will benefit TMD.[14,25,27] Likewise, other dental treatments such as prosthodontic and orthodontic treatments are not recommended as a primary treatment for the management or prevention of TD.[6,26] However, patients with TMD may require these procedures as part of normal dental care. In these cases, care should be exercised in minimize additional strain to the muscles and joints and aggravation of an existing TMD during these procedures.[25]

6.12.Low Level Laser Therapy

Low Level Laser Therapy (LLLT) uses light energy from Low Level Lasers or Superluminous Diodes (SLDs) to reduce pain, modulate the immune response and stimulate healing. There are also a number of secondary effects from LLLT, including stimulation of β-endorphins, fibroblasts for soft tissue repair and osteoblasts for the repair of bone. LLLT has been demonstrated both clinically and in research to be effective for post-surgical pain and swelling, better integration of implants, healing of soft tissue lesions, and nerve regeneration.[91]

LLLT is an effective tool in the treatment of TMJ disorders. Studies have shown that LLLT can decrease pain, muscle trismus and swelling. There has also been some evidence to show that LLLT can help stimulate fibroblasts to form a pseudo disc in cases of disc degeneration.[92,93]

In a recent study, Kobayashi et al hypothesized that one of the pain relief mechanisms when using LLLT in the treatment of TMJ disorders is the improved microcirculation in the temporal and masseter muscles.4 This improved circulation helps to remove noxious deposits associated with hypertension of the tissues. Pain relief is also felt by normalizing the intramuscular pressure on sensory nerve endings.5 Other studies have demonstrated that LLLT was shown to be effective for those with chronic pain and in those who did not respond to other previous conservative treatments.6 Further, in over 30 years of research, there have been no negative side effects associated with LLLT treatments.[91]

LLLT involves the use of both Low Level Lasers and SLDs. Low level lasers are most frequently used to treat joint spaces and trigger points, whereas SLDs are often found in clusters and can be used to cover larger muscles.[91,92,93] Although it is beneficial to use both types of devices, either can be used to effectively treat TMJ disorders.

When treating TMJ disorders, LLLT should include treatment of one or more of the following points:

- Temporomandibular Joint (opened and closed)
-Styloid Process (which includes the joint capsule)
- Lateral Pterygoid
-Trigger Points in the Sternocleidomastoid muscles
- Li4 Acupuncture Point
- Masseter Muscle Trigger Points
-Temporalis Muscle Trigger Points

It should be noted that treatment does not include all of the above points in every case; treatment locations are determined by the diagnosis and area of pain.

LLLT is most effective for acute conditions and often can be used as the sole treatment tool. In acute cases, the patient should be treated 3-4 times for one week and then left for two weeks before being reassessed. Chronic conditions often require a combination of LLLT, splints and other physical therapy. Patient should generally be treated 2-3 times per week for 3 weeks before being reassessed. It's important to note that if the patient doesn't experience any improvement the condition should be reassessed.[91,92,93,94,95]

VII. Conclusion

The concept of TMDs treatment procedures is indirectly connected with the already established symptoms and signs from the medical history and clinical examination. Since the exact pathophysiological mechanism of TMDs development has not been fully explained, the main goal of the treatment is the management, reduction and removal of temporomandibular pain **[Figure 10]**. Treatment procedures are divided into reversible and irreversible procedures. Since the treatment is mostly empirical, that is, performed by evaluating the clinical significance if the established symptoms, the reversible procedures are mostly used.

Treatment indications, type of treatment procedures and their practical application are based on the existence of a specific form of myogenic and/or arthrogenic disorder in the stomatognathic system accompanied by certain intensity of temporomandibular pain. TMD management in these patients cannot be achieved by the traditional methods of TMD treatment alone and, as such, certain modifications are required for these patients even during routine dental treatment. A multidisciplinary approach for the management of the underlying FSS is required and the dentist should be an active participant in this team.

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