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Temporomandlbular Joint Arthralgia Or Osteoarthritis? A Diagnostic And Clinical Dilemma

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Abstract:
Introduction: *Osteoarthritis of the Temporomandibular Joint is a chronic inflammatory and destructive process of the soft and hard tissues. The process is considered as one or the final stage of the so-called internal derangements of the temporomandibular joint. Because osteoarthritis is a long – standing pathological process, it is expected that at least one pathological disorder, in this case TMJ arthralgia precedes the development of full characteristics of TMJ osteoarthritis.*
Aim: *Provide further theoretical support to the notion that there exists arthralgia as an another TMJ internal derangement that precedes the development of osteoarthritis sign and symptoms.*
Methods: *Using the search terms "Internal derangements. Temporomandibular joint. Osteoarthritis" or "Temporomandibular joint. Internal derangements. Arthralgia" or "Temporomandibular joint. Arthralgia. osteoarthritis", information about arthralgia and/or osteoarthritis was obtained from www.google.com.*
Outcome: *Even though we obtained about 60 papers or documents mentioning "arthralgia and/or osteoarthritis of the temporomandibular joint", 25 papers or documents were discarded on the basis of insufficient or irrelevant information about TMJ arthralgia. Thus, 35 papers or documents were selected on the basis of relevant information about different topics under investigation, reliability of the source of information (journal), and recency of publication. These 35 papers were used to discuss TMJ osteoarthritis and TMJ arthralgia as two different pathological entities.*
Conclusion: *TMJ arthralgia is an independent internal derangement of the temporomandibular joints that precedes the development of osteoarthritis. There is scarce information indicating a progression from disc displacement without reduction to arthralgia and then to osteoarthritis in all clinical cases. Further studies on this subject are needed.*
Keywords: *Temporomandibular Joint. Osteoarthritis. Arthralgia. Internal Derangements.*

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I. Introduction

The temporomandibular Joint: The temporomandibular joint (TMJ) is the articulation of the body that connects the temporal bone with the distal and upper part of the lower jaw. This joint is involved in a number of organic functions including chewing, speech, swallowing and posture of the lower jaw. The TMJ is a very complex joint capable of performing both hinge and sliding movements. Even though the TMJ is a very small articulation, it is recognized as a very complex joint in which with the exception of the joint disk, it is provided with abundant innervation and blood supply. According to one investigation [1], the fibrocartilaginous disc has the capability of protecting the joint components against excessive biomechanical stress and tension, a function facilitated by the absence of both blood vessels and nerves in the joint disc.

II. Internal Derangements Of The Temporomandibular Joint:

Internal derangements (IDs) of the TMJ or TMJ-IDs is a new concept in Orofacial Pain (OFP) and Temporomandibular Disorders (TMDs) developed in the eighties used to describe abnormal function in the anatomic components of the TMJ, usually related to a pathologically displaced disc and local inflammation. TMJ-IDs are also defined as a joint dysfunction type associated with abnormal disc position [2] that interferes with normal joint movements usually associated with pain and inflammation. Both macro and micro trauma are the most common etiological agents in the development of TMJ-IDs sign and symptoms. Both trauma and

parafunctional behaviors lead to inflammation and pain in the first stage of the disease and in inflammation and tissue destruction in the last stages of the disorder. Some patients presenting with TMJ-IDs heal spontaneously whereas in other cases the disease process follows a course in which degenerative alterations are seen in the last stages of the disease [2]. TMJ-IDs are usually correlated with three interrelated phenomena: Disc displacement, inflammation and pain.

III. Terminology About Arthralgia

The terms arthralgia is frequently used in the study of TMJ-IDs and OFP with two connotations:
1.A state of pain in the TMJ based on the Greek root of the term: Arthros=articulation or joint and algios=pain
2.An advanced internal derangement of the TMJ which usually develops following a severe disc displacement, inflammation and pain, that precedes the development of signs and symptoms of osteoarthritis (OA).

In the current investigation the terms "arthralgia of the TMJ" will be used with the connotation of "an inflammatory and painful TMJ-ID characterized by inflammation, pain and probably early manifestations of TMJ-osteoarthritis".

IV. Arthralgia

Arthralgia can be defined as a TMJ-ID characterized by the presence of an inflammatory process and subtle osteoarthritic alterations and/ or as a stage that precedes the development of TMJ OA. There is a paucity of research about the relationship between arthralgia and disc position. However, it is expected that severe changes in both position and shape of the joint disc have occurred before the development of arthralgia and TMJ-OA. Because some signs, symptoms and characteristics are present in arthralgia, for both diagnostic and therapeutic reasons, arthralgia should be considered as another stage in TMJ-IDs. De Souza [3], points out that arthralgia is another inflammatory disorder of the TMJ with neuropathic components based on research by other investigators. TMJ arthralgia is considered as a more localized form of pain usually described as sharp pain of moderate intensity in the TMJ that radiates mostly to the ear region [4]. It is very likely that severe inflammation represents a mechanism central to the initiation of destructive activity in the joint disc, cartilage and bone. Thus, arthralgia may represent not only a different type of TMJ-ID, but the point of departure for the initiation of osteoarthritic changes in the TMJ.

It is very likely that arthralgia encompasses a first stage characterized by intense inflammation and a second stage that paves the way for the development of degenerative changes leading to OA. In line with this point of view, one investigation [5] asserts that TMJ arthralgia may be caused by inflammatory and/or degenerative changes in the joint. Based on the review of some studies it seems that some osteoarthritic changes may or may not be present together with arthralgia. Regarding the importance of inflammation in arthralgia and before the development of osteoarthritic changes, one investigation [6] asserts that even though the relationship of inflammatory fluid to pain and OA is not yet clear, joint effusion is a sign that may be observed before osteoarthritic changes occur, indicating that joint inflammation is a key element both to arthralgia and OA. Inflammation may be a key component of the arthralgia and OA process. For instance, in writing about TMJ and osteoarthritis and early diagnoses, Das [7] points out that regarding OA, the radiological signs may increase while the pain decreases. This observation probably indicates that inflammation is a key element in both arthralgia and OA, operating through different mechanism. For instance, more severe inflammation co-occurs with arthralgia and facilitates the development of OA signs and symptoms. As OA signs and symptoms progress, inflammation decreases slowly. Thus, a higher level of inflammation may sustain signs and symptoms in arthralgia, whereas the development of early signs and symptoms of OA, are signaled by a decrease in the level of inflammation.

V. Etiology

TMJ arthralgia may be the result of micro or macro traumatic events from extrinsic overloading of the TMJ [7] including from a combination of persistent both diurnal and nocturnal oral jaw habits that overcome the adaptative capacity of the anatomic components of the TMJ including the joint disc, capsule and synovial membrane. Joint overload can be classified in two types: Joint overload associated with a number of oral jaw habits with more or less intense and or frequent forces applied on a relatively normal dentition and joint overload associated with oral jaw habits applied on a masticatory system with lower capability to resist such forces as posterior teeth are not present in the individual´s dentition. Joint overload is the most common type of etiological element causing signs and symptoms of TMJ-IDs. Joint overload causes excessive friction both on the joint disc and on the head of the mandible. Excessive and sustained loading facilitates the development of inflammation by pressure induced posteriorly on the joint disc; an anatomic area profusely innervated and vascularized. Persistent parafunctional jaw habits may continuously overload articular structures beyond their adaptive capability leading to pain, synovitis and intra-articular adhesions, OA. These changes may ultimately result in the perforation of the articular disc [2]. The concept of biomechanical stress is gaining force in the current literature about etiology of TMJ-IDs. Some researchers defend the notion that TMJ-IDs including arthralgia are initially produced by an

intense stimulus, supposedly, mechanical stress and tension. Because anxiety is an important element in the etiology of TMD-IDs, anxiety associated oral jaw habits that produce high levels of mechanical tension in the TMJ tissues, are frequently considered in the etiology of arthralgia, OA and other TMJ-IDs.

At the psychosocial level, if parafunctional habits occur frequently in individual with TMJ-IDs including arthralgia and OA, the clinician and researcher should consider the importance of somatization in the context of etiology. Somatization usually includes components of anxiety and depression in this complex psychiatric construct. Biomechanical factors including parafunctional behaviors and macro traumatic events, are usually considered in the etiology of TMJ-IDs including arthralgia. Further, higher [8] levels of somatization are associated with TMDs of arthrogenous and myogenous origin.

VI. Clinical Characteristics

Arthralgia is a more advanced TMJ-ID characterized by a more localized and sharp pain of moderate intensity occurring in the TMJ and adjacent anatomic structures which may radiate to the ear. Pain in this clinical situation may become more intense and frequent during loading and function of the joint [7]. Intense loading may severely limit and impair jaw movements. Even though one investigation evaluated a small sample of subjects presenting with TMDs, researchers reported that pain described as burning indicating arthralgia was reported by 100% of subjects in the experimental group [9]. Further, researchers also reported that unilateral and bilateral click and even crepitus were reported by 72% of the group. Bilateral reciprocal click occurred more frequently in the population of 25 subjects with arthralgia. The high presence of joint noises in this investigation is in line with one research [10] reporting that restricted jaw opening, blockages (probably periods of jaw locking), and joint noises are common clinical characteristics in TMJ-arthralgia.

Arthralgia of the TMJ is characterized by spontaneous pain perceived from the TMJ region in addition to pain on palpation of the lateral pole or posterior attachment of the TMJ on the same side [11]. Because pain on palpation of the TMJ may also be observed in the presence of capsulitis, retro discal pain, disc-attachment pain and other disorders, only spontaneous pain conserves clinical relevance in the diagnosis and clinical characterization of arthralgia. A relevant clinical characteristic [12] in TMD patients with arthralgia is a painful response contralaterally to the site of application of a compression test in the posterior teeth. TMJ arthralgia may be caused by inflammation and/or degeneration of the articular surfaces. If joint palpation causes pain but joint crepitus is not observed during any jaw movement, the clinician should suspect of arthralgia without joint degeneration [5].

Because some TMJ-IDs slowly progress to a more advanced stages, if not treated this more severe and chronic type of TMJ-ID may worsen and pain becomes more intense and frequent [7], more specifically in the presence of the phenomenon of central sensitization and is reported more frequently by those patients presenting with anxiety, depression and somatization.

VII. Prevalence

Cordeiro and associates [13] evaluated a sample of 33 patients presenting with rheumatoid arthritis referred consecutively over a period of one year. Clinical and radiographic evaluation using CBCT were carried out. They found that 4/33=12,12% patients demonstrated arthralgia alone and 8/33=24,2% demonstrated characteristics of both arthralgia and OA. Arthralgia is a common type of TMJ-IDs that ca be found in approximately 8% of the general population and is closely associated with the presence of OA [10] which may be a cause or a consequence. Because of the presence of burning pain in the clinical description of arthralgia, such type of TMJ-ID should be considered as a component of neuropathic pain. Thus, regarding prevalence, one investigation [14] indicates that a form of neuropathic TMJ pain is present only in a small percentage of patients with TMJ-IDs. Subjects with TMDs may occasionally complain of additional orofacial pain complains including neuropathic ones. In the investigation using a sample of 501 TMD subjects, Dupont [15] reported a prevalence of 12% trigeminal neuritis. Notwithstanding the relevance of such findings, Dupont did not report if such pain was also present in the anatomic structures of the TMJs.

Pedulla and associates [14] assessed a series of 82 consecutive patients referred to craniofacial facility. Clinical and RMI assessment were carried out and researchers reported a frequency of 13/82=15,9% subjects and 16 joints presenting with signs and symptoms of TMJ-IDs and concomitant neuropathic pain. Researchers indicated that a closer proximity between the displaced TMJ disk and the mandibular nerve could represent one mechanism causing neuropathic pain in patients with TMJ-IDs. One investigation [12] evaluated a population of 300 subjects with TMJ signs and symptoms. Because researchers used test biting on the contralateral molar region and obtained a positive response in 60/300= 20% participants, a frequency of 20% arthralgia was found in such investigation.

VIII. Physiopathology In Arthralgia And Burning Pain

Patients presenting with TMJ-IDs usually describe their pain as dull, aching, constant, intermittent and sometimes shooting. In arthralgia most patients describe their pain as burning, but other descriptors including dull, aching or sharp can be used. The term "burning" has the connotation of a neuropathic component of pain and probably indicates that a specific fiber type has been affected. Burning pain can be elicited by intraneural micro stimulation of C nociceptive fibers although some role of myelinated fibers has been indicated. Burning pain may indicate some role of regenerating C fibers in neuropathic pain. Such fibers show hyperexcitability and spontaneous activity [16]. Burning pain is also associated with the phenomenon of ectopic activity in nerve-end-neuroma, regenerating sprouts, neighboring uninjured neurons and compressed nerves [16]

Both arthralgia and OA of the TMJ may be viewed as a failure of reparative mechanisms in which remodeling, destruction and deformation take place. Thus, there is an imbalance in the reparative-destructive processes favoring destructive and remodeling mechanisms without the capability for repairing and reconstructing the normal anatomy and function of the involved tissues.

In TMD arthralgia and OA the development of signs and symptoms usually indicates the presence of a disorder in the balance of the articular tissue, in which a chain of events favors the destruction of articular tissue overcoming the capability of local tissues to adapt and produce normal tissue- In this case, the synovial membrane store elevated levels of proinflammatory cytokines, enzymes that degrade local tissue and breakdown products that not only show difficulties to be eliminated, rather, they function sometimes as elements or foreign bodies called oxidative stress products that perpetuate a local inflammatory response [10] thus resulting in more intense pain, inflammation, and protective reflexes.

IX. Is Arthralgia A Different TMJ Internal Derangement Closely Associated Or Preceding The Development Of OA Sign And Symptoms?

Based on the review of the current literature, on the description of pain and its close association with OA, there are reasons to believe that arthralgia is a different TMJ-ID which may be viewed as "an advanced stage of TMJ-ID characterized by an inflammatory reaction in the unprotected joint surfaces which opens the way for the development of osteoarthritic joint changes". This point of view is echoed by some investigations, as follows:

Khotari and associates [6] compared somatosensory function between TMJ arthralgia and TMJ osteoarthritis. Researchers evaluated 43 arthralgia and 15 TMJ OA patients using CBCT as the imagen technique and found that out of 43 patients with a previous diagnosis of arthralgia, 21/43 had degenerative changes whereas out of 15 OA patients diagnosed clinically only 9/15 showed degenerative changes using CBCT. These findings indicate that in theory the use of RMN or CT is essential to compare clinical findings and imaging findings in both patients with arthralgia and in those with OA. Researchers reported that even when 6 patients presenting with no degenerative changes using CBCT, they were classified as presenting arthralgia regardless of the coarse TMJ crepitus. In fact, many patients classified clinically as presenting arthralgia based on clinical characteristics including age, description of pain, a long history of TMJ-ID and a report of burning pain demonstrate presence of a dull joint sound or noise perhaps a residue or a degeneration of a reciprocal clicking. Noteworthy to mention is that in the study by Kothari and associates [6] they considered arthralgia and osteoarthritis as different yet related TMJ-IDs. A descriptive summary illustrates clinical and experimental observations indicating that arthralgia is a TMJ-ID different from OA:

1.Patients describe TMJ pain in arthralgia as burning, indicating that this type of derangement is unique and different from OA and other internal derangements of the TMJ.

2.Based on one investigation [9], the frequency of unilateral and bilateral clicking occurred very frequently in patients with arthralgia, whereas crepitus occurred more frequently in OA subjects. The difference in the types of joint noises strongly support the notion that arthralgia precedes the development of OA.

3.Patients presenting with arthralgia sign and symptoms are usually younger as compared to subjects with OA signs and symptoms.

4.Pain in subjects with arthralgia may be induced by experimental overload on the molar area contralateral to the affected TMJ. If pain is induced, the symptom indicates the presence of arthralgia in the contralateral TMJ.

5.Joint and muscle stiffness in the masseter muscle have less likelihood of being observed in subjects with arthralgia, but this likelihood increases in subjects with TMJ OA.

6.Inflammatory and neuropathic components may be found more frequently in subjects with arthralgia rather than in those presenting with OA [3].

7.A pain description of burning, a painful TMJ response to overloading in the contralateral side (molars) and no observation of crepitus during joint palpation, are important determinant characteristics of TMJ arthralgia.

X. Diagnostic Considerations

TMJ arthralgia is not a well-known TMJ-ID. Thus, it is rarely mentioned in the current literature. Clinical observations have led some clinicians to believe that such a disorder precedes the development of OA changes in

the TMJ. For some researchers particularly for those who base the diagnosis on the use of sophisticated methods including CT and/ MRI, it is very difficult to establish when a disorder has characteristic of arthralgia without OA signs. Notwithstanding this, there are some characteristics that indicates the presence of arthralgia in a clinical case:

1.A complaint of pain over the TMJ including pain with function or with assisted or unassisted mandibular movements [7].

2.No coarse crepitus [7]

3.Pain may be induced by movements that cause rubbing or friction of the join surfaces, for instance, instructing the patient to perform protrusive jaw movements without separation of the teeth. This maneuver induces rubbing or close contact of the joint surfaces affected by inflammation and low-level degradation, thus resulting in local pain and discomfort.

4.One investigation [9] described the clinical and diagnostic characteristics of arthralgia as follows: The patient reports a long history of TMJ-IDs, pain is described as burning, different types of joint noises including reciprocal clicking or ill-defined crepitus and pain induced by a protrusive movement of the lower jaw without separation of the teeth. This maneuver or test causes rubbing of inflamed, unprotected and deformed joint surfaces, thus resulting in pain. Patient age is also an important consideration in the diagnosis process. It is true that arthralgia is a chronic inflammatory and painful process. However, in OA, both the inflammatory and degenerative process are more chronic. Thus, mean age in arthralgia patients is usually around 40 years whereas older individuals are usually observed in samples of OA patients.

5. In the study by Khotari and associates [6] they established the diagnosis of arthralgia based on the presence of pain, coarse crepitus at the TMJ on clinical examination and absence of osteoarthritic changes using CBCT.

6.If patients with arthralgia are examined using MRI, the presence of joint effusion is an additional characteristic of arthralgia and is observed before OA changes occur [6]. The clinical significance of this observation is that "the affected region is still an inflammatory process, indicating more inflammatory than OA changes".

7.One investigation [13] evaluated the clinical and radiographic characteristics of patients presenting with signs and symptoms of rheumatoid arthritis. Patients were evaluated clinically and using CBCT. Researchers reported that the presence of arthralgia may indicate the early stages of OA. Pain and inflammation predominate in the arthralgia stage whereas pain is absent in patients with advanced stage of rheumatoid arthritis and osteoarthritis although some OA and rheumatoid arthritis patients complain of sporadic pain. Rheumatoid arthritis most of the cause progresses to an OA stage in most patients.

8.Coarse crepitus is not included in the characterization of arthralgia according to a publication by the International Association for the Study of Pain [4]

XI. Treatment Or Management

Because arthralgia involves the presence of severe inflammation or a combination of inflammation and subtle degenerative process and is also considered an advanced stage of TMJ-IDs, its treatment may involve the use of conservative and nonconservative modes of treatment. Treatment for signs and symptoms of TMJ-IDs and OFP may be combined with management of psychological or psychiatric disorders. In this case, symptoms of moderate to severe depression, anxiety or significant disability observed during the diagnostic assessment, indicate referral to a psychologist or psychiatric and a physical therapist may be necessary [5]. As part of the treatment for OFP and – or TMJ-IDs, Interocclusal devices to reduce biomechanical stress in muscles and joints is highly recommended. Further, muscle relaxants, and pain killers should be used during a certain and controlled period in order to prevent side effects of medication.

Because metabolic waste products including residues of the local oxidative stress, inflammatory molecules and even pro inflammatory cytokines may be found in the synovial fluid and they are responsible for sustaining pain and inflammation, some minimal invasive methods including arthrocentesis or arthroscopic lysis and lavage may be used in many cases. Self-care techniques including jaw stretch, hot water on the masseter muscle and separation of the teeth during the day may be used to increase jaw opening, induce local elimination of waste products, relaxation of jaw elevator muscles and reduce loading on muscles and joints. Patient´s education may be necessary in some TMD patients with arthralgia to modify the personal belief system and the negative influence of some psychological factors including catastrophizing and pain amplification. These techniques may be applied by a skilled clinician or by a psychologist and/or psychiatrist trained in behavioral psychology especially in those patients presenting with high scores in somatization, anxiety and depression. Because bruxing behavior usually occurs associated with many other oral jaw habits, it is recommended that the patient becomes aware of the destructiveness of such behaviors and how they induce pain, inflammation and muscle tension. In cases and when anxiety, stress and depression have been recognized by the skilled clinician, some drugs including clonazepam 0.5mg, cyclobenzaprine 5mg and/or amitriptyline 10 or 25mg may be used to control the negative effects of such psychological states. The negative effects of myofascial pain dysfunction that

usually occur together with TMJ-IDs, may be managed with a combination of interocclusal devices, muscle relaxant drugs, jaw exercises and even anesthesia to painful trigger points.

XII. TMJ Osteoarthritis

As a musculoskeletal disorder, OA is a chronic disease resulting from a complex and multifactorial causes or etiologies. Such factors are different when compared with other body joints and may or may not result in severe disability and loss of quality of life. OA [17] may be interpreted as the result of a cascade of pathological events in which a sustained inflammatory process induced by metabolic or mechanical factors cause the degradation of cartilage and bone remodeling in the TMJ. OA of the TMJ also known as a degenerative and inflammatory disorder is an age-related condition characterized by the progressive destruction of articular surfaces of the mandibular condyle and glenoid fossa in which sustained loading of those surfaces predominate [18]. OA may also be defined as "a metabolically active repair process that takes place in all joint tissues and involves loss of cartilage and remodeling of the underlying subchondral bone" [19]

XIII. Etiology

Joint overload from parafunctional behaviors may also be a frequent cause of both arthralgia and OA of the TMJs. Joint overload is a complex disorder associated with oral parafunctions, acute or chronic trauma, unstable occlusion and increased joint friction [2] probably associated with disc displacement in which the joint disc no longer protects the joint surfaces. Excessive and chronic overload to the joint disc, bone and cartilage causes fibrillation of cartilage leading to biomechanical failure impairing the normal sliding function of the joint surfaces. Such failure may be suspected by the presence of joint clicking whereas anatomic damage and deformation of the condylar head is shown by the presence of crepitus in the joint during careful palpation on closing and opening. Because a set of parafunctional behaviors are present persistently in many individuals, this set of behaviors characterized by sustained forces, overcomes the threshold for adaptation and resistance to such forces thus resulting in inflammation, pain, synovitis, disc displacement, deformation and excessive pressure or compression. Sustained tension or compression maintains pain and set the stage for the development of OA changes. Synovitis with the presence of inflammatory cells and inflammatory cytokines are probably the first reaction to such sustained load from parafunctional jaw habits.

Overload is not the only the cause of TMJ arthralgia. Other factors include macro traumatic events, for instant, motor vehicle accidents, trauma from surgical interventions like extraction of third molars, orthognathic surgery, severe distention to the orofacial region during intubation, a blow to the face and so on. Pain in TMJ arthralgia is usually described as dull, sharp or burning. It is likely that anatomic displacement of some nervous structures may cause trauma to nerve terminals in the restricted anatomic space in the joint. In line with these observations, one investigation [20] indicates that the auriculotemporal nerve may have its course almost in contact with the condyle, the masseteric nerve may be compressed during excessive condylar translation and the deep posterior temporal nerve may pass too close to the anterior insertion of the joint capsule on the temporal bone. Further, the inferior alveolar and the lingual nerves may pass close to the anterior part of the condyle. In such cases, the aforementioned nerves may be exposed to irritation, anatomical contact and even compression resulting in the development of neuropathic pain [20].

XIV. Clinical Characteristics

OA is usually described as the result of a long pathological process associated initially with a chronic inflammatory process and many years later with a destructive mechanism characterized by the destruction of cartilage and bone. According to one investigation [21] OA pain is often described as a disorder that worsens with exercise and movement and is associated with brief morning stiffness that disappears during the rest of the day and improves with jaw movements or daily activities. Other clinical characteristics of OA include, morning stiffness during sitting, lying down or swelling, reduced, range of motion, strength, balance and proprioception and joint locking [21]. In theory joint stiffness in patients that report crepitus during jaw movements, is practically pathognomonic of OA.

TMJ OA is characterized by intense, intermittent pain on a background of persistent aching pain sometimes associated with hyperalgesia and resting pain. Pain in OA has nociceptive, neuropathic and other components. Nociceptive pain is closely associated with the degree of inflammation and surrounding tissue damage whereas neuropathic pain indicates some degree of nerve damage. Neuropathic pain in OA is caused by a lesion within the somatic and sensory systems and is characterized by burning or electrical shooting pain often triggered by light touch [21].

XV. Prevalence

OA may be a common condition in the general population. Notwithstanding this, OA does not occur very frequently in the population of TMD patients and its prevalence varies according to the method used in the

examination process. The prevalence of OA is very low in groups of patients with TMDs. Approximately 10% of TMD subjects present with signs and symptoms of OA using clinical examination methods and this prevalence may be higher when using computed tomography and/or MRI. TMJ OA is the most common degenerative joint disease in the masticatory system. The frequency of this condition is approximately 11% in subjects with signs and symptoms of TMD [22]. However, its prevalence may increase if using transcranial, computed tomography and or MRI methods in the diagnosis process. The frequency of OA is higher in females, its prevalence and severity increase with age, thus, higher frequencies may be observed in the sixth and seventh decades of life [22]. When using a combination of clinical and MRI methods, the prevalence of OA may increase to 25% in subjects in the age range of 20-49 years old [23]. The use of CT and/or MRI drastically inflates the prevalence of OA in subjects presenting with TMDs. Alzahirani and associates [22] evaluated a large sample of 145 randomly selected computed tomographic scans of 145 subjects presenting with signs and symptoms of TMDs and reported a prevalence of 65,5% of OA signs using appropriate imaging criteria.

XVI. Pathophysiology

Mechanical and biological events associated with tension, pressure, compression, elongation and deformation that occur concomitantly with local inflammation initially result in arthralgia and posteriorly in cartilage and subchondral bone alterations resulting in degeneration, resorption, destruction, erosion, flattening, sclerosis and osteophyte formation leading to severe deformation and loss of function in different joint structures. As resorption, deformation and destruction continue, the inflammatory events become less relevant with time and degeneration becomes the major event. Even though pain may be reported by OA patients, this pathological response is not so significant as compared to the biological and pathological events that ultimately result in severe joint deformation. Pain [24] in patients presenting with signs and symptoms of arthralgia may arise from stimulation of numerous pain receptors found in the posterior attachment of the joint where pressure and or damage to discal attachments occurs frequently in cases of both micro and macro traumatic events [24]. A macro traumatic event is a sudden and short-lasting source of trauma that causes severe damage to the joint whereas micro trauma is repetitive, more frequent in which low grade but destructive forces operate.

There is a strong psychosomatic component in OA patients. Such characteristics render OA patients more vulnerable to musculoskeletal diseases, loss of the effectiveness of descending inhibitory pain mechanism, hyperalgesia, lower pain thresholds and increased sensitivity to pressure, ischemia and innocuous warm stimulus. Further, a widespread mechanism controlled by the central nervous system, modulates the response of the individual to OA [25]. An ineffective descending inhibitory system, may indirectly enhance ascending trajectories responsible for the maintenance of chronic pain, thus, contributing to more long-lasting pain in OA through a decrease in the release of some substances including serotonin and descending endogenous opioids. OA is significantly influenced by both systemic and psychological disorders including somatization.

Damage to nerve terminals normally supplying the subchondral bone has been implicated in the nociceptive mechanisms of neuropathic pain in OA. It seems apparent that abnormal firing activity of the damaged nerves contribute to chronic pain [26]. It is very likely that these neurophysiological or neuropathic alterations that result in pain may be more associated with arthralgia than to OA and may be described more frequently in those patients in the initial process of the disease.

XVII. Diagnostic Considerations

Even though some observations during the initial interview including evaluation of pain history, patient´s age, the disease as a long pathological process and the description of a burning joint pain, may contribute with the diagnosis of OA, some signs or characteristic found using tomographic or MRI images may contribute the most in the diagnostic of OA. Kothari and associates [6] assert that erosion, osteophyte formation, generalized sclerosis and the formation of subcortical cysts are determinant in the diagnostic process. Further, synovial hypertrophy, effusion formation, increased vascularization and disc displacement [6] observed if appropriate imaging methods are used may further contribute with the diagnosis of OA. At the clinical level when using questionnaires clinical examination and imaging methods in OA patients, the clinician frequently reports that frequency and intensity of pain are not proportional when compared to the severity of joint damage, for instance, a very displaced and deformed disc, presence of osteophytes or beaks, superficial condylar flattening, condylar deformation and subchondral bone resorption. This observation is corroborated by the studies of Das [7] reporting that in TMJ OA, the clinical and radiological signs of OA may increase while the pain is decreasing. In other words, pain is more severe and probably occurs more frequently in patients with disc displacement with reduction and intermittent locking than in OA subjects.

XVIII. Treatment Or Management Of OA

The treatment of TMJ-OA is highly influenced by the clinical and psychological profile of the patients including chronicity, severity and frequency of pain, psychological factors, comorbidity and level of damage to

the joint structures. Notwithstanding this, the objectives of treatment include reduction of pain and inflammation, elimination of abnormal patterns of jaw movements, decrease tension and stiffness in the masticatory muscles, neutralize the role of parafunctional behaviors and reduction of anxiety, depression and somatization. Such objectives may only be attained when the patient is managed and treated using a multidisciplinary approach in which the role of psychological factors is also neutralized and the clinical complaints are treated or managed.

According to one investigation [8] conservative options are used in the first step of treatment and include the use of occlusal devices to manage joint associated with parafunctional behaviors, jaw stretch, pain killers and muscle relaxants in order to eliminate abnormal functional disorders of the masticatory muscles, local anesthesia to manage pain and contracture in trigger points in the masticatory system, patient instruction to use muscle exercises or jaw stretch, heat and massage to improve jaw function, antidepressants including amitriptyline to reduce depression and anxiety.

In the last two decades the use of minimally invasive modes of treatment including arthrocentesis and arthroscopic lysis and lavage has become popular and is used more frequently to eliminate effusion, inflammatory cytokines and residues of oxidative stress. Arthroscopic lysis and lavage are used to reshape the articular surfaces and to eliminate waste products, fibers, residues of oxidative stress, elements that facilitate a state of chronic inflammation. Because [8] conservative modes of therapy are less likely to induce damage on the articular surfaces, they are frequently used in the early stage of treatment.

Internal derangements of the TMJ including OA do not occur as isolated disorders. A long-standing condition favors the development of associated diseases including myofascial pain of the masticatory and cervical muscles. Thus, additional modes of treatment in the domain of physical therapy becomes necessary. It [8] has been proved that myalgia and myofascial pain respond positively to the use of physiotherapeutic techniques. Drugs that protect the life of surviving chondrocytes are capable of restoring the metabolic balance of cells in the TMJ cartilage and have both analgesic and anti-inflammatory effects {2}. A combination of glucosamine and chondroitin being structural molecules in the normal joint cartilage may be necessary in the synthesis of both proteoglycan and glycosaminoglycans [2]. Additional modes of treatment to reduce pain, inflammation and restore normal muscle and joint function include laser sessions, anti-spasmodic and muscle relaxant drugs (cyclobenzaprine), repositioning exercises [2] and electrical stimulation.

The goals of treatment of TMDs including arthralgia center around the elimination of pain, inflammation and disability, for instance, poor jaw function. Additional, other goals include reduction of tension, stiffness, and poor muscle function associated with parafunctional behaviors. Because conservative methods are less likely to cause any harm to the patient, the careful clinician opts for the use of conservative methods, for instance occlusal splints, analgesics, anti-inflammatory drugs and muscle relaxants in the initial stages of the disease [8].

One investigation carried out by Vos and associates [26] in patients presenting with signs and symptoms of arthralgia used arthrocentesis compared to conservative therapy and reported that from an economical point of view, arthrocentesis produces rapid clinical results and is less expensive when compared to conservative treatment of TMJ arthralgia.

Current modes of treatment for arthralgia of the TMJ usually center around reducing joint load from micro or micro trauma related to parafunctional habits and unstable occlusion and elimination of local inflammation. Thus, a soft diet, manual exercises at home including jaw stretch, pain killers and anti-inflammatory drugs, use of a stabilization or an anterior superior bite splint are recommended to accomplish such goals. Even though this therapeutic approach may be very effective, in actuality is highly dependent of patient´s cooperation. The advantages of arthrocentesis include its proved effectiveness, is considered a minimally invasive technique, symptoms often disappear rapidly and reduces medical and dental costs [26]

XIX. Reasons Or Evidence That TMJ Arthralgia Is An Independent Disorder

What follows is a summary of relevant evidence indicating that TMJ arthralgia and TMJ OA are different, independent yet correlated disorders:

1.Pain in TMJ arthralgia is described as burning, dull, aching or sharp. Pain in TMJ OA is not described as burning.

2.Many researchers establish a differentiation between TMJ arthralgia and TMJ OA

3.TMJ arthralgia is a panful inflammatory disorder with minimal bone or cartilage alterations whereas OA is considered as a less inflammatory, but more degenerative disorder. In one investigation in TMD subjects using imaging techniques, researchers found that 1/3 of joints of OA patients were painless [27], thus, corroborating that OA is in essence, a more degenerative disorder.

4.In one investigation, mean age in TMJ arthralgia was about 35,8 as compared to 46,8 in a subgroup of subjects with both arthralgia and OA and the difference was statistically significant (p=0,02).

5.Arthralgia is a less chronic TMJ internal derangement as compared to TMJ OA.

XX. Discussion

1.Arthralgia is an independent TMJ internal derangement that precedes the development of signs and symptoms of OA.

Because TMD patients with a diagnosis of OA rarely or never describe their pain as sharp and burning, but younger patients use this description during the diagnosis process, we hypothesized that another TMJ ID may be present immediately before the development of OA sign and symptoms. Such a disorder that in theory is more inflammatory than degenerative "paves the way" for the development of destructive TMJ mechanisms that ultimately facilitates the development of a degenerative process with time. In the current investigation, we hypothesized that arthralgia constitutes a relatively independent process different from TMJ-OA. Congruent with these points of view [28], arthralgia is described as a sudden, sharp and intense pain that is closely associated with joint movements, probably those that cause friction, rubbing or compression on delicate nociceptive structures presenting with some degree of local inflammation. Further, destruction of local tissue combined with local inflammation may lead to a loss of normal articular surfaces generating a type of pain that has its source on the subarticular bone [28].

Even though arthralgia represents the initiation of a degenerative disease in which inflammation still predominates, it is OA that is considered a degenerative condition. These considerations are congruent with one investigation [18] suggesting that "because pain related variables were not associated with degenerative bony changes, such lack of association could be explained by the difference or "time elapsed" between the onset of pain and detectable radiographic bony changes. Put it in another way: a stage in which pain predominates corresponds to arthralgia and the presence of bony changes, to OA. The stage of arthralgia is more likely to last a shorter period of time, as inflammation is more likely to induce bony changes sooner. On the other hand, a patient with OA is very likely to demonstrate bony alterations for a longer period of time as degenerative changes is a long-lasting pathological process. Additional studies are needed in this complex field of research. The assumption that TMJ arthralgia is an independent and different disorder different yet related to OA is contradicted by one investigation [17] asserting that arthralgia of the TMJ is a common symptom of TMJ osteoarthritis.

2.Many signs and symptoms or characteristics may be used by the clinician in the diagnosis of TMJ arthralgia.

A major concern in the current investigation was to discern about the most relevant characteristics or signs and symptoms which the clinician could use in the diagnosis of TMJ arthralgia. It is true that pain, disc displacement usually without reduction and difficulties to perform normal jaw movements constitute TMD characteristics. Notwithstanding this, those characteristics can be observed in other TMJ-IDs. Consequently, the clinical characteristics of interest in the current study should be those unique to TMJ arthralgia in such a way that facilitate the diagnosis for the astute clinician. Because a description of sharp and burning pain differentiates TMJ arthralgia from other TMJ-IDs, such a description has enormous diagnostic significance. Okeson [28] describes sharp, sudden and intense pain that is closely associated with joint movement as major characteristics of arthragia. In the study carried out by Padulla and associates [14], researchers used the description of burning, tingling and electric shock in order to gather data about the presence of neuropathic pain and simultaneous TMJ / IDs in a group of 82 patients with TMDs.

A medially displaced disc in patients presenting with severe TMJ/IDs, could interfere mechanically with the auriculotemporal, masseteric, and posterior deep temporal nerves and cause sharp and shooting pain felt locally in the joint during jaw movements [20]. It has been reported recently that TMJ arthralgia is initially produced by intense stimulus presumably a mechanical stress [29]. In clinical practice one may suspect of very severe nocturnal bruxing behavior causing severe mechanical stress, tension and compression as one key element in the development of signs and symptoms of arthralgia. In TMJ arthralgia patients report spontaneous pain or pain on movements of the TMJ and pain on palpation of the lateral pole or posterior attachment of the TMJ on the same side [6]. A complaint of persistent pain that is affected by jaw movements, function or parafunctions indicates the presence of TMJ arthralgia. Further, replication of this pain occurs with provocation testing of the TMJ, for instance, palpation of the lateral pole of the joint, with maximum unassisted jaw movements or during an experimental protrusive movement [30] with tooth contact. The rationale using this test is that a protrusive movement with tooth contact induces a rubbing action on the articular surfaces causing pain in areas with severe inflammation, nerve damage, local bone or cartilage destruction in which pain receptors have a low threshold for pain.

3. Because pain in TMJ arthralgia is described as burning, a neuropathic component, perhaps nerve damage may contribute to the development of this TMJ-ID.

One investigation [31] evaluated the role of sport injuries in the development of neuropathic pain in the masticatory system. Even though such disorder occurs rarely in the context of OFP and TMJ-IDs, the investigator asserted that nerve injuries in the TMJ clinically present as an area of persistent pain, represent a diagnostic

challenge and show difficulties to respond satisfactorily to several modalities of treatment, The presence of TMJ neuropathic pain may explain an unsatisfactory response to appropriate therapies for TMJ dysfunction [31]. The TMJ disk may cause damage to the mandibular nerve and its branches and this occurs when the disk causes compression, traction or rubs against the nerve during jaw opening [14]. Damage to a nerve innervating an affected joint may cause central sensitization from chronic nociceptor stimulation leading to modifications in the transmission of nociceptive information to central areas of the brain [32] Nerve damage is not recognized as a major feature of OA. Notwithstanding this, there may be a subclinical damage to small peripheral nerves innervating OA joints [32]. Because some signs or symptoms of arthralgia may overlap with those in OA patients, it is very likely that the mechanism of neuropathy explained in the investigation by Hochman and associates [32], is also valid in patients with signs and symptoms of arthralgia. In this case, the description of burning pain may have the connotation of damage to a specific group of nerve fibers.

Given the destructive character of disk displacement, severe inflammation and perhaps, the initiation of severe anatomic alterations in TMJ arthralgia and OA, nerve compression, damage and more severe pain are very likely to occur in arthralgia of the TMJ. Thus, a description of burning pain may be a reliable indicator of such changes. These observations are in line with one radiological and histological investigation [20] of the topographic relations of some nerves adjacent to the TMJ. Researchers asserted that some nerve branches of the mandibular division of the trigeminal nerve including the auriculotemporal, masseter and deep posterior temporal nerve, may be contacted or compressed by a severely displaced disc and cause mechanical irritation which would provide a neurophysiological explanation for a description of sharp, intense, intermittent, shooting pain observed during jaw movements in some patients. Another possibility is that some type of neuropathic pain explained by the description of burning and sharp may be the expression of anatomical exposure of C fibers following cartilage and subchondral bone even when such exposure occurs in a restricted anatomic area in the mandibular condyle. Thus, disc displacement and associated micro trauma including compression and mechanical friction between the bony articular surfaces may be interrelated phenomena. Even strain of some discal ligaments may cause arthralgia and some signs and symptoms of neuropathic pain [24].

Further theoretical support to the idea of a neuropathic component in arthralgia of the TMJ in considered in one investigation [19], indicating that a neuropathic component may be predominant in individuals with minor joint changes but with high levels of pain refractory to analgesic treatment. Very likely, high levels of pain, more severe inflammation and minor joint changes represent characteristics of arthralgia whereas gross bony changes would represent the osteoarthritic stage or diagnosis. A number of scientific publications provide strong support to the idea that a neuropathic pain mechanism contributes to the pain experience at least for a subset of the OA population [19]. Notwithstanding this, the same point of view can be accepted in the case of TMJ arthralgia.

4. Severe inflammation may be a major contributing factor in the mechanisms of TMJ arthralgia.

Inflammation is a powerful mechanism that usually results in pain, disc displacement and initiation of a destructive response in the TMJ. Because TMJ-OA has been correlated with a destructive and remodeling process, we assumed that more intense inflammation would be found more frequently in subjects with arthralgia and disc displacement without reduction. TMJ arthralgia without associated general disease is a painful disorder assumed to be associated with local inflammation. This disorder is also described as inflammation of the synovial lining of the TMJ [33]. Inflammation of the articular surfaces (arthralgia or arthritis), constitute a subgroup of TMJ disorders in which there is an alteration in the morphology of the osseous tissue [34] and may play a more significant role in TMJ arthralgia and degenerative joint disease (DJD) based on associations with several altered markers in the joint or in the synovial fluid [35]. In both arthralgia and DJD, the synovial fluid of the TMJ contains elevated levels of proinflammatory cytokines, matrix degradation enzymes and breakdown products that often are directly related with symptoms of pain, restricted mouth opening, locking and joint noises [10].

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Table 1: Authors that make reference to TMJ Arthralgia as a diagnosis of TMJ internal derangement no just as °joint pain°.

Authors	Relevant information about arthralgia
Khotani and associates J. Headache and pain 2016.	Authors evaluated 58 TMD patients using different imaging techniques. After each examination patients were classified into osteoarthritis or arthralgia.
Tang and colleagues. JOMFS, 2023	Arthralgia of the TMJ is common type of TMJ disorder that affects 8% of the general population. This disorder is closely associated with the presence of TMJ osteoarthritis.
Li and Leung Diagnostics 2021	Arthralgia of the TMJ, headache attributed to TMDs, myalgia and myofascial pain constitute painful TMJ disorders described in axis I of the DC/TMDs.
Harrison, Thorp, Ritzline JOSPT 2014	TMJ arthralgia may be due to inflammation and/or degeneration of the articular tissue. Arthralgia may occur with or without degenerative alterations.
Milan SB Oral Maxillofac Clin NA 2000	It is very likely that TMJ arthralgia is initially produced by an intense stimulus, presumably a mechanical stress. Pain may be the result of both injury and inflammation with the release of inflammatory molecules.
Cerda-Trujillo et al. Avances en Odont 2015.	Arthralgia of the TMJ is defined as a pain of joint origin that can be induced by jaw movements and/or pressure, function and or parafunctions.

Table 2: Clinical characteristics of arthralgia of the TM

Author Publication	Clinical description and/or characteristics
Okeson JP. Management of TMDs. Elsevier 8th edition	Arthralgia from healthy structures of the joint is described as sharp, sudden and intense pain that is closely associated with joint movement.
Finnerup P et al. Physiol Rev 2021	Burning pain (characteristic of arthralgia and neuropathic pain), can be elicited by intraneural stimulation of nociceptive C fibers. Burning pain is closely associated with nociceptive C fibers that attempt to regenerate.
IASP 2009	Absence of coarse crepitus is a characteristic of TMJ arthralgia.
Molina and associates. J.Dent Med Sci 2020.	A long history of TMJ pain, pain described as burning, high frequency of reciprocal click, ill-defined crepitus, pain induced during experimental protrusive jaw movements as rubbing against inflamed and unprotected joint surfaces causes pain, constitute some characteristics of arthralgia of the TMJ.
Thirumaran AJ et al J Pers Med 2023	Nociceptive pain indicates and ongoing joint inflammation and surrounding tissue damage while neuropathic pain in osteoarthritis indicates a degree of nerve damage. In osteoarthritis pain may be the result of sensitization of peripheral receptors in the synovium and subchondral bone.
De Souza JB. Rev Cienc Saude, 2017.	Arthralgias are frequently describe as inflammatory pains that occasionally present a neuropathic component.
IASP 2009	Arthralgia of the TMJ may result from trauma or from intrinsic and extrinsic overloading of the TMJ.
Pedulla et al. AJNR 2009	Neuropathic pain component in the TMJ may be associated with a sort of nerve damage associated with rubbing, traction, friction or compression of the nerve.
Harrison et al. JOSPT 2014	TMJ arthralgia may be caused by inflammation and/or degeneration of the joint structures.
Lovgren et ali Clin Oral Invest 2019.	Forceful contralateral biting that causes pain in the opposite side, may indicate the presence of arthralgia of the TMJ.

Table 3: Additional information or evidence indicating that arthralgia precedes the development of OA signs and symptoms.

Authors and source of information	Relevant information
Molina et al. Cadernos Unifoa 2010.	Patients presenting with signs and symptoms of arthralgia are usually younger as compared to OA patients.
Khotari et al. J. Headache and Pain 2016.	Arthralgia and OA are different types of TMJ-IDs.
Cordeiro et al. Acta Odont Latinoam 2016	The presence of arthralgia may indicate an early stage of OA.
Das SK. J Oral Biol Craniofac Res 2013	Inflammation predominates before the onset of an osteoarthritic process. When OA signs and symptoms can be observed, inflammation decreases and destructive processes predominates.
Khotari SF et al. J. Headache Pain 2016	Joint effusion is a sign that may be observed before osteoarthritic changes occur.