EDENTULISM AND TEMPOROMANDIBULAR JOINT-A MINIREVIEW

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ABSTRACT:

One of the most complicated joints in the human body is the temporomandibular joint (TMJ). The disc of the joint is crucial to the joint's normal function because of the unusual combination of rotation and translation movement it undergoes. Temporomandibular joint disorders (TMD) are defined as clinical problems that affect the masticatory muscles, temporomandibular joint (TMJ) and the related structures or both. Since the teeth are one of the most important components of the masticatory system and have a close relationship with muscles and TMJ they can induce changes in these components.

Key words: *TMJ disorder, edentulism, etc.*

BACKGROUND

Among the human body's most complicated joints is the temporomandibular joint (TMJ), which plays a significant role in dental occlusion and the neuromuscular system [1]. The TMJ is a complex joint, which by definition requires at least three bones, however it is really made up of only two. Okeson reasoned that the articular disc is a nonossified bone whose primary role is to promote joint mobility in order to permit more complex movement [2,3].

In contrast, McKay et al. [4] considered the TMJ to be two separate joints, one above and one below the other, on the grounds that the articular disc between the mandibular fossa of the temporal bone and the condyle is not a true bone. In a healthy disc, the articular portion is made up of dense, fibrous connective tissue that lacks nerves and blood arteries, while the posterior attachment is heavily vascularized and innervated [5,6].

While rotation occurs in the space between the condyle and the inferior surface of the disc during early opening (the inferior joint space), translation happens in the space between the superior surface of the disc and the fossa during late opening. TMJ articulating surfaces are coated with thick fibrocartilage as opposed to hyaline cartilage like most synovial joints [7]. In comparison to hyaline cartilage, it is more resilient to the effects of time, less likely to degenerate, and has a greater capacity for repair [8]. Due to the widespread presence of symptoms, TMJ functional abnormalities are likely the most often observed pathology.

LITERATURE REVIEW

- *History*

Only a small number of dentists became interested in treating mandibular dysfunction discomfort [9,10] in the late 1930s, when bite-raising appliances were the primary treatment option. By the 1950s, dentists were beginning to dispute the efficacy of appliance therapy and focus instead on occlusal interferences as the root of many TMD problems [11,12].

In the 1950s, researchers discovered that electromyographic investigations may be used to connect relationships between

occlusal position and masticatory muscle performance [13,14]. It wasn't until the 1970s that researchers realised that occlusion and, later, emotional stress were key contributors to masticatory system dysfunction.

Then in the 1970s, as documented by Farrar and McCarty [15], there was a flurry of activity about pain disorders with intracapsular origins. Until the 1980s, the profession did not completely recognise and grasp the intricacy of TMDs and orofacial aches, nor did it actively seek to identify its proper role in their therapy.

- *Epidemiology*

Approximately 4% of persons (between the ages of 18 and 44) who have never had TMD before will develop clinically verified primary onset painful TMD each year. Peak prevalence of TMD has been recorded between the ages of 35 and 44, at 4.5 % [16].

Due to differences in data collecting, descriptive terminology, analytic methods, and the individually selected criteria, the prevalence of TMDs from cross-sectional epidemiological research differed greatly from study to study. When only studies using the Research Diagnostic Criteria for TMD [17,18] were considered, a systematic review indicated a prevalence of up to 13% for masticatory muscle pain, 16% for disc derangement disorders, and 9% for TMJ pain disorders in the general population.

Persistent TMD is more common in women than men, but women have a somewhat higher risk of being impacted.The age-specific incidence pattern varies significantly between ethnic groups, according to reports [19].

- *Etiology*

There has been a lot of back and forth over the years on what causes TMD and what the underlying pathophysiology is. As with other types of chronic pain, TMD fits the profile of the biopsychosocial model of illness, which has recently gained acceptance in the medical community.Individual differences in psychological make-up and pain threshold have been linked to TMD vulnerability [20,21].

Associations between genetic risk factors for clinical, psychological, and sensory characteristics and the development of TMD were found in a study of single-nucleotide polymorphisms (SNP). Five single-nucleotide polymorphisms (SNPs) out of a total of 3295 investigated, representing 358 genes involved in pain perception, were found to be highly predictive of TMD and pain prevalence [23].

Researchers have discovered that the activity of catecholamine-O-methyltransferase (COMT) has a significant impact on pain sensitivity and the onset of TMD via adrenergic pathways. Catecholamines and enkephalins are engaged in a wide variety of neurological processes, and this enzyme plays a role in their control [24].

These potential processes and pathways that may predispose, begin, or maintain chronic pain in the TMD do not operate in isolation. They probably work together to make the neurons in the brain's peripheral and central nociceptive pathways more excitable and boost the efficiency of their synapses. Trigeminal neuralgia (TMD) has been linked to cerebral and peripheral sensitization, namely sensitization of trigeminal neurones and amplified pain signalling.16 Since TMD patients generally have higher peripheral and central sensitization, they are also more likely to suffer from other types of persistent pain, such as headache and fibromyalgia.

- *Diagnosis*

TMDs are complicated and multifaceted, making it difficult to pinpoint a single cause. Predisposing variables are those that raise the likelihood of developing TMDs, initiating factors are those that lead to the development of TMDs, and perpetuating factors are those that impede healing or promote the advancement of TMDs. The development of TMDs cannot be attributed to a single etiologic component, nor can it be explained by a single theoretical paradigm.

Several methods of evaluating TMD have been proposed over the years. The Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) is the most well-known of these instruments [25].The DC/TMD's stated purpose is to offer a standardised and operationalized instrument

for the diagnosis of TMD by means of screening for psychosocial and comorbid variables and physical evaluation of the masticatory structures .

- *Clinical changes*

Digital palpation, in which the fingertips are put over the lateral sides of both joint locations at once, reveals pain or soreness and joint noises. For a proper clinical assessment of posterior capsulitis and retro discitis, the patient should be in a fully extended position while the fingers are rotated slightly posteriorly to apply pressure to the posterior portion of the condyle. Instead, then only feeling around the joint, a doctor can use a stethoscope to listen for additional sounds.

The TMJ is an intricate joint that allows you to open and close your mouth. Major extracellular matrix (ECM) components of joint function include the articulating surfaces of the mandible condyle and the temporomandibular joint (TMJ) disc. Fibrocartilage tissue in a TMJ acts as a lubricant for the joint's moving parts, making it possible for the joint to perform its complicated mechanical functions. The viscoelastic compressive characteristics of the tissue may be responsible for the reduction of joint friction at the TMJ disc and articulating surfaces [26-28].

When the TMJ is afflicted by degenerative change, dysfunctional remodelling is simple to achieve.

Wear and tear on the articular surfaces are signs that the body is under more stress than it can handle. Internal disarray and subsequent inflammatory changes result from these alterations.

Synovial fluid from the TMJ is another possible avenue for uncovering the disease's root cause. The articular disc makes it easy to divide the TMJ into an upper and lower cavity when discussing its anatomy. Type A synovial lining cells are similar to macrophages, and type B synovial lining cells are similar to fibroblasts; these cells create collagen and fluids that facilitate pain-free jaw movement [29,30].

More and more research has been conducted in recent years to better understand the correlation between TMJ synovial fluid changes and internal disarray. Biomolecular analysis reveals that TMJs with internal disarray produce more cytokines such IL-1b and IL-6. These cytokines play a crucial role in the pathophysiology of TMD because they stimulate the destruction of cartilage and bone by triggering the production of proteinases and inflammatory mediators. various investigations have already rearranged the interaction of various cytokines between up- or downstream [64], and this includes some pro-inflammatory cytokines. The mechanisms of TMD should be well understood [31-33].

- *Conservative treatment*

a. **Education**: a patient's uncertainty around the diagnosis of TMD can significantly affect their quality of life and may lead to care pathways that are unsuccessful or aggravate the presenting complaint further.23 It is therefore very important to educate patients on the usually non-progressive and benign nature of TMD at an early stage [34].
b. **Self-care techniques:** stopping parafunctional habits (such as nail biting, teeth grinding/clenching, or chewing gum), consuming less caffeine, and practising better sleep hygiene are also recommended. Direct application of cold or warm packs to the affected muscles may also help alleviate symptoms. Finally, diaphragmatic breathing exercises and meditation can help you unwind completely and fortify your resilience [35].
c. **Intraoral appliances**: a splint (either soft polyethylene or hard acrylic) worn over the upper or lower teeth is a widely recognised treatment for TMD [36].
d. **Physical therapy and acupuncture:** physiotherapy, like as massage or specific exercises, may help with TMD, but the evidence is weak [37-39].
e. **Pharmacotherapy**: simple analgesics, neuromodulatory medications, and intramuscular injectables are just some of the

pharmaceutical options for treating TMD. Medication for chronic pain should be used in conjunction with other treatments, and doctors should weigh the benefits of drug use against any downsides or interactions [40].

- *Irreversible and invasive treatment*

a. **Surgical management**: Interventions including arthroscopy, arthrocentesis, arthroplasty or joint replacement can be considered for patients with arthrogeneous TMD [41].
b. **Secondary care or specialist management**: Referral to secondary care is suggested if the diagnosis of TMD is unclear or if symptoms have persisted or worsened over 3 months despite primary reversible treatment. Further to this, if there is marked psychological distress associated with symptoms or if there is unexplained persistent pain or chronic widespread pain, secondary care referral should be considered as these factors are associated with a poor long-term prognosis.

PERSPECTIVES AND HYPOTESIS:

Based on this literature mentioned above, it can conclude that there are more and more studies tend to discuss about inflammatory micro-environment in TMJ and try to clarify the tissue response between TMJ disc and condyle change during TMD. T MJ with internal derangement is no longer considered as a derangement condition of TMJ disc but affected the entire TMJ. In the future, when TMJ degradation happen, the strategy of TMJ treatment should be multidirectional instead of persisting in one point.

CONCLUSIONS:

TMD are a complex group of illnesses that can cause long-lasting discomfort. The complex aetiology of painful TMD is becoming better understood as research progresses. This will likely lead to the creation of better techniques for preventing, diagnosing, and treating TMD that target specific pathways and causes.

In order to effectively treat TMD, it is recommended to use a biopsychosocial paradigm of care that incorporates a multidisciplinary approach through a unified treatment pathway. There is evidence to suggest that a more streamlined and cost-effective method of managing orofacial pain within secondary or tertiary care could be achieved through the formation of dedicated regional centres.

Clinic reports suggest that more than 20% of the population suffers from TMJ internal defragmentation. There is still no consensus on what caused it. Recent research confirmed its identity as a symptom of a more widespread disease. In the next decade, it is possible that regenerative medicine will replace traditional methods of treating joint disorders.

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