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ANATOMICAL VARIATIONS INVOLVED IN TEMPOROMANDIBULAR DYSFUNCTION: A REVIEW

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Abstract: There are several diseases that can affect the temporomandibular joint (TMJ), including cancer, trauma or fracture, congenital malformation, osteochondritis and anatomical factors. The aim of this study was to review the anatomical factors involved in temporomandibular dysfunction. A systematic review was carried out on PubMed on 17/06/2024 using the keywords "ANATOMICAL VARIATIONS", "TEMPOROMANDIBULAR JOINT", "TEMPOROMANDIBULAR DYSFUNCTION". Studies with adult patients diagnosed with TMJ dysfunction were included and documented anatomical variations, excluding irrelevant papers or those without full text. The data extracted (country, demographics and variations) was analyzed and presented in narrative text and tables.The present study showed some anatomical factors that lead to TMD, which included asymmetry of the mandibular condyle, orofacial sensory changes, variation in intra-articular space, alterations in the lateral pterygoid muscle and mandibular hypomobility associated with the coronoid process. TMD can develop into worrying conditions, generating not only pain and discomfort, but also directly affecting the patient's quality of life.
Keywords: Temporomandibular Dysfunction; Temporomandibular Joint; Anatomical Variations; Tmj; Tmd; Pain.

The temporomandibular joints (TMJ) are the connections between the temporal bones of the skull and the mandible. There are two temporomandibular joints, one on each side of the face, just in front of the ears. Ligaments, tendons and muscles support the joints and are responsible for the movement of the jaw[1] . TMD is the acronym used to designate "temporomandibular dysfunction", which is the name given to the set of alterations that mainly involve the TMJ and the muscles that work on jaw movements. These conditions can be

accompanied by orofacial pain (OOF), including headaches. TMD/OFD cases are not the same. There are types and subtypes of TMD and ODD and the same person can have more than one type of TMD and ODD, which can make diagnosis difficult. TMD is most common among women in their 20s and those between 40 and 50. In rare cases, babies are born with abnormalities of the temporomandibular joints. Temporomandibular disorders include problems with the joints, the muscles and the sides of the fibrous tissue that connects them (fascia)[1,2].

Several factors are involved in TMD, the combination of muscle tension and anatomical problems in the joints. Sometimes there is a psychological component, as well as other factors, including genetic factors, a history of head and neck trauma. Clenching and grinding of the teeth (bruxism), systemic disorders (such as osteopenia, autoimmune disorders or genetic bone disorders), infections, injuries, misalignment of the teeth and even constant chewing of gum can cause symptoms. It is now said to be a "multifactorial" condition[1,2].

Symptoms of TMD include headaches, tension in the chewing muscles and clicking, popping or locking of the joints. Sometimes the pain seems to occur close to the joint rather than inside it. TMD can be the reason for recurring headaches that don't respond to the usual medical treatments. Other symptoms include pain or stiffness in the neck and shoulders, dizziness, earaches or blocked ears and interrupted sleep. People with TMD often find it difficult to open their mouths very wide. For example, most people without TMD can place their fingertips vertically in the space between their upper and lower teeth without straining. For people with TMD (with the exception of those with hypermobility), this space is markedly smaller [3,4].

Treatment depends on the type of TMD the patient has, but in general, clinical practice based on scientific evidence recommends that no irreversible treatment should be carried out. The irreversible procedures to which the authors refer are: occlusal adjustment (wearing down teeth or adding restorative material), bite correction appliances (orthodontic and/or orthopedic), and prosthetic oral rehabilitation. Even surgeries, which have been widely used in cases of TMD, have very limited indications and are performed rarely and in very specific cases. The aim of this study is to review the anatomical factors involved in TMD3,[4,5].

REVIEW

The temporomandibular joint (TMJ) occurs between the mandibular fossa and the articular tubercle of the temporal bone, superiorly, and the head of the mandible, inferiorly. The joint is laterally subcutaneous and medially related to the sphenoid spine and spinous foramen, anteriorly to the lateral pterygoid and posteriorly to the parotid gland, auriculotemporal nerve and superficial temporal vessels. As the mandible is a single bone, the two temporomandibular joints function as a unit. As such, they can be considered together as a bicondylar synovial joint of the gymnoglossus[6,7] type.

The joint capsule, which is loose, is inserted into the articular tubercle, the tympanosquamous fissure and the margins of the mandibular fossa and inferiorly into the neck of the mandible. Anteriorly, it received part of the insertion of the lateral pterygoid muscle. A separate synovial membrane covers the capsule in each of the two compartments of the joint, but covers neither the articular surfaces nor the articular disc. It has some reinforcing ligaments: the lateral (temporomandibular) ligament, which extends from the root tubercle of the zygomatic process to the lateral surface

of the neck of the mandible - it reinforces the joint laterally and with the post-glenoid tubercle prevents posterior dislocation of the joint; the sphenomandibular ligament, which is medial, extending from the spine of the sphenoid bone (and other neighboring structures: anterior process and ligament of the malleus, lips of the petrotympanic fissure) to the lingula of the mandible[6,7] .

It relates laterally to the lateral pterygoid muscle and the auriculotemporal nerve, superiorly, and then to the maxillary vessels and the neck of the mandible; inferiorly, to the inferior alveolar nerve and vessels and a portion of the parotid gland. It relates medially to the pharynx above and to the medial pterygoid muscle below. It develops from the cartilage sheath of the first pharyngeal arch. It is mainly responsible for passively supporting the mandible, although the tone of the masticatory muscles generally supports the weight of the mandible. However, the sphenomandibular ligaments act as an 'oscillating hinge' for the mandible, serving as a support bridge and as a controlling ligament for the movements of the mandible in the TMJ; the stylomandibular ligament, which extends from the styloid process to the angle and posterior margin of the ramus of the mandible, is a thickening of the fibrous capsule of the parotid gland. Some authors admit that the sphenomandibular and stylomandibular ligaments have little functional relationship with the temporomandibular joint[6,7].

An articular disc divides the joint cavity into two separate compartments: one superior, between the temporal bone and the disc, and one inferior, between the disc and the head of the mandible. It is inserted firmly into the neck of the mandible, so that the articular disc adapts to the shape of the surfaces and to joint sliding movements; it is concave-convex on the upper surface and concave on the lower surface. The disc is an oval sheet of fibrous tissue (sometimes containing areas of fibrocartilage), the circumference of which connects to the joint capsule. Behind it, it is lost in elastic fibers and a retroauricular venous plexus. Anteriorly, the disc is anchored to the lateral pterygoid tendon. The disc is strongly attached to the condyle, so that it follows the mandible in sliding movements. The upper surface of the disc is concave-convex and the lower surface concave. The disc is irregular in thickness, but rarely perforated[6,7] . In an anteroposterior view, the disc is semilunar and often slightly thicker medially than laterally. The thickness of the disc in the middle part is approximately 1 mm, and anteriorly and posteriorly, it is approximately 2 and 3 mm, respectively. Excessive displacement of the condyle is restricted by the temporomandibular ligament located on the lateral wall of the capsule [8].

The muscles of mastication are the masseter, temporalis and medial and lateral pterygoids. The masseter is connected to the maxillary process of the zygomatic bone and the zygomatic arch (proximally) and the angle and ramus of the mandible (distally). It mainly elevates and extends the mandible. While the temporal also elevates the mandible, its attachments proximal and distal to the temporal fossa of the temporal bone and to the coronoid process and anterior border of the ramus of the mandible, respectively, are better suited to retracting rather than protracting the mandible[9] . The medial pterygoid assists in mandibular elevation and protrusion through its attachments to the lateral pterygoid plate and medial surface of the ramus of the mandible. While the medial pterygoid also facilitates lateralization movements. The synergistic action of the temporalis, masseter and medial pterygoid muscles closes the mandible vertically during mastication. In contrast, the lateral pterygoid is divided into two heads, both closely related to the TMJ[9]. Classically, the

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superior head of the lateral pterygoid extends from the infratemporal crest of the sphenoid bone and inserts onto the anterior surface of the articular disc. Based on the connections of the superior head, several researchers have suggested that it may contract to pull the disc forward during mandibular depression. Hyperactivity of the superior head of the lateral pterygoid can easily be implicated in anterior displacement of the articular disc [9].

Bilateral contraction of the inferior head works with the suprahyoid and digastric muscles to pull the mandible anteriorly and inferiorly out of the mandibular fossa during mandibular opening [9] . The superior head of the lateral pterygoid is mainly active during mandibular elevation and the inferior head is active during mandibular depression. The ipsilateral activation of the superior and inferior heads of the lateral pterygoid has also been implicated in ipsilateral and contralateral jaw movements, respectively. In doing so, the lower head can provide the horizontal forces required for mastication and parafunctional activities. Thus, while the masseter, temporalis and medial pterygoid provide the forces necessary for mastication in the vertical plane, the lower head of the lateral pterygoid appears to facilitate horizontal forces. The reciprocal actions of the superior and inferior head in the horizontal plane also suggest a role in fine motor control of the mandible during jaw movements. Perhaps the best argument for the distinct functioning of the two heads of the lateral pterygoid muscle is their neural innervation. The superior head is innervated by the buccal nerve, while the inferior head is innervated by the mandibular nerve trunk [9] .

There are various diseases that can affect the TMJ, including cancer, trauma or fracture, congenital malformation and osteochondritis [10]. temporomandibular dysfunction (TMD) is a general term that includes a series of clinical alterations involving the TMJ and related structures, such as the masticatory muscles. These muscles are often related to TMD as they receive an overload, usually caused by parafunctional habits and occlusal disorders, so that the clinical manifestation of this condition is translated into pain[11]. TMD is a complicated and multifactorial condition and although the exact etiology is still unknown, genetic, anatomical and hormonal factors seem to predispose the joint to problems[9] .

In 1934, an otorhinolaryngologist called James Costen described a series of signs and symptoms, including pre-auricular pain, which came to be called Costen's syndrome [12]. After Costen's publication, a concept of TMD developed and publications began to address a syndrome rather than specific pathologies such as arthritis, dislocation, subluxation or ankylosis. Over the next 20 years, the name of the syndrome changed, and many of the signs and symptoms described by Costen were not related to TMJ involvement, and most of his anatomical explanations were incorrect [13]. However, both doctors and researchers still continued to make the same mistake - diagnosing patients with a variety of etiologically unrelated conditions because the symptoms were similar. Today, many still diagnose conditions that involve TMJ problems with a condition that mainly involves the muscles of mastication (orofacial pain - OFP). Some patients with DOF may ultimately develop degenerative joint disease, or vice versa, but there is no reason to include them in the same diagnostic classification. The similarity of clinical findings in patients with various forms of TMD and those with ODD has led to an ongoing search for more objective technologies to help improve clinical diagnostic capabilities [13].

METHODOLOGY

A systematic PubMed literature search was performed on June 17, 2021, using the keywords "ANATOMICAL VARIATIONS", "TEMPOROMANDIBULAR JOINT", "TEMPOROMANDIBULAR DYSFUNCTION". All identified keywords and terms were combined using the "OR" operator and the "AND" operator. Additional articles were retrieved by screening the reference lists of the studies.

To be included in the final review, the following inclusion criteria were used: (1) included patients diagnosed with TMJ dysfunction; (2) publications were original full-text articles; (3) the mean or median age of the study population was above 18 years; (4) there was some anatomical variation involved in the case; (5) the number of patients with corresponding imaging features were reported in the study. Studies were excluded if (1) they related to other diseases unrelated to temporomandibular joint dysfunction or (2) they did not have full texts available.

Studies selected for inclusion were assessed for methodological quality. The following categories of data were collected when available: country, patients, demographics and anatomical variations found. The results were presented in a narrative form, including tables to aid data presentation where appropriate.

ANALYSIS OF

This literature review included 8 articles, which totaled 496 patients with TMD. Information was extracted from these 8 studies regarding the anatomical factors that led to TMD, the total number of patients and the number of women and men used in the study (Table 1). With regard to TMD, we found that the most common anatomical factors found were: asymmetry of the mandibular condyle, orofacial sensory changes, variation in intra-articular space, alterations in the lateral pterygoid muscle and mandibular hypomobility associated with the coronoid process.

The subjects in the study by Sheppard[14] exhibited considerable condylar anatomical abnormalities and discrepancies even in the absence of subjective symptoms. Differences in the length of the mandibular condyle--ramus from one side of the mandible to the other were found. Bone enlargements and irregularities in the region of the angle of the mandible have been observed and appear to be associated with increased muscle activity. All these anatomical changes are visible on routine panoramic radiographs and can be predisposing factors for subjective symptoms, especially when altered maxillomandibular relationships are made. These variations or abnormalities can act as predisposing factors for TMD and give rise to symptoms following stress, trauma, or iatrogenic insult[14] .

Dysesthesia is an abnormal and unpleasant sensation produced by normal stimuli. Paresthesia is an abnormal sensation, such as burning, tingling, pins and needles or a tingling sensation. Hyperesthesia is increased sensitivity to stimulation. Hypoesthesia is decreased sensitivity to stimulation. The trigeminal nerve is mainly a sensory nerve with a small motor component. It has three divisions: the ophthalmic, maxillary and mandibular. Branches of the third division, the mandibular nerve, are the most susceptible to entrapment because of their close relationship with muscles, especially the lateral pterygoid. Spasms in the lateral pterygoid muscle due to active trigger points can result in entrapment of the nerve, causing weakness, numbness, TMD and paresthesia in individuals with normal anatomy[15].

Fushima et al (2003)[16], analyzed the TMJ space from data obtained after reconstruction and animation of the TMJ with real anatomy and kinematic data. The results showed that during unilateral mastication the minimum joint space in balancing is smaller than the working joint. This suggests greater loading on this side. It seems to occur more in the medial part of the posterior inclination of the articular

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Anatomical factors	No. of Studies	No. (%) of reported cases / total no. of patients	Number of women and men (M/H)
Asymmetry of the mandibular condyle [14]	1	62/286 (21,5%)	177/109
Orofascial sensory changes [15]	1	4/30 (13,3%)	25/5
Variation in intra-articular space [16]	1	9/10 (90%)	7/3
Changes in the lateral pterygoid muscle [11,17,18]	3	56/165 (33,9%)	113/52
Mandibular hypomobility associated with the coronoid process [19,20]	2	5/5 (100%)	2/3

Table 1 - Anatomical factors that cause TMD.

*Data taken from the articles: Sheppard, (1982)[14], DuPont *et al.* (2003)[16], D'Ippolito *et al* (2010)[11]; Yilmaz-Taskaya *et al* (2005)[17]; Stratmann *et al* (1997)[19]; Zhong *et al* (2009)[20].

eminence and at the end of the closing phase. In addition, the reduction in the minimum joint space seems to decrease over the chewing cycles, coinciding with food comminution. Abnormal mechanics lead to stresses on healthy structures and may be related to articular cartilage degeneration, osteoarthritis and TMD.

The lateral pterygoid muscle makes a unique contribution to mandibular movement control by virtue of its attachments to the TMJ disc and condyle. The superior head of this muscle contributes mainly to the apposition of the disc, condyle, and eminence in the closure of the mandible, while the inferior head contributes mainly to the opening of the mandible. The function of the lateral pterygoid muscle is complex. The two heads of the lateral pterygoid muscle are reciprocally innervated so that during mouth opening the lower head contracts and the upper head relaxes, while during mouth closure the situation is reversed. However, the role of the superior head in influencing disc and condyle rotation and translation and the relationship with internal derangement and TMD is considered[18] . The theory of internal derangement of the TMJ involves anterior displacement of the disc, which may be the result of hyperactivity of the lateral pterygoid muscle. Since the superior head only inserts into the disc, it will dislocate the disc more anteriorly. This will reduce the function of the superior head and cause muscle atrophy [11]. Pathological changes have been found more frequently in the superior rather than inferior head of the lateral pterygoid muscle. The activity of the inferior head is significantly greater when the superior head loses its function of stabilizing the articular disc. Spasm of the lateral pterygoid muscle will cause the disc to dislocate and then atrophy and degeneration of the lateral pterygoid muscle will cause the disc to continue to dislocate. Trauma, functional overload, joint laxity, parafunctional habits and degenerative joint diseases, and increased friction between moving parts are considered to play an important role in the causes of disc displacement[17] .

Hypomobility of the mandible can be caused by a change in the size and shape of the coronoid process or by structures and pathologies that surround it. Bilateral coronoid process hyperplasia can develop, with painless progressive limitation of mandibular movement, due to the increased impact of the coronoid process on the adjacent zygomatic process and zygomatic arch[19] . Jacob's disease is a rare condition, can be considered a subtype of coronoid process hyperplasia, and its incidence rate is very low. This pathology causes mandibular hypomobility and restricted mouth opening. TMD is related to hyperplasia of the coronoid process, and hyperplasia of the coronoid process was also one of the consequences of articular disc displacement. Hyperplasia and enlargement of the coronoid process are directly related to TMD [20].

CONSIDERATIONS

This study was limited to articles published in English. Another limitation of the study is that most of the studies included did not distinguish between patients with mild, moderate or severe symptoms. In addition, many did not report whether the patients had any comorbidities or other diseases that could be associated with TMD.

This literature review provides an insight into the anatomical factors involved in the onset of TMD. The present study showed some anatomical factors, such as mandibular condyle asymmetry, orofacial sensory changes, intra-articular space variation, changes in the lateral pterygoid muscle and mandibular hypomobility associated with the coronoid process.

The diagnosis of TMD is usually obtained through a thorough history and careful physical examination, but the clinical similarity of the findings in TMD patients to those in DOF patients has led to an ongoing search for more objective technologies to help improve the clinician's diagnostic capabilities. The proven technologies for diagnosing TMD patients at this point appear to be: 1) hard tissue radiographs and soft tissue imaging techniques that show, within their limitations, the integrity and anatomical relationships of structures in the TMJ; and 2) arthroscopy for direct examination of the joint.

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