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TRIGEMINAL NEURALGIA: IMPACTS ON QUALITY OF LIFE

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Abstract: Introduction: Trigeminal neuralgia (TN) is a neurological condition characterized by episodes of intense, paroxysmal pain in the face, related to irritation or compression of the trigeminal nerve, which is responsible for facial sensation. This pain is usually triggered by stimuli such as touching, chewing or exposure to wind. The trigeminal nerve has three branches: the ophthalmic (V1), the maxillary (V2) and the mandibular (V3), and the disease can affect any of these branches. Objective: To describe trigeminal neuralgia, with an emphasis on its impact on patients' quality of life and to discuss possible treatments. Methodology: This is a Literature Review including studies from the PubMed, UpToDate and Scielo platforms from the last 5 years. The descriptors used were "trigeminal neuralgia", "quality of life" and "treatment". Results and Discussion: Chronic pain associated with TN has a significant psychological impact on patients' lives, such as the development of anxiety and depression due to constant suffering. In addition, the unpredictability of crises can limit daily activities such as eating, personal hygiene and communication, leading to social isolation and reduced productivity at work. Sleep quality can also be compromised due to episodes of nocturnal pain, resulting in fatigue and decreased general well-being. Carbamazepine (CBZ) remains the treatment of choice for trigeminal neuralgia, showing good results in many patients by reducing the intensity of the pain. In cases where medication is not effective or has side effects, surgical options such as microvascular decompression may be indicated. Conclusion: TN, which has an annual incidence of 4.3 per 100,000 in the general population, affects patients' mental health due to its intense pain episodes. From this perspective, we conclude that the management of trigeminal neuralgia should consider not only pain control, but also strategies to minimize the emotional and social impacts of the condition. Multidisciplinary approaches, including psychological support and complementary therapies, are essential to ensure better adaptation to the disease and promote a better quality of life for patients.

Keywords: Trigeminal Neuralgia, Quality of Life, Anatomy and Treatment

INTRODUCTION

Trigeminal neuralgia (TN) is a condition caused, in particular, by compression of one of the three branches (V1, V2 and V3) of the fifth (V) cranial nerve - the trigeminal nerve. The mandibular branch (V3) is the most affected by the syndrome, followed by the maxillary branch (V2). TN is usually manifested by intense but brief, unilateral pain - similar to an electric shock in the affected area - triggered by previously innocuous triggers. There are different conditions to explain the origin of TN, both primary and secondary: compression of the nerve root, lesions in the brainstem or no previously recognized structural cause, which can be explained by genetic predisposition and/or central sensitization.

In this article, trigeminal syndrome will be addressed through its anatomical and physiological aspects, establishing a correlation between symptoms and the quality of life of those who suffer from it. The condition can affect patients in their daily activities, including their mental health. It is a complex syndrome in its diagnosis and management, which deserves special attention from the main health agencies. In order to optimize the monitoring and treatment of people affected by NT, it is essential that it is assessed and understood in a broad way, not only from a biological perspective, but also by looking at the psychosocial aspects linked to this condition and its possible consequences in the lives of patients.

OBJECTIVES

GENERAL OBJECTIVE

• To understand the impacts on the quality of life of individuals with trigeminal neuralgia, analyzing how this condition affects the physical health, emotional and social well-being of patients. This article seeks to elucidate the limitations imposed by pain and its repercussions on daily activities.

SPECIFIC OBJECTIVES

- Describe in detail the anatomy of trigeminal neuralgia and point out the main symptoms;
- To determine the prevalence of trigeminal neuralgia in different age groups and genders, considering gender as the main variable;
- To identify and classify the main symptoms presented by patients with neuralgia, including the intensity, frequency and factors that contribute to triggering the pain;
- To point out the main forms of treatment currently available for trigeminal neuralgia, including pharmacological options and surgical intervention.

METHODOLOGY

This is a qualitative study carried out through a literature review using the PubMed, Up-ToDate and SciELO databases. Sixteen articles published in the last five years were selected, including studies in English, Spanish and Portuguese. The selection of articles prioritized criteria such as relevance, originality and direct relation to the theme of trigeminal neuralgia, exploring anatomical, diagnostic and therapeutic aspects. The search was conducted using specific descriptors, such as "trigeminal neuralgia", "quality of life" and "treatment", in order to locate up-to-date publications with high scientific rigor. In addition to the databases, an anatomical image was extracted from the book "Anatomy Oriented to the Clinic", by Keith L. Moore, duly referenced, to enrich the analysis and provide a visual anatomical basis for the study.

The review aimed to compile information that would favor a better understanding of the condition, highlighting its effects and the advances in recent literature. The entire selection and analysis method prioritized the consistency and relevance of the information, ensuring the quality and accuracy of the data presented, in line with the research's methodological proposal.

RESULTS AND DISCUSSION

TRIGEMINAL ANATOMY

The trigeminal nerve, made up of its three branches V1, V2 and V3, as shown in *Figure 1*, is the anatomical basis of primary headache and facial pain syndromes (11). The three branches originate in the semilunar ganglion and exit the skull through the superior orbital fissure, foramen rotundum and foramen ovale, respectively (16).

The V2 and V3 branches are the most common to be involved in trigeminal neuralgia, with an incidence of 28 to 33% of cases (16). On the other hand, headache syndromes are mainly associated with V1 (11).

The functional representations of the three branches in the brainstem, especially below the pons, remain unknown in humans, even though the anatomical location of the trigeminal spinal nucleus is known (11).

The trigeminocervical complex, known as the nuclei of the trigeminal nerve and the greater occipital nerve, has not yet had its precise anatomical correlation demonstrated, although there is strong evidence of its existence (11), such as the efficacy demonstrated in the



Figure 1. Distribution of the trigeminal nerve branches. Source: Moore L. K, et al. Clinical Oriented Anatomy 8th Ed Philadelphia: Wolters Kluwer, [2018]

pharmacological blockade of the greater occipital nerve in many cases of facial pain syndrome and headaches. The exact location and mechanisms of interaction remain unknown, even though it is known that the interaction is likely to be in the central nervous system (7).

TRIGEMINAL NEURALGIA

Trigeminal neuralgia (TN) is a condition characterized by recurrent episodes of intense, brief, unilateral pain similar to electric shocks that are abrupt in onset and termination. The distribution of pain can occur in one or more divisions of the fifth cranial nerve and is typically triggered by innocuous stimuli (6).

The most commonly affected division is the mandibular, followed by the maxillary, and the least affected is the ophthalmic. However, when the pain is located close to the boundary of another division of the nerve, it may appear to expand into the neighboring division. Although often spontaneous, TN often has triggers, such as touching a sensitive spot, brushing teeth, talking, chewing, eating or drinking hot or cold food or liquids. These stimuli are not harmful in normal individuals. Most NT sufferers have spontaneous and stimulus-evoked pain episodes. The pain episodes can last seconds, with a persistent post--shock pain that lasts one or two minutes, but does not have the sharp, penetrating pain of the initial shock. In milder cases, attacks can occur only a few times a day, while in more severe cases, recurrent episodes can last for several minutes or even hours (4).

Several different conditions can give rise to TN, whether primary or secondary. Compression of the trigeminal nerve root is the main cause, usually occurring a few millimeters from the entrance to the pons. The transition from central oligodendroglial myelination to peripheral Schwann cell myelination in this root entry zone may be responsible for susceptibility to compression. However, brainstem lesions are also responsible for a small proportion of cases, such as tumors or demyelinating plaques, which can also contribute to TN when the trigeminal pontine pathways are involved. Other cases of TN may be idiopathic with no structural cause found, in which central sensitization and/or genetic susceptibility may contribute to these cases (6).

IMPACTS ON QUALITY OF LIFE

Trigeminal Neuralgia (TN) has a significant impact on patients' quality of life due to episodes of chronic and intense pain. Individuals with TN experience abrupt orofacial pain, which can be triggered by simple activities such as talking or chewing. Such events can occur several times during the day, impairing performance in daily activities and contributing to the emergence of psychological problems such as anxiety and depression (2).

NT is a facial pain that needs to be seen from a global perspective, encompassing not only biological characteristics, but also its social and psychological influences (12). Recognizing the psychological components of pain is fundamental to conducting effective treatment.

The explanation is often neglected by health professionals because they think it is too complex to present to the patient, especially as it refers to more specific knowledge. Paradoxically, studies report that patients who understand their illness have increased their ability to cope with it (14).

A study of 10 healthy patients and 10 patients affected by TN revealed an average pain intensity of 73.30 on the Visual Analog Scale (VAS), as well as an average duration of pain of 102.70 months. Chronic pain was reflected in a mean score of 16.90 on the Oral Health Impact Profile (OHIP) questionnaire, which assesses the impact of pain on oral health and general well-being. In comparison, the healthy control group had a score of only 1.10, highlighting the dissonance in quality of life between the two groups (5).

Although no significant association was found between pain duration and quality of life (p=0.17), the data indicate that trigeminal neuralgia profoundly affects patients' oral health and daily functions. These results reinforce the need for continuous medical follow-up and effective therapeutic strategies to minimize the negative effects of this condition (5). In this sense, the impacts of TN are not limited to physical pain, but also affect patients' emotional health and social relationships. Constant suffering can lead to social isolation and make it difficult to maintain professional activities, as well as increasing the risk of psychological disorders. This scenario reinforces the importance of therapies that consider patients' individual needs (2).

Considering that many patients find it diflicult to follow traditional treatments due to side effects such as sedation and dizziness, it is becoming increasingly essential to explore therapeutic alternatives that can improve the quality of life of these individuals (2).

Thus, the use of new therapeutic alternatives, such as botulinum toxin and lidocaine, are already emerging as management possibilities to provide pain relief without side effects, offering hope for improving the wellbeing and quality of life of patients dealing with this condition (2).

PREVALENCE

The general prevalence of NT is 5.9 per 100,000 in women and 3.4 per 100,000 in men (15). The annual incidence of NT is 4.3 per 100,000 in the general population, with a slight predominance in females (3:2). This rate increases with age, with rates of 17.5 cases/100,000 people per year in the population aged between 60 and 69 and 25.6 cases among those aged 70 and over. The average age of onset is 53.9 years, and it is uncommon for it to occur before the age of 40 (8).

The incidence tends to be slightly higher in women, with the right side of the face more affected than the left, which is mainly due to the smaller size of the foramen ovale and round (15), but this suggestion is still under study.

In approximately 60% of cases, only one branch is involved, the maxillary or mandibular branch, while in approximately 35% of cases, both are involved. On the other hand, the ophthalmic branch is rarely affected (i.e. in less than 4% of patients) (3).

No ethnic or geographical differences in incidence have been identified, although some diseases, such as multiple sclerosis (MS), seem to increase the risk of TN. Patients with MS are 20 times more likely to develop TN (8).

CLASSIFICATION OF NEURALGIA

Neuralgia can be idiopathic (ITN), classical (CTN) or secondary (STN), the clinical and evolution of which are different from the former. TN is characterized by similar pain in both the classic and secondary forms, the latter being attributed to trauma, intracranial tumours, demyelinating diseases, conditions such as osteitis, osteomyelitis, primary and secondary tumours of the jaws, dental causes and post-herpes zoster (10). Generally, the former is linked to neurovascular compression (9), as already mentioned, while the latter is linked to other problems, such as multiple sclerosis (13).

In the idiopathic form, no neurophysiological or MRI changes are detected, while an underlying cause can be identified in NTS (8). For this reason, there is an importance indicated by studies for the use of magnetic resonance imaging in order to rule out possible adjacent problems, such as tumors or multiple sclerosis itself (9).

CTN refers to cases probably caused by compression of the nerve root by a tortuous blood vessel. TNS presents with "recurrent paroxysms of unilateral facial pain that meet the diagnostic criteria for TN, either purely paroxysmal or associated with concomitant continuous or near-continuous pain," in patients with a documented underlying disease recognized as the cause of the neuralgia, which would explain the pain. Approximately 15% of cases of TN are secondary (10).

DISEASE TREATMENT

There are two types of therapy for NT: drug therapy and surgical therapy (8). Initially, pharmacological therapy is monotherapy; however, combined therapy with different drugs can be used when the efficacy of monotherapy is low (3).

Antiepileptic drugs have been used to treat pain since the 1960s, especially neuropathic pain, especially when it comes to stabbing or burning pain. Carbamazepine (CBZ) is one of the most widely used antiepileptic drugs. It is also used in the treatment of other diseases, such as neuropathic pain and bipolar disorder (12).

The onset of CBZ's effects on pain paroxysms is usually very rapid and seems to be mainly related to the blocking of sodium channels in neuronal membranes during high-frequency stimulation, thus reducing the propagation of the electrical signal and limiting the spread of ectopic activity. However, long-term treatment with CBZ has been associated with many side effects, including drowsiness, tiredness, dizziness, nausea, vomiting, ataxia, kidney and liver toxicity, allergic reactions and recurrence of symptoms in up to 50% of patients (1).

Lamotrigine (LTG) is a more modern antiepileptic, like CBZ, it is also used for bipolar disorder and mood disorders, it is an alternative second-line drug for the treatment of NT, often used in patients who have not tolerated the use of CBZ (14).

However, when necessary (i.e. due to failure or toxicity), other drugs can be combined with any of them or used instead. Alternatives include other anticonvulsants (e.g. pregabalin, gabapentin and phenytoin), baclofen and botulinum toxin type A (BTX-A) (3). Patients who do not respond to pharmacological treatment or who have severe side effects are candidates for more invasive strategies, such as nerve blocks or surgery (1).

CONCLUSION

Based on the above, it can be concluded that TN, which has an annual incidence of 4.3 per 100,000 in the general population and a slight predominance in females, is characterized by recurrent episodes of intense, brief and unilateral pain similar to electric shocks caused by stimuli in the orofacial region, with the mandibular branch being the most affected, followed by the maxilla. The main cause of the disease is compression of the trigeminal nerve root, which usually occurs a few millimeters from the entrance to the bridge. In other cases, lesions in the brainstem, central sensitization, genetic susceptibility and idiopathic causes can also trigger TN.

In view of the aspects presented, it was observed that TN has a significant impact on patients' quality of life, since episodes of chronic and intense pain are capable of affecting the mental health of sufferers of the disease, triggering anxiety and/or depression due to the difficulties encountered when dealing with persistent pain. In addition to the psychological impacts, it is worth reiterating that TN can also affect the patient's daily life and the maintenance of their professional activities when they experience long episodes of intense pain.

Parallel to this perspective, one study emphasized that patients who had an understanding of their disease increased their ability to cope with it. This bias highlights the importance of health teams explaining the anatomical and physiological aspects of NT to those with the disease, in order to obtain better treatment results.

Finally, in terms of treatment, studies show that drug therapy is carried out through the use of antiepileptic drugs, with CBZ being the most widely used, followed by LTG. When monotherapy isn't enough, some of the other anticonvulsants are added. Other therapeutic alternatives to provide pain relief without side effects are botulinum toxin and lidocaine. In patients who don't have good results with pharmacological treatment, more invasive techniques are used, such as nerve blocks or surgery.

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