

Trigeminal neuralgia: literature review

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Abstract

The trigeminal nerve, fifth equal of cranial nerves, a mixed nerve is considered by possessing motor and sensitive components. The sensitive portion takes to the Nervous System Central somesthetics information from the skin and mucous membrane of great area of the face, being responsible also for a neural disease, known as the Trigeminal Neuralgia. The aim of this study was to review the literature on the main characteristics of Trigeminal Neuralgia, the relevant aspects for the diagnosis and treatment options for this pathology. This neuralgia is characterized by hard pains and sudden, similar to electric discharges, with duration between a few seconds to two minutes, in the trigeminal nerve sensorial distribution. The pain is unchained by light touches in specific points in the skin of the face or for movements of the facial muscles, it can be caused by traumatic sequels or physiologic processes degenerative associate the vascular compression. Prevails in the senior population, frequently in the woman. In a unilateral way it attacks more the maxillary and mandibular divisions, rarely happens in a simultaneous way in the three branches of trigeminal nerve three branches.

Keywords: trigeminal neuralgia, anatomy, pain.

1 Introduction

Trigeminal neuralgia (TN) is a pathology characterized by paroxysmal pain so excruciating and sudden, like a shock, which lasts a few seconds to two minutes. It is most common between the pains orofacial, returns in irregular periods, being triggered by stimuli not painful, as the simple act of speaking, or chewing (TODA, 2007).

Neuralgia occurs more frequently in the second and third divisions of the trigeminal nerve, so deep and limited to the territory of trigeminal distribution. The attacks may be accompanied by salivation, lacrimation, rhinorrhea, nasal mucosa congestion, skin redness, facial swelling, or clonic contraction ipsilateral hemifacial spasm and/or contraction of the muscles acting on the jaw. It is also characterized by regions of increased arousal, called trigger zones. In general, it affects more women between the sixth and eighth decade of life and may persist days, weeks or even months (GOTO, ISHIZAKI, YOSHIKAWA et al., 1999).

Türp and Gobetti (1996) reported that the most likely mechanism for the development of neuralgia is the association of degenerative processes of aging and vascular compression acting for years about the root of the trigeminal nerve. It is believed that the myelin sheath around the nerves disappear over time. TN may also be related, late, incorrect therapy in maxillofacial trauma (FRIZZO, HASSE and VERONESE, 2004).

Due to the difficulty in diagnosing the mechanisms triggering TN, it is important to conduct the interview, considering the symptoms reported by patients, and additional exams, such as imaging exams, studies for clinical evaluation of the anatomy of the trigeminal nerve and adjacent structures (LUNA, GRAÇA, SILVA et al., 2010).

The treatment of TN can be medical or surgical (QUESADA, BAPTISTA, DAIANA et al., 2005). According Mattos, Bueno and Mattos (2005), the medical therapy is the first choice, resorting to the functional neurosurgery only in cases where clinical therapy proves ineffective.

Drug treatment is the use of anticonvulsants, centrally acting muscle relaxants, neuroleptics and local anesthetics. Surgical treatment aims to interruption of peripheral trigeminal pathways or the elimination of possible causes (BROW, 2009).

Thus, the aim of this study was to review the literature on the main characteristics of TN, the relevant aspects for the diagnosis and treatment options for this pathology.

2 Material and methods

The literature review was searched on databases: PubMed, Scielo, Scopus and Lilacs, using the keywords: trigeminal neuralgia, anatomy, pain. The manuscripts and books evaluated were published from 1963 to 2010.

3 Literature review

3.1 Main characteristics of trigeminal neuralgia and epidemiology

TN is a pathology characterized by paroxysmal pain so excruciating and sudden, like a shock, which lasts a few seconds to two minutes. It is most common between the pains orofacial, returns in irregular periods, being triggered by stimuli not painful, as the simple act of speaking, or chewing (TODA, 2007).

Galassi, Blasi, Galassi et al. (1985) reported that the pain crisis may occur when the individual handling given areas of the face, called trigger zones. These areas are located ipsilateral to pain, usually around the nose and near the lips. The attacks often stimulate other responses such as salivation, facial flushing, lacrimation, or rhinorrhea. During a refractory period of pain, even in the presence of stimuli on the trigger zones, the painful process is not triggered.

Meneses, Clemente and Russ (1995) evaluated the medical records of 50 patients operated by decompression neurovascular of trigeminal nerve, and found patients with a mean age of 60 years and 03 months, with higher incidence in females (60%). The maxillary and mandibular divisions of the trigeminal nerve are most affected.

According Hotta, Bataglion, Bataglion et al. (1997), during an episode of pain, facial muscles related to speech and chewing, initiate a state of contraction intense. Loh, Ling, Shanmuhasuntharam et al. (1998) investigated the clinical characteristics of TN in 44 Asian patients, found a female predominance, and the right side most affected, the largest frequency was observed in the age group between 60 to 70 years and greater involvement of nerve was the mandible. In addition, found that the inability to carry out daily activities prejudice the quality of life of patients, leading to depression and even suicide.

TN can be unilateral or bilateral, when unilateral, the right side is most affected, and its incidence five times greater than the left side (GOTO, ISHIZAKI, YOSHIKAWA et al., 1999).

Gusmão, Magaldi and Arantes (2003) evaluated 135 patients with TN, 57 (42.2%) were male and 78 (57.8%) were female. The age ranged from 48 to 82 years, mean 61 years. The pain predominated on the right (57%) and was located in the territory of innervation nerve ophthalmic in 2 patients (1.5%), maxillary nerve in 35 (25.9%), and mandibular nerve in 43 (31.9%).

Clinically, the TN can be confused with disorders related to teeth, facial bones and paranasal sinuses, leading to a variety of therapeutic incorrect. Disease severity is underscored by having one of the highest suicide rates in relation to any disease, and is considered one of the most painful diseases known (NEVILLE, DAMM, ALLEN et al., 2004).

The TN is a painful disease, unilateral face. The pain is often triggered by trivial stimuli, such as washing, shaving, smoking, talking, eating, applying makeup and brushing your teeth, but may occur spontaneously. The pain is of sudden onset and termination, and may decrease in intensity for varying periods (ESKANDAR, BARKER and RABINOV, 2006).

In a survey conducted in the U.S.A., it was found that the TN is more common in women than in men, identified 05 new cases of Idiopathic Trigeminal Neuralgia every year 100,000 women and 2.7 new cases per each year 100,000 men (YOSHIMASU, KURLAND and ELVELVACK, 1972). Katusic, Beard, Bergstrahl (1990) reported that there were on average 4.3 new cases per year of Idiopathic Trigeminal Neuralgia every 100,000 inhabitants in Rochester (Minnesota - USA), between the years 1945 and 1984. Jacob and Rhoton (1996) reported that the incidence of TN is 4.3 cases per 100, 000 people and occurs predominantly in women.

Siqueira, Nóbrega, Valle et al. (2004) evaluated the dental conditions of 50 patients with TN and found that 72% had intraoral trigger zone, 16% had temporomandibular disorders and 6% burning mouth syndrome; found a patient with temporomandibular disorder had trismus secondary to compression of the trigeminal ganglion with balloon and 42% of patients showed limitations for the performance of daily activities.

3.2 Etiology of trigeminal neuralgia

The distortion of the “zone of trigeminal root entry” by arteries or veins appear to be related to the occurrence of Idiopathic Trigeminal Neuralgia because the neurovascular decompression of the region often results in improvement of pain (JANNETA, 1963).

According to Adams and Victor (1985) the TN can be classified as primary or idiopathic and secondary or symptomatic. The primary would have no specific cause, but it turned out that the main cause is a neurovascular conflict, caused by small branches of arteries.

It is believed that the vascular compression occurs near the trigeminal nerve, generates areas of demyelization, which would cause functional changes in axonal levels, creating “short circuit”. These would work by changing the original impulses so that a tactile stimulus would not reach the cord and bulb-core would be interpreted as pain and not touch, causing trigeminal neuralgia (BROW, 2009).

According to Linskey, Jho and Jannetta (1994), some anatomical changes related to aging of the physiological human body, such as the appearance of atherosclerosis, has its increased predisposition to age along with the factor of hypertension, sometimes causing the contact neurovascular in human trigeminal nerve roots.

Thus, the mechanism responsible for triggering neuralgia would be the conjunction of the degenerative processes of aging associated with vascular compression, acting for several years on the posterior root of the trigeminal nerve (TÜRPE and GOBETTI, 1996).

Luna, Graça, Silva et al. (2010) reported that TN may be idiopathic or triggered by trauma, emotional factors, neoplasms and external stimuli.

According to Siqueira, Nóbrega, Valle et al. (2004), TN may present as a cause brain lesions, arachnoid adhesions, vascular loops or plaques of demyelination located at the entrance of the nerve root, which will generate potential ectopic. Other phenomena that are related to the TN are: autosomal dominant, autosomal recessive, possibly X-linked, as well as phenomena related to senility.

The TN is a disease with pathophysiological mechanisms not fully understood, being related to compression of the trigeminal nerve by intracranial peripheral vessels, classified as Idiopathic Trigeminal Neuralgia (SIQUEIRA, NÓBREGA, VALLE et al., 2004).

Neville, Damm, Allen et al. (2004) affirmed that the TN may be idiopathic, but is generally associated with a condition at some point during the course of the nerve, such as an intracranial tumor, being referred to as trigeminal neuralgia symptomatic.

The TN source has symptomatic causes such as viral infections, tumors, multiple sclerosis, aneurysms and impaired alveolar post-dental extractions. The TN symptom may also be related to therapy later incorrect in maxillofacial

trauma, such as facial bone fractures, especially fractures of the zygomatic-maxillary complex, especially when it involves the floor of the orbit, injuring the infra-orbital nerve (FRIZZO, HASSE and VERONESE, 2004).

Bennetto, Patel and Fuller (2007) found that 05-10% of patients with TN have a cause: tumor, multiple sclerosis, abnormalities of the cranial base, or arteriovenous malformation.

Approximately 80-90% of cases classified as idiopathic TN are caused by compression of the trigeminal nerve, immediately on its exit from the brainstem by a handle aberrant arterial or venous, especially the superior cerebellar artery (KRAFFT, 2008).

3.3 Diagnosis

In the TN, the patient has some clinical aspects that are characteristic and help in correct diagnosis, for example, often prostrate, and immobilization of his face with his hands between attacks to prevent any mobility in hopes of preventing further episodes (BAYER and STENGE, 1979).

The diagnosis of TN is important to perform the analgesic block (at least at the beginning of neuralgia), rapidly stopping the pain and triggering due to the occurrence of these in the area of the affected nerve. The neurogenic pain in the orofacial region may be mistaken for masticatory pain. Therefore, we must consider the characteristics of each one (FARDY and PATTON, 1994).

Neurogenic pain is located, stimulating, disproportionate to the stimulus, presents characteristics plus spatial and temporal, not accompanied by impaired chewing and held by anesthesia. While chewing the pain is poorly localized "depression" more proportional to the stimulus, does not have characteristics of addition, it relates to the symptoms of masticatory dysfunction, is held only by analgesic block the source (the joint itself or the mastication muscles) (FARDY and PATTON, 1994).

The diagnosis of TN is mainly based on clinical signs and symptoms mentioned by the patient, as typical paroxysms, refractory periods and zones trigger (TÜRPE and GOBETTI, 1996).

Olesen (1997) established the following criteria for diagnosis of TN: paroxysmal attacks, facial pain or front, that lasts a few seconds to less than two minutes, the pain presents distribution over one or more branches of the trigeminal nerve, pain is sudden, intense, sharp, superficial, stabbing or burning character, of great intensity. The pain is precipitated from trigger areas or by certain daily activities such as eating, talking, washing your face or brushing your teeth. In addition, between paroxysms, the patient is completely asymptomatic. It is also reported that the patient has no neurological deficit, crisis are invariable for each patient, when necessary, should be excluded other causes of facial pain by history, physical examination and special investigations.

In the diagnosis should be excluded from the primary headaches of short duration (MONZILLO, SANVITO, COSTA et al., 2000). Tronnier, Rasche and Hamer (2001) reported that atypical symptoms and signs warn of other diseases, such as abnormal neurological examination, abnormal oral examination; younger than 40 years, bilateral symptoms, dizziness or vertigo, hearing loss or disorder, numbness, duration pain greater than two minutes, pain outside the distribution of the trigeminal nerve, and visual changes.

According Neville, Damm, Allen et al. (2004), a distinguishing characteristic of the TN is that signs of sensory loss can not be demonstrated in a physical examination. It is also necessary that the pattern of pain falls on some criteria, such as the "attack" of pain is abrupt, often initiated by a gentle touch on a specific trigger point and constant, the pain is extreme, and paroxysmal excruciating, the length of a single pain "spasmodic" is less than 2 minutes, although the full attack can consist of numerous short spasms; during the refractory period, additional attacks usually can not be provoked by touch of the trigger point; occur spontaneous remissions lasting for more than six months, especially during the initial phase of the disease.

Krafft (2008) affirmed that the diagnosis is essentially clinical, but may need some evaluations of image or specialized tests in patients who have unusual characteristics such as bilateral symptoms, dizziness or vertigo, age less than 40 years, hearing loss or disorder, episodes of pain with longer than two minutes, visual disturbances and pain that is not in the trigeminal distribution.

3.4 Treatment

The TN treatment may be medical or surgical (QUESADA, BAPTISTA, DAIANA et al., 2005). According Mattos, Bueno and Mattos (2005), the medical therapy is the first choice, resorting to the functional neurosurgery only in cases where clinical therapy proves ineffective.

At first, carbamazepine and oxycarbazepina drugs must be administered at lower doses, but if necessary, doses may be increased gradually progressively (QUESADA, BAPTISTA, DAIANA et al., 2005).

The choice of method depends on the surgical condition where the patient is the etiology of facial pain, and the skill of the neurosurgeon. Among the surgical procedures most widely used are neurovascular decompression as the primary ablative technique, the radiofrequency rhizotomy, balloon compression and glycerol rhizotomy and radiosurgery (SIQUEIRA, NÓBREGA, VALLE et al., 2004).

According to Patterson (1999), only 25% of patients present with TN total pain control only with the use of drugs over time. The drugs most commonly used are: local anesthetics, neuroleptics, muscle relaxants, and anticonvulsants.

Surgical treatment is based on the assumption that the cause is of peripheral origin, such as trigeminal nerve damage in a blood vessel, by a tumor or an inflammatory lesion (MAESTRI and HOLZER, 1993).

In recent years, the two most common procedures used were: a differential percutaneous electrocoagulation of the trigeminal nerve and trigeminal vascular decompression and also with radiofrequency thermocoagulation of the Gasser ganglion an effective method, widely used in patients over 50 years (FRIZZO, HASSE and VERONESE, 2004).

According to Tronnier, Rasche and Hamer (2001), the decompression technique that is considered to promote relief for a longer time, with control of pain by more than 70% of patients over 10 years. The decompression is indicated in young individuals who want to preserve the facial sensitivity, when there is suspected lesion towards trigeminal neuralgia or when this is combined with other facial neuralgia or hemifacial spasm.

In decompression, is performed to remove the irregularities bone, skull base, which are close to the trigeminal nerve and/or microvascular decompression with the removal of blood vessels, which are located on the trigeminal nerve, leading them to a position closer to the normal. Thus the blood vessels are deprived of nerve fibers of the ganglion (NEVILLE, DAMM, ALLEN et al., 2004).

The glycerol rhizotomy is performed by injecting a caustic material around nerves that leave or enter the ganglion of the trigeminal nerve. In percutaneous radiofrequency rhizotomy, there is selective destruction of sensory nerve fibers by crushing or by applying heat (GUSMÃO, MAGALDI and ARANTES, 2003).

In rhizotomy occurs traumatization or destruction of nerve fibers selected close to or within the trigeminal ganglion. With the patient is sedated puts a needle driven by X-ray fluoroscopy, inside the ovale foramen. Being driven by the sensitivity of the patient, are located on the nerve fibers painful condition involved, and the fibers are selected termocoagulação destroyed by a radiofrequency or by deposition of a toxic substance such as glycerol (by glycerol rhizotomy) (GUSMÃO, MAGALDI and ARANTES, 2003). Is an initial improvement in the patients to 97.5% (PETERS and NURMIKKO, 2002). This technique damages irreversibly thin myelinated and unmyelinated fibers heated at temperatures ranging from 55 °C to 70 °C (YOON et al., 1999).

The compression balloon is simpler than radiofrequency rhizotomy can be performed under local anesthesia and the patient is awake, but without their participation. The type of anesthesia varies with the experience of professionals involved in the surgery. May be general, with or without intubation, or by blocking the region to be treated, lidocaine or other drug equivalent (PETERS and NURMIKKO, 2002).

Balloon compression is a technique that offers comfort for a longer time and with lower recurrence rates (approximately 30%), and have lower morbidity and no mortality. The procedure lasts a few minutes and does not require cutting. Carried out a small hole with a diameter equivalent to the tip of a ballpoint pen, is inserted a catheter into the patient face, inside the cheek. A small balloon is inflated at the end of the catheter, squeezing the trigeminal ganglion and causing the pain is eliminated in 98% of cases (PETERS and NURMIKKO, 2002; NURMIKKO and ELDRIDGE, 2001).

4 Conclusion

In conclusion, TN is more common among facial neuralgias, and since, in most cases, the patient seeks the dentist because of the location of pain, it is important that these professionals have knowledge of anatomy and function of the trigeminal nerve, keeping up with studies conducted in this area in order to correctly diagnose and perform control pain through medication or indicate surgical treatment.

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