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Diagnosis and Treatment of Patients with Trigeminal Neuralgia: A Review

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Abstract: Trigeminal neuralgia (TN) is one of the most common forms of neuropathic pain found in the head and neck region, the diagnosis of which is clinical. Long-term oral medication offers no benefits for patients with TN. Interventions for these patients include percutaneous rhizotomy, radiofrequency thermocoagulation, balloon compression, botulinum neurotoxin, stereotactic Gamma knife radiosurgery and microvascular decompression. The aim of the present study was to investigate aspects of the diagnosis and treatment of patients with trigeminal neuralgia through a literature review. A bibliographic survey was conducted through searches of the SciELO, BIREME, Medline and Google Scholar electronic databases for relevant articles published in the English language. The articles found in the literature were divided into four groups: Group 1 – definition and epidemiology (references 1 to 9); Group 2 – diagnosis (references 10 to 12), Group 3: treatment (references 13 to 26); Group 4: interventional approaches (references 27 to 40). TN is one of the most common forms of neuropathic pain found in the head and neck region. The diagnosis is based on a history of pain such as an "electrical shock" felt in a division of the trigeminal nerve. Approximately 50% of patients with TN do not achieve long-term benefits from drug therapy. Interventional approaches require more scientific research. Future studies should focus on genetics, unexplored etiological factors, sensory function, neurosurgical outcomes and complications, combinations and neuromodulation as well as the development of new drugs with greater tolerability.

Keywords: Neuropathic Pain, Chronic Facial Pain, Untreatable Pain, Trigeminal Nerve, Trigeminal Neuralgia, Diagnosis, Treatment

Introduction

According to the International Association for the Study of Pain (IASP), pain is defined as an unpleasant sensory and emotional experience associated to real or potential injuries and described in terms of such injuries.[1] Neuropathic pain is caused by an injury or dysfunction in the central or peripheral somatosensory nervous system and affects approximately 8% of the population.[1,2]

The trigeminal nerve is the fifth pair of cranial nerves and is responsible for the general sensitivity of the head and face. Trigeminal neuralgia (TN) is the most common form of severe facial pain[3] and one of the most common forms of neuropathic pain found in the head and neck region. TN manifests as attacks of "electrical shock", "burning" or tingling at undefined intervals, generally triggered by non-nociceptive stimuli, such as chewing, combing the hair, brushing the teeth, shaving or even gentle touches in the involved region, which is a phenomenon known as allodynia.[4,5]

Most cases of TN are caused by neurovascular compression, but a significant quantity is secondary to inflammation, tumor or trauma.[3] TN is often misdiagnosed or under-diagnosed[6] and can be confounded with pain of an odontogenic origin. The incidence ranges from 4.3 to 27 new cases per 100,000 people per year.[7-9] The occurrence of TN is greater in women and increases with age.[7]

Diagnosis

The diagnosis of TN is clinical and based on patient history, as there are no laboratory exams or definitive diagnostic exams. The diagnosis may also be complemented by infrared thermography.[5]

The classic diagnosis is based on a history of pain such as an "electrical shock" felt in one of the branches of the trigeminal nerve that lasts for a variable period of time (normally short). There is often a stimulus, but not necessarily only one. Attacks are typically infrequent in the beginning, but become more frequent over time and can increase to a frequency of up to hundreds of times per day.[3] Remissions occur, but relapses become more frequent with age. Some patients report atypical histories in which the pain crosses the divisions of the trigeminal nerve or longer paroxysms of pain than a lightning attack.

The neurological exam is normal in classic TN. The motor and sensory exam of the face, in particular, is normal in classic TN, but is useful to the identification of secondary dysfunction of the trigeminal nerve that may lead to a diagnosis of secondary TN or trigeminal neuropathy. The same is true for the blink reflex and other trigeminal reflex tests, as the presence or absence of an abnormal result does not affect the diagnosis of TN, but may indicate the need for exams for causes of secondary TN.[3]

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Each case of TN can be investigated using magnetic resonance of the head to evaluate the pontine region for neurovascular compression, which can be treated with microvascular decompression. A small but important percentage of patients have a different type of structural injury compressing the trigeminal nerve. No available data were found in the literature on the incidence of the first identification of multiple sclerosis (MS) through the diagnosis of TN, but TN-related MS occurs in patients with a diagnosis of MS that can be confirmed by magnetic resonance.[10,11]

The electrodiagnostic test can be useful in distinguishing primary and secondary TN. According to the European Academy of Neurology guidelines on TN, the trigeminal reflex text has high specificity and sensitivity for secondary TN, whereas the evoked response test has high sensitivity but low specificity and does not safely distinguish between primary and secondary TN.[12]

Treatment

Different drug and interventional therapies are currently used for TN. Therapeutic approaches include interventions such as percutaneous rhizotomy, radiofrequency thermocoagulation, balloon compression, botulinum neurotoxin, stereotactic Gamma knife radiosurgery and microvascular decompression.[13]

Therapeutic conducts for TN follow the guidelines of the American Academy of Neurology (AAN) and European Federation of Neurological Societies (EFNS), which recommend pharmacological treatment as the first choice. The aim of treatment for TN is to reduce the frequency and intensity of facial pain paroxysms or eliminate them completely. Most therapeutic studies use pain relief as the main outcome measures, although there is no consensus on measuring this outcome.[14] Pain intensity is not the same degree as pain relief.

A large part of studies on the treatment of TN used the visual analog scale (VAS) for pain or the Barrow Neurological Institute pain intensity scale. A small number of studies used attack frequency, daily functioning measures, the emotional impact of TN or satisfaction with treatment.[14]

Pharmacological treatment

The initial treatment of TN is normally pharmacological (even for secondary TN) using anticonvulsants. Carbamazepine is generally considered the drug of choice and is effective in 60-100% of cases (at least for some time), although the long-term failure rate can reach 50%.[12,15] Moreover, side effects are associated with this drug.

Oxcarbazepine has fewer side effects and is often used rather than carbamazepine, although there are few experimental data in opposition to clinical experience to support its use. The therapeutic target of these drugs in voltage-dependent sodium channels. There is evidence of low quality for the use of anticonvulsants such as lamotrigine and gabapentine.[12] New drugs, such as eslicarbazepine, which is an active metabolite of oxcarbazepine, and vixotrigine, which is a Nav1.7 channel blocker, are being evaluated as treatment.[14]

Botulinum neurotoxin

Botulinum neurotoxin (BoNT) is derived from the bacterium *Clostridium botulinum* and characterized by a group of homologous chain proteins with seven serotypes (A, B, C1, D, E, F e G).[16] It is widely used in esthetic treatments and in cases of disorders, such as dystonia and muscle spasms.[17,18]

Type A BoNT (BoNT/A) inhibits the release of acetylcholine (ACh) at the cholinergic nerve endings of the motor nerves, as it prevents ACh vesicles from binding to the membrane for the release of content and subsequent binding to receptors on the postsynaptic membrane.[16] This blockade leads to the desired esthetic and therapeutic effect, as it weakens the muscle for a period of three to four months.[16]

BoNT/A has a beneficial effect in the treatment of neuropathic pain,[19-22] with antinociceptive and anti-inflammatory activity. BoNT/A acts at peripheral and central sites. Peripherally, it blocks the coupling of intraneuronal vesicles in the inner membrane of the nerve ending, inhibiting the release of neuropeptides and neurotransmitters. Consequently, reductions occur in extracellular concentrations of acetylcholine, substance P, serotonin, calcitonin gene-related peptide (CGRP), glutamate and proinflammatory mediators.

Plasma levels of CGRP diminish in patients with TN who respond well to treatment with BoNT/A, whereas non-responders to this treatment do not exhibit a reduction in plasma CGRP levels.[19] Centrally, BoNT/A acts on the dorsal horn of the spinal cord as a result retrograde toxin transport. Microglial activation, which is an important component of nociception, is also attenuated.[20,23]

A randomized controlled clinical study with 40 patients (individuals with structural lesions were excluded) found a significant benefit.[24] BoNT/A was administered to the area of pain through subcutaneous and submucosal injections. The main side effect was temporary facial weakness.

Two systematic reviews with meta-analysis on the effectiveness and safety of BoNT/A for the treatment

of TN were published in 2016, citing four randomized clinical trials with a total of 178 patients (99 received BoNT/A and 79 received placebo).[25,26] There was no standardized dosage or injection method. Doses of BoNT/A ranged from 25 to 100 units. Injections were generally administered subcutaneously or intradermally in the region of clinically evident pain. Pain intensity and the frequency of attacks were significantly lower with BoNT/A compared to placebo, with a prolonged benefit for three months. Temporary facial asymmetry and edema were the two main side effects, both of which were well tolerated.

Percutaneous approach

Percutaneous therapeutic approaches are ablative procedures directed at the trigeminal ganglion (Gasser or semilunar) located in Meckel's cave.[27,28] The three common ablative techniques are chemical (glycerol rhizotomy), mechanical (balloon compression) and thermal (radiofrequency thermocoagulation).

The aim of treatment is to selectively destroy A delta and unmyelinated C fibers that mediate pain, while preserving A alpha and beta fibers that mediate touch.[29] These techniques generally have a high initial reduction rate of pain and attacks, but the benefit diminishes over time. The procedures can be repeated, if necessary.

Stereotactic Gamma knife radiosurgery

Stereotactic Gamma knife radiosurgery (GKRS) is a minimally invasive approach for the treatment of TN refractory to medication.[30,31] This type of treatment produces axonal degeneration, ion channel destruction and an electrophysiological block that reduces nociceptive input.[28] Highly precise localization of the trigeminal nerve or trigeminal ganglion is possible, which limits side effects.

GKRS results in a pain reduction of at least 50% in 75 to 95% of cases.[32] Systematic reviews and meta-analyses compared GKRS to microvascular decompression, reporting that GKRS directly corrects the cause of TN in at least 50% of cases.[33-36]

Microvascular decompression

Microvascular decompression is surgical treatment indicated when patients do not respond to clinical treatment or when there is vascular compression of the trigeminal nerve detected by diagnostic imaging exams, such as magnetic resonance.[37]

Among surgical procedures, microvascular decompression is considered the gold standard for the treatment of TN caused by vascular compression of the trigeminal nerve.[33,38] The procedure consists surgically moving and isolating the vessel (artery or vein) from the proximities of the nerve and, although more invasive than other methods, is considered safe.[39]

A meta-analysis of 46 studies (seven prospective and 39 retrospective) totaling 3897 patients revealed a long-term absence of pain in 76% of patients.[40] The greater probability of a successful outcome was associated with a period of five years of less, compression by the superior cerebellar artery, compression by an artery (including the anterior inferior cerebellar artery) rather than a vein and classic TN rather than atypical TN.

Table 1 presents a summary of therapeutic options used in patients with TN.

Table 1 - Summary of therapies commonly used for trigeminal neuralgia.

Modality	Evaluation	Comments
Pharmacological	Carbamazepine: moderate level of evidence for long-term benefit, but 50% failure rate in long term Oxcarbazepine, lamotrigine, gabapentin: commonly used but low quality or insufficient evidence	High degree of side effects with carbamazepine
Peripheral nerve intervention	Percutaneous rhizotomy (glycerol), radiofrequency thermocoagulation and balloon compression: high level of evidence for long-term benefit	Loss of benefit over time for the three techniques Low incidence of severe side effects, but painful anesthesia could be severe side effect No consensus on ideal temperature for radiofrequency thermocoagulation
Botulinum toxin	High quality of evidence for benefit	Low incidence of temporary side effects, but treatment must be repeated to maintain benefit
Radiosurgery	High quality of evidence in favor of long-term benefit. Benefit reduces by nearly half in 5 to 10 years, but treatment can be repeated	Onset of improvement is delayed 2 to 6 months after treatment Low incidence of side effects is increased with repeated treatment
Microvascular decompression	Level of evidence for long-term improvement is maintained for more than 5 years	Low incidence of side effects Endoscopic microvascular decompression had higher rate of benefit and lower rate of relapse with few side effects compared to traditional open microvascular decompression

Modified from Gerwin[3]

Objective
The aim of the present study was to investigate aspects of the diagnosis and treatment of patients with trigeminal neuralgia through a literature review.

Methods
A literature review was performed on aspects of the diagnosis and treatment of patients with trigeminal neuralgia. A bibliographic survey was conducted in the SciELO (*Scientific Electronic Library Online*), BIREME (Latin American and Caribbean Center on Health Sciences), LILACS (Latin American and Caribbean Literature on Health Sciences), Medline (Medical Literature Analysis and Retrieval System Online) and Google Scholar electronic databases for relevant articles published in the English language.

The following keywords were used: "trigeminal neuralgia", "facial pain", "chronic pain" "neuropathic pain", "diagnostic criteria" and "treatment". The keyword search "trigeminal neuralgia" individually resulted in 1746 articled published in the Medline database in the last five years.

The initial selection was based on the reading of titles and abstracts. From this pre-analysis, 40 full articles published in scientific periodicals were read and analyzed.

The inclusion criteria were full-text articles containing the keywords, studies involving humans (open studies and randomized, placebo-controlled, duly covered clinical trials) and reviews on the diagnosis and treatment of TN in English or Portuguese.

Results and Discussion
The articles found in the literature were divided into four groups: Group 1 – definition and epidemiology (references 1 to 9); Group 2 – diagnosis (references 10 to 12), Group 3: treatment (references 13 to 26); Group 4: interventional approaches (references 27 to 40).

In Group 1, references 3 to 5 present the definition of TN as the most common form of severe facial pain, whereas references 6 to 9 emphasize misdiagnosis, under-diagnosis, the higher incidence in women and the increase with aging.

The references in Group 2 highlight the fact that the diagnosis of TN is clinical and based on patient history, as there are no definitive laboratory exams or diagnostic exams. Each case can be investigated by magnetic resonance of the head to evaluate the pontine region for neurovascular compression. An electrodiagnostic test may be useful for distinguishing primary from secondary TN. The trigeminal reflex test has high specificity and sensitivity for secondary TN, whereas the evoked response test has high sensitivity but low specificity and does not securely distinguish between primary and secondary TN.

In Group 3, aspects of treatment for TN are investigated, including pharmacological treatment of type A botulinum neurotoxin (BoTN/A), for which references 12 (Bendtsen et al., 2019), 25 (Morra et al., 2016) and 26 (Shackleton et al., 2016) stand out. Reference 12 discusses the effectiveness of carbamazepine in 60 to 100% of cases, which is considered the drug of choice, although the long-term failure rate can reach 50%. References 25 and 26 present the results of two systematic reviews and meta-analyses on the effectiveness and safety of BoTN/A for the treatment of TN in a total of 178 patients. The authors found that pain intensity and the frequency of attacks were significantly lower with TNBo/A compared to placebo, with a prolonged benefit lasting three months.

Group 4 comprises studies on interventional approaches in the treatment of TN, for which references 27 (Missios et al., 2014), 32 (Constanzo et al., 2019) and 40 (Holste et al., 2020) stand out. Reference 27 analyzes minimally invasive percutaneous techniques, such as glycerol rhizotomy, balloon compression and radiofrequency thermocoagulation. According to reference 32, stereotactic Gamma knife radiosurgery for the treatment of patients with TN refractory to drug treatment results in a reduction in pain of at least 50% in 75 to 95% of cases. Reference 40 is a meta-analysis of 46 studies totaling 3897 patients. The authors found a long-term absence of pain in 76% of patients. The authors also reported an association between a greater probability of a successful outcome and a period of five years of less, compression by the superior cerebellar artery, compression by an artery (including the anterior inferior cerebellar artery) rather than a vein and classic TN rather than atypical TN.

Conclusions
The analysis of the literature on aspects of the diagnosis and treatment revealed that TN is one of the most common forms of neuropathic pain found in the head and neck region. The diagnosis is clinical and based on a history of pain such as an "electrical shock" felt in a division of the trigeminal nerve.

Approximately 50% of patients with TN do not achieve long-term benefits with the use of oral medication. Interventions such as percutaneous rhizotomy, radiofrequency thermocoagulation, balloon compression, botulinum neurotoxin, stereotactic Gamma knife radiosurgery and

microvascular decompression require more long-term scientific research.

Future studies should focus on genetics, unexplored etiological factors, sensory function, neurosurgical outcomes and complications, combinations and neuromodulation as well as the development of new drugs with greater tolerability.

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