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Review Article

Trigeminal Neuralgia Prevalence and Management

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Abstract

Background: Trigeminal neuralgia is a sudden, unilateral, brief, stabbing, recurrent pain in one or more branches of the fifth cranial nerve distribution that affects quality of life of the affected patient. Diagnosis is made using the history alone, based on the pain characteristics. Pain occurs in paroxysms, which can last between a few seconds and a few minutes. The frequency of the paroxysms varies from a few to hundred times per day.

Aim: The objective of this article is to provide an up-to-date review regarding the prevalence, etiology, diagnosis, and the management of trigeminal neuralgia.

Conclusion: TN is a rare disease but often associated with debilitating pain and disability. TN management is a concern for neurologists and neurosurgeons alike. Progress has been achieved in recent years, leading to the introduction of neuro-radiological approaches for both pathogenesis and surgical treatment. Medical treatment should be done in TN. With an growing variety of medications available, it is possible the surgical alternative may not be available for several years.

Keywords: *Trigeminal Neuralgia; Trigeminal Neuralgia Management; Diagnosis of Trigeminal Neuralgia; Prevalence of Trigeminal Neuralgia*

Introduction

Trigeminal neuralgia (TN) is the craniofacial region's most common neuropathic pain. It is one of the most severe orofacial conditions and can be characterized as a repeated and sudden stabbing, paroxysmal, electric shock-like, or burning pain [1]. TN's approximate annual incidence is 12.6/100,000 persons/year and increases the incidence with age [2]. TN affects 1 or more of the trigeminal nerve branches, mainly the second or third division. It may last a few seconds and can last for up to 2 minutes in some cases [3]. TN may be triggered by stimuli that are not normally irritating, such as facial caress, gentle breeze, chewing, talking, or brushing teeth, which may induce TN [4]. TN pain episodes can occur on a daily basis for days, weeks or months at a time and can occur quite a few times a day in serious cases [5]. Pain distribution is unilateral and follows the sensory distribution of cranial nerve V, usually radiating to the region of the maxillary (V2) or mandibular (V3), or occasionally to the region of the ophthalmic (V3), sometimes, both distributions are affected [6].

TN is classified as classical TN and symptomatic TN. Classical TN is caused by conflict with the neurovascular system and is classified into types 1 and 2. Type 1 is primarily paroxysmal where the patient has no pain between attacks where Type 2 is present between

attacks of constant continuous facial background pain. Type 2 is also known as atypical TN; central sensitization can be responsible for the chronic pain of this case. Type 2 neurovascular conflict may not be obvious and is considered to be resistant to different treatment methods [4]. Symptomatic TN is usually caused by disease leading to neural damage such as multiple sclerosis, herpes zoster, infection, physical injury or angle tumors [7]. Classical TN is characterized by very intense, sudden, serious, shock-like pain paroxysms that typically appear on one side of the face in the second and/or third trigeminal area [8].

Trigeminal neuralgia (TN) is often recognizable by the patient's history alone. Typically, physical examinations exclude possible diagnosis. Signs in other cranial nerves dysfunction or any neurological abnormality preclude the diagnosis in typical trigeminal neuralgia and indicate the discomfort may be due to a pathological lesion [9]. TN is often misdiagnosed due to the absence of consistent clinical or laboratory testing and several times before a confirmed diagnosis is made, patients seek help from multiple physicians [10].

The management options for trigeminal neuralgia patients depend on a number of factors including age, general health, seriousness of condition, and the underlying cause. The decision will be taken following an interview between the patient doctor [11].

The objective of this article is to provide an up-to-date review regarding the prevalence, etiology, diagnosis, and the management of trigeminal neuralgia.

Prevalence

Trigeminal neuralgia has a prevalence of 0.1 to 0.2 per 1,000 and an incidence of 4 to 20 cases per 100,000 persons per year [12]. The ratio female to male is about 3:2. Early literature indicated high female preponderance; however, current statistics show that only around 60% of TN patients are female [13]. For women the annual incidence is about 5.9 cases/100,000 women. For men, that is about 3.4 cases/100,000 people [14]. Though common after 50 years, TN is uncommon in young adults and uncommon in infants [15]. In most of the patients, TN affects only one side of the face, it was estimated that the right side of the face is more frequently involved [16].

Etiology

The trigeminal nerve is the fifth cranial nerve which is responsible for the sensory supply of the face and the motor and sensory supply to the muscles of mastication [17]. The exact cause of trigeminal neuralgia is unknown [18]. TN is classified into the classic, idiopathic, and secondary. Idiopathic TN is characterized by unknown causes which, even after surgical procedures or magnetic resonance imaging, remain undiagnosed. Classic TN is in the trigeminal root entry zone associated with NVC. An underlying disease such as tumors, artery malformations, multiple sclerosis can cause secondary TN [19].

Most cases are referred to as idiopathic but others are associated with the systemic compression of the trigeminal nerve by an aberrant loop of an artery or vein near its exit from the brainstem. In classical TN, compression of the trigeminal nerve root occurs within a few millimeters of its entry into the pons. Between 80% and 90% of the cases of TN are caused by compression by an adjacent artery or a vein [20]. The most frequent artery involved in this condition is the superior cerebellar artery, as seen in 75 to 80% of cases of TN.

A minority of cases are caused by diseases (secondary TN) such as multiple sclerosis or tumor obstruction of the nerves as other rare causes of trigeminal neuralgia include focal arachnoid thickening, adhesion, traction, binding or torsion, fibrous ring around the root, cerebellopontine angle tumors, infarction of the brain stem, aneurysm and arteriovenous malformation [21].

Pathophysiology

TN is thought to be related to demyelination of the nerves that arises at the compression site. There is no clear explanation for how demyelination contributes to TN symptoms. This is believed to be due to the generation of ectopic impulses formed by the demyelinated

lesion, thereby triggering ephaptic transmission [22]. Radiological and pathological studies have shown such vessels to be close to the trigeminal nerve root. The mainly involved vessel is the superior cerebellar artery. This hypothesis is further reinforced by symptom relief following surgery to separate the offending vessels from the nerve. Multiple other conditions like amyloid infiltration, bony compression, arteriovenous malformation and small infarcts in the medulla and pons, have been described to cause TN [23].

Trigger zones can be found in affected nerve distribution. Typically, those are situated near the midline. They were recorded mainly in the perioral and nasal regions [24]. Triggers reported to induce paroxysms of trigeminal neuralgia include teeth brushing, rashing, face washing, drinking, chewing, laughing, grimacing, or exposure to cold weather [25]. A prospective clinical and neuroimaging research in MS patients showed a substantial correlation between neurovascular compression and TN secondary to MS, indicating that a pontine plaque affecting primary afferents intra-axial and neurovascular compression in combination may induce TN secondary to MS by a double-crush process involving inflammatory demyelination and mechanical demyelination, of the same first-order neurons [26].

Diagnosis

Historically, physicians have sometimes ignored neuropathic pain, and patients have been diagnosed as being hypersensitive. Recent research has shown that neuropathic pain can be the underlying cause of a variety of secondary symptoms which significantly affect patients ' quality of life [27].

Doctors usually evaluate a patient's pain by personal records and conduct a physical examination but are analytical in the test. While various neurophysiological procedures can be used to examine the trigeminal function, in patients with secondary TN, trigeminal reflex testing has a diagnostic accuracy and responsiveness close to 90% [28]. This approach is simpler and less invasive than the approach evoked, with the detection of some phenomenon indicating an underlying mechanism. Autonomic symptoms such as conjunctival tearing or facial flushing are significant distinguishing points between TN and the rarer autonomic trigeminal cephalalgia. Pain localized to the first trigeminal nerve division needs to be evaluated more carefully, as it is more likely to be a trigeminal autonomic cephalalgia than TN [29].

Reliable method for diagnosing NVC in patients with refractory TN undergoing microvascular decompression (MVD) is preoperative high-resolution three-dimensional MRI. Imaging consisting of high-resolution, three-dimensional fast low-angle shot with three-dimensional positive steady-state interference may illustrate the relationship between the intracisternal segment of the trigeminal nerve and the adjacent vessels [30].

Further standardization with which physicians will detect neuropathic pain is also a need. Recently developed screening questionnaires and diagnostic techniques such as standardized sensory assessment, pain-related evoked ability and skin biopsy have advanced the mechanistic approach to pain control and helped develop the so-called sensory profiles [31].

Management

In the field of neurology and neurosurgery, the management of trigeminal neuralgia is challenging. Management strategies for trigeminal neuralgia patients depend on a variety of factors including general health, age, disease severity and underlying cause [32]. The therapeutic approach varied from physiotherapy, to local anesthesia use, pharmacological diagnosis, and surgical administration. Health treatment is counseling on first phase [33].

Local anesthetics: TN patients with a specific trigger zone, particularly those intra-orally, can benefit from local blocks of anesthesia such as those provided by dentists, although with a short-term relief [34]. In certain patients can be very useful in intravenous injection of a combination of magnesium and lidocaine. For the treatment of TN, local anesthetics such as alcohol, glycerol, phenol, tetracaine, or

bupivacaine injections are used. In some TNs, five percent lidocaine plaster [35] and eight percent capsaicin patch [36] may be useful. Such blocks can also have several hours of full analgesia, which is particularly helpful in acute TN exacerbations when waiting for systemic drugs to become therapeutic [37].

Pharmacological management: Carbamazepine is considered the first-line drug for immediate therapeutic management of signs of trigeminal neuralgia, since it has demonstrated improved pain reduction relative to placebo. The analgesic mechanism is unknown [38]. Its effect may be related to the blocking of voltage-sensitive sodium channels resulting in stabilization of hyperexcited neural membranes, inhibition of repetitive firing or reduction of synaptic impulse propagation [39]. Carbamazepine has side effects such as drowsiness, dizziness, fever, injury to the liver and ataxia. It is typically begun at a small dosage, and the dosage is raised slowly until the pain is managed. For most patients it reduces discomfort in the early stages of the disease [40]. In certain patients, however, carbamazepine's efficacy diminishes over time.

There is a lack of studies evaluating the efficacy of carbamazepine care, but general expert opinion indicates that long-term (five to 10 years) pain control may present a risk of failure exceeding 50%. You will be given the normal doses of carbamazepine (200 to 1200 mg/day) and oxcarbazepine (600 to 1800 mg/day) [41]. Oxcarbazepine is an effective alternative to carbamazepine, started twice daily at 150 mg and increased as absorbed by 300 mg per three days before pain relief is obtained. Twice regular the treatment doses range from 300 to 600 mg. Gabapentin is effective and widely used for neuropathic pain although trigeminal neuralgia lacks evidence [42]. Therefore, the application of gabapentin depends on the similarity between trigeminal neuralgia and other neuropathic pain, rather than on their clear distinctions. Gabapentin is started at 300 mg daily, which can be increased when tolerated slowly by 300 mg per 2 - 3 days. Gabapentin does not interact with other drugs and has relatively minor side effects that may include dizziness, drowsiness, headache, diarrhoea, confusion, nausea and swelling of the ankles [43].

When carbamazepine is ineffective, or gives only partial relief, other drugs can be attempted. If applicable, these may be replaced or added to carbamazepine. Baclofen (Lioresal) has been shown to be helpful in dosages of 10 - 80 mg daily. Specific therapies with recorded success include smaller trials or case reports include pregabalin, phenytoin (Dilantin), lamotrigine (Lamictal), gabapentin (Neurontin), topiramate (Topamax), clonazepam (Klonopin), pimozide (Orap), and valproic acid (Depakene) (12 mg/day) [44].

Surgical management: A variety of surgical techniques are available for patients with refractory pain, including MVD, percutaneous radiofrequency rhizotomy, percutaneous glycerol rhizotomy, percutaneous balloon compression, and stereotactic radiosurgery [45]. The recommendation to perform an invasive neurochirurgical procedure or a minimally invasive stereotactic radiotherapy treatment will be focused on the clinical evidence and not solely on results from neuroimaging. Both treatments have a high initial reaction time, with the exception of stereotactic radiosurgery, which typically takes full effect within one to two months [46].

Microvascular decompression is one of the most common procedures used to treat trigeminal neuralgia. This is helpful to TN patients, where nerve root compression is the cause. Microvascular decompression provides the highest chance of long-term pain relief, with very low risk of visual facial deprivation and other mild complications [47]. In some patients this procedure may result in longer than 10 years of sustained pain relief. While this technique is the most effective, it is also the most invasive. In addition, nothing is mentioned about the quality of life after MVD in the literature, but it has been seen as patients undergoing primary surgery without recurrence and showing no signs of depression and is very satisfied after MVD. Complications described after this procedure include infections, facial palsy, facial numbness, cerebrospinal fluid leakage and hearing deficiency with a mortality rate of 0.1 percent [48].

Ablative procedures include rhizotomy with thermocoagulation or chemical injection. Such treatments include destroying the base of the trigeminal nerve, thereby interrupting the signals of pain transfer through the brain. Thermocoagulatory rhizotomy uses an electrode to apply heat to damage the nerve fibers [49]. Chemical rhizotomy involves injecting the drug, glycerol into the trigeminal nerve and

thereby causing damage. Glycerol injection into the trigeminal cistern determines pain relief by demyelination and axonal displacement in patients with TN [50]. There is evidence that the effectiveness of glycerol rhizotomy relies on a degree of sensory deprivation postoperatively and that if facial pain was present during glycerol injection, the odds of successful outcome will be improved [51].

Percutaneous balloon compression (PBC) Balloon compression involves pushing a small balloon to the point where nerve fibers are located. Percutaneous trigeminal balloon compression rhizotomy is typically intended for patients who are unable to withstand or refractory to the above procedures. Thermocoagulated rhizotomy uses an electrode to conduct heat to destroy the nerve fibers. Balloon compression means pushing a small balloon to the point where the nerve fibers are located [52].

Radiofrequency thermocoagulation: This technique involves the use of instrumentation in radiosurgery. This is a non-invasive procedure, in which a highly concentrated dose of ionizing radiation is delivered at the trigeminal nerve root to an accurate target [53]. The radiation produces a lesion at the root of the nerve and thereby blocks the signs of pain from delivery to the brain. An initial pain relief was reported in excess of 90 per cent with a recurrence rate of up to 25 per cent. The reported side effects, such as masticatory weakness, dysesthesia and corneal numbness, appear to be related to significant individual variations in somatotopic organization of trigeminal nerve fibers and irreversible damage to small, unmyelinated pain fibers [54]. In addition, the use of neuronavigator and computer tomography to boost needle position seems to be correlated with lower complications and recurrence rates in recent studies relative to traditional fluoroscopy [55].

Peripheral neurectomy and nerve block: neurectomy can be performed on peripheral trigeminal nerve branches such as supraorbital, infraorbital, lingual, and alveolar nerves [56]. Alcohol injection, incision, cryotherapy or radiofrequency lesioning can accomplish this. Peripheral neurectomy in older patients in remote and rural areas may be safe, where neurosurgical facilities are not readily available [57].

Conclusion

TN is a rare disease but often associated with debilitating pain and disability. TN management is a concern for neurologists and neurosurgeons alike. Progress has been achieved in recent years, leading to the introduction of neuro-radiological approaches for both pathogenesis and surgical treatment. Medical treatment should be done in TN. With an growing variety of medications available, it is possible the surgical alternative may not be available for several years.

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