Review Article

Trigeminal neuralgia: Revisited

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ABSTRACT

Trigeminal neuralgia (TN) is an extremely painful condition characterized by recurrent attacks of sharp, intermittent, pricking type pain in the trigeminal nerve distribution, primarily affecting the elderly woman. The pain is nearly always unilateral, and it may occur repeatedly throughout the day. The diagnosis is typically determined by thorough clinical examination, history taking, and should be discriminated from other type of facial pain. This article highlights the general features, historical perspectives, etiological characteristics, pathophysiology, differential diagnosis, therapeutic options, and responsibilities of the oral healthcare professionals in diagnosing and managing patients with TN.

Key words: Facial pain, pain, tic douloureux, trigeminal nerve

INTRODUCTION

"Pain is a perfect misery, the worst of evils; and excessive, over-turns of all patience" says John Milton, paradise lost.

Pain is often the primary motivator for patients to seek healthcare in general and dental treatments. Dental treatment is closely associated with pain. Most dental patients expect to experience some degree of pain during treatment and dentists often use pain as a diagnostic tool. The ability to diagnose a disease and treat the patient depends on the knowledge of the mechanism and behavioral characteristics of pain in its various manifestations.

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Trigeminal neuralgia (TN), often called as tic douloureux, is the most common of the cranial neuralgias with an incidence of 4.3 per 100,000 persons per year. The International Association for the Study of Pain (IASP) defines TN as "a sudden, usually unilateral, brief stabbing recurrent pain in the distribution of one or more branches of the fifth cranial nerve".[1]

It is characterized by a sudden brief, severe, electric shock-like, or stabbing pain typically felt on one side of your face, provoked by light touch, which may remit for varying periods and is limited to one or more branches of the trigeminal nerve. Mostly it involves the maxillary or mandibular distribution of the nerve. TN is a severe unilateral paroxysmal facial pain, often described by patients as the "the world's worst pain."[2]

History of TN

This condition has been known since ancient times and the first ever clinical description of this condition was given by Aretaeus of Cappadocia in the 1st century AD. In 11th century, Jujani, discussed a condition of unilateral facial pain and suggested that it could be resulted from 'the proximity of the artery to the nerve'.[3] In 1677, John Locke was the first physician to state that this condition was due to the neuralgia of trigeminal nerve.

Nicolaus Andre, a French physician described it as a distinct clinical entity and coined the term tic douloureux in 1756.[4] The first written description of tic has been ascribed to

Johannes Bausch in 1672. Fothergill provided a vivid description of this pain syndrome in 1773. Early medical therapies were not efficacious; the first useful treatment was probably trichloroethylene inhalation, which was initiated in the 1920s. It is believed that the 16th century stone carvings in well cathedral depict the pain of TN. Mareschal, surgeon of King Louis XIV of France, cut the peripheral branches of the trigeminal nerve for pain.[5]

Etiopathogenesis

The precise mechanism of TN still remains unclear. It is often idiopathic, but is usually associated with pathosis somewhere along the course of the trigeminal nerve. When younger individuals are involved, suspicion of a detectable underlying lesion such as a tumor, an aneurysm, or multiple sclerosis must be increased and are referred to as secondary neuralgia.[6] Because so many of its features are consistent with a central nervous system (CNS) disease, TN has been called "a pain syndrome with a peripheral cause but a central pathogenesis".

Currently, there are three most popular TN etiologic theories. First theory is based on diseases-related, second is direct trauma to the nerve, and the third theory propagates the polyetiologic origin of the disease [refer Table 1].

International Classification of Headache Disorders II (ICHD-II) further subdivides TN into "classic TN" and "symptomatic TN".

Classic TN is defined as: "A unilateral disorder characterized by brief electric shock-like pains, abrupt in onset and termination, and limited to the distribution of one or more divisions of the trigeminal nerve". Pain is commonly evoked by trivial stimuli including washing, shaving, smoking, talking, and/or brushing the teeth (trigger factors) and frequently occurs spontaneously. Small areas in the nasolabial fold and/or chin may be particularly susceptible to the precipitation of pain (trigger areas). The pains usually remit for variable periods.[3]

Symptomatic TN is defined as: "Pain indistinguishable from classic TN, but caused by a demonstrable structural lesion other than vascular compression".

Clinical features

TN is the most common facial neuralgia.[8] Characteristic clinical features, which include episodes of intense shooting stabbing pain that lasts for a few seconds and then completely disappears. The pain characteristically has an electric shock-like quality and is mostly unilateral. The maxillary branch is most commonly affected, followed by the mandibular branch and (rarely) the ophthalmic branch.

Women are almost affected twice as men. The incidence gradually increases with age and is rare below 40.[2] Although individual pains or pain spasms last only a few seconds, several attacks may follow each other for up to 30 min of rapidly repeating volleys. Patients often clutch at the face and experience spasmodic contractions of the facial muscles during attacks, a feature that long ago led to the use of the term tic douloureux ("painful jerking") for this disease. This refractory period can be useful, clinically, in distinguishing neuralgic pain from a stimulus-provoked odontogenic pain source.

Pain in TN is precipitated by light touch on a "trigger zone" present on the skin or mucosa within the distribution of the involved nerve branch. Common sites for trigger zones include the nasolabial fold and the corner of the lip. Shaving, showering, eating, speaking, or even exposure to wind can trigger a painful episode. Intraoral trigger zones can confuse the diagnosis by suggesting a dental disorder. The number of attacks may vary from one or two per day to several per minute.

The diagnosis of TN is made clinically by excluding other possible causes of facial pain and is based on signs and symptoms from the patient history, such as a trigger zone, typical unilateral lancinating paroxysms following neural disturbance, and a refractory period.[4] The International Headache Society (IHS) has published criteria for the diagnosis of classical and symptomatic TN.

Classical TN

a. Paroxysmal attacks of pain lasting from a fraction of a second to 2 min, affecting one or more divisions of the trigeminal nerve, and fulfilling criteria B and C.
b. Pain has at least one of the following characteristics:

Table 1. The most accepted theories of trigeminal neuralgia[7]

Diseases related	Direct injury to the trigeminal nerve		Polyetiologic origin
	Peripheral part of TNS	Central part of TNS	
Vascular diseases, multiple sclerosis, diabetes mellitus, rheumatism and others	"*Allergic hypothesis*" due to odontogenic inflammatory diseases, otolaryngological pathology, getting cold and others. "*Compression syndrome hypothesis*" due to narrowing of the osseous canals, trauma.	"*Neurovascular compression hypothesis*" at the root entry zone due to arteriovenous malformation, vestibular schwannomas, meningiomas, epidermoid cysts, tuberculomas, various other cysts and tumors, aneurysm, vessel aggregation and occlusion due to arachnoiditis and others.	All possible etiological factors that can affect TNS and evoke demyelination and dystrophy.

TNS – Trigeminal nervous system

1. Intense, sharp, superficial, or stabbing.
2. Precipitated from trigger zones or by trigger factors.

c. Attacks are stereotyped in the individual patient.
d. There is no clinically evident neurologic deficit.
e. Not attributed to another disorder.

Symptomatic TN

a. Paroxysmal attacks of pain lasting from a fraction of a second to 2 min, with or without persistence of aching between paroxysms, affecting one or more divisions of the trigeminal nerve, and fulfilling criteria B and C.
b. Pain has at least one of the following characteristics:
   1. Intense, sharp, superficial, or stabbing.
   2. Precipitated from trigger zones or by trigger factors.
c. Attacks are stereotyped in the individual patient.
d. A causative lesion, other than vascular compression, has been demonstrated by special investigations and/or posterior fossa exploration.

When these criteria are partially fulfilled, alternative terms such as atypical TN, atypical facial pain, and atypical facial neuralgia are applied.

Disorders like cluster headache, dental pain, glossopharyngeal neuralgia, giant cell arteritis, multiple sclerosis, migraine, intracranial tumors, otitis media, paroxysmal hemicranias, postherpetic neuralgia, sinusitis, temporomandibular joint (TMJ) syndrome, and trigeminal neuropathy should be included in the differential diagnosis of TN.[9]

Management

The initial treatment of choice for TN is medical therapy, and most patients have at least temporary relief with the use of selected agents. Patients who have no response to or who relapse with medical therapy or who are intolerant of medical treatment should be considered for surgical treatment.

Anticonvulsant drugs are most frequently used and are effective. Carbamazepine is the drug of choice. Skin reactions, including generalized erythema multiforme, blood dyscrasias should be taken into account before prescribing it. Baclofen or by combining carbamazepine with baclofen was indicated in few cases. Gabapentin, a newer anticonvulsant drug, is effective in some patients. Additional medications with reported success in smaller studies or case reports include phenytoin, lamotrigine, gabapentin, topiramate, clonazepam, pimozide, valproic acid, type A botulinium toxin, topical capsaicin, intramuscular sumatriptan, intranasal lidocaine, and dextromethorphan.

Surgical therapy is indicated where drugs are ineffective. It may be percutaneous or open. Percutaneous techniques include glycerol injection, radiofrequency rhizotomy, balloon decompression, and gamma knife stereotactic radiosurgery. Open techniques include partial trigeminal rhizotomy and microvascular decompression.[10]

CONCLUSION

Healthcare professionals, especially the dentists who frequently encounter the pain in head and neck region on their routine practice should have a sound knowledge over the diagnosis and management of this disease. Patients with suspected TN should be examined carefully by specialists who have expertise in assessing and diagnosing possible pathological processes and be able to eliminate the contributing factors for effective management.

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