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Review Article

Trigeminal Neuralgia: Causes and its Management

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***ABSTRACT*:**
Various drugs and surgical procedures have been utilized for the treatment of trigeminal neuralgia (TN). Despite numerous available approaches, the results are not completely satisfying. The need for more contemporaneous drugs to control the pain attacks is a common experience. Moreover, a number of patients become drug resistant, needing a surgical procedure to treat the neuralgia. Nonetheless, pain recurrence after one or more surgical operations is also frequently seen. These facts reflect the lack of the precise understanding of the TN pathogenesis. Classically, it has been related to a neurovascular compression at the trigeminal nerve root entry-zone in the prepontine cistern. However, it has been evidenced that in the pain onset and recurrence, various neurophysiological mechanisms other than the neurovascular conflict are involved. Recently, the introduction of new magnetic resonance techniques, such as voxel-based morphometry, diffusion tensor imaging, three-dimensional time-of-flight magnetic resonance angiography, and fluid attenuated inversion recovery sequences, has provided new insight about the TN pathogenesis. Some of these new sequences have also been used to better preoperatively evidence the neurovascular conflict in the surgical planning of microvascular decompression. Moreover, the endoscopy (during microvascular decompression) and the intraoperative computed tomography with integrated neuronavigation (during percutaneous procedures) have been recently introduced in the challenging cases. In the last few years, efforts have been made in order to better define the optimal target when performing the gamma knife radiosurgery. Moreover, some authors have also evidenced that neurostimulation might represent an opportunity in TN refractory to other surgical treatments. The aim of this work was to review the recent literature about the pathogenesis and medical and surgical treatments of trigeminal neuralgia, and discuss the significant advances in all these fields.
Key words: Pain, trigeminal neuralgia, tic douloureux,

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INTRODUCTION

Trigeminal neuralgia (TN) is defined by recurrent unilateral brief electric shock- like pain that is abrupt in onset and termination. Pain is restricted to one or more of the trigeminal divisions and is triggered innocous sensory stimuli.[1] It is a facial pain syndrome characterised paroxysmal, shock-like pain attacks located in the somatosensory distribution of the trigeminal nerve.[2]

In 1773, trigeminal neuralgia was denoted as tic doloureux or Fothergill's disease. During a pain episode the facial muscles related to expression and mastication usually contract, which justifies the definition tic douloureux.[3]

Trigeminal neuralgia pain is not only extremely painful, it is also characteristic that the pain is sudden and unexpected and short lasting , hence the term paroxysm.[1] The pain is often evoked by trivial stimulation of appropriately named " trigger zones".[4] Stimulation is usually caused by activities such as hair combing, swallowing, shaving, yawning, tooth

brushing and touching one's face[3]. The prevalance of TN in the general population is 0.015%.

Facial pain has a considerable impact on quality of life. It has been recently shown that TN is the most frequent type of facial pain and that, among facial pain syndromes, the overall incidence of TN has remained constant ranging from 12.6L/100,000/year 27/100,000/year.[2]

EPIDEMIOLOGY

The incidence of TN is variably reported between studies, with a range from 4.3 to 27 new cases/100,000/year. The incidence is higher among women and increases with age. The lifetime prevalance was estimated to be 0.16-0.3 % in population based studies. The average age of onset is 53 years in classical TN and 43 years in secondary TN, but the age of onset can range from early to old age.[1]

CLASSIFICATION

According to the beta version of the 3rd edition of the International Classification of Headache Disorders (ICHD-3 Beta), trigeminal neuralgia has been classified as:

ICHD3-beta

Criteria:

A. At least three attacks of unilateral facial pain fulfilling criteria B and C.

B. Occurring in one or more divisions of the trigeminal nerve, with no radiation beyond the trigeminal distribution.

C. Pain has at least three of the following four characteristics:

- recurring in paroxysmal attacks lasting from a fraction of a second to 2 minutes
- severe intensity
- electric shock-like, shooting, stabbing or sharp in quality.
- precipitated by innocuous stimuli to the affected side of the face.

D. No clinically evident neurological.

E. Not better accounted for by another ICHD-3 diagnosis.

Subclassification:

Classical TN

- TN with purely paroxysmal pain
- TN with concomitant persistent pain

Symptomatic TN

- TN associated to multiple sclerosis (MS): MS has been diagnosed and MRI demonstrates MS plaque affecting the trigeminal nerve root or electrophysiological studies indicate impairment of the affected nerve(s). Pain is not necessarily unilateral.
- TN associated to space-occupying lesion: contact between a space-occupying lesion and the affected trigeminal nerve has been demonstrated by imaging and pain has developed after contact occurred between the lesion and the trigeminal nerve, or led to its discovery .

Recently the International Association for the Study of Pain (IASP) has produced independent classification, definition and diagnostic process of trigeminal neuralgia.

IASP

Definition

Orofacial pain restricted to one or more divisions of the trigeminal nerve. With the exception of TN caused by multiple sclerosis, the pain affects one side of the face. It is abrupt in onset and typically lasts only a few seconds (2 minutes at maximum). Patients may report their pain as arising spontaneously but these pain paroxysms can always be triggered by innocuous mechanical stimuli or movements. If patients experience additional continuous pain in the same distribution and same period as the paroxysmal pain it is considered to be TN with concomitant continuous pain and this phenotype may be present in each of the three subclassification categories:

Criteria:

A. Orofacial pain distributed within the trigeminal facial or intraoral territory.

B. Paroxysmal character of pain.

C. Pain triggered by typical maneuvers.

Subclassification :

- Idiopathic TN: no apparent cause.
- Classical TN: caused by vascular compression of the trigeminal nerve root resulting in morphological changes of the root.
- Secondary TN: caused by major neurological disease, e.g., a tumour of the cerebellopontine angle or multiple sclerosis.[5]

ETIOLOGY

Etiologically established TN , that is based on identification of a cause for the trigeminal neuralgia, corresponds to 2 categories: classical and secondary TN are defined by an underlying cause. Both diagnostic entities qualify as definite neuropathic pain.

However, in a relatively small proportion of patients with clinically established TN , even the most advanced diagnostic investigations fail to show a cause. This condition is categorised as idiopathic trigeminal neuralgia.[6]

80-90% of cases that are technically still classified as idiopathic are caused by compression of trigeminal nerve (fig1) close to its exit from the brain stem by an aberrant loop of artery or vein.

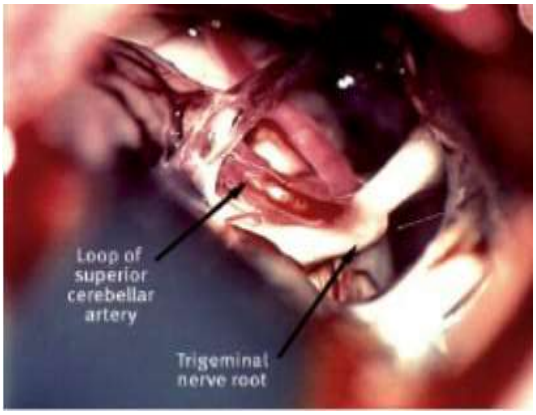

Fig 1 | Compression of the trigeminal nerve root by an aberrant loop of the superior cerebellar artery

Importantly, compression is of the root entry zone, where axons are coated with central nervous system myelin rather than peripheral nerve myelin. Similarly, vascular compression of the facial and glossopharyngeal nerve is thought to be responsible for most cases of hemifacial spasm and glossopharyngeal neuralgia, respectively.[4]

- Classical TN is defined as a specific category of TN in which MRI demonstrates vascular compression with morphologic changes of trigeminal nerve root.[6] As early as 1934, Dandy proposed that in atleast 30% of TN patients the pain as caused by a blood vessel compresssing the trigeminal nerve.
  Today, it is generally agreed that the most common cause of classical TN is compression or other morphological changes of the trigeminal nerve by a blood vessel, usually an artery in cerebellopontine cistern. This is termed a neurovascular conflict with compression.
- In secondary TN, pathophysiological mechanism is most likely the same as in classical TN but the etiology is dependent on the specific structural lesion, most frequently an MS plaque affecting the trigeminal root or a space-occupying lesion in the cerebellopontine cistern such as epidermoid tumours, meningiomas,arteriovenous malformations or aneuryms.[1]

CLINICAL FEATURES

- Trigeminal neuralgia is of a searing, stabbing or lancinating type.[7] Although one single pain paroxysm may recur, after a refractory period,many times a day and the may come in series of attacks with many paroxysms close together.[1]
- It affects more commonly maxillary and/or mandiular branches i.e., 2nd and 3rd branch of trigeminal nerve distribution(fig2) than the ophthalmic.[7]

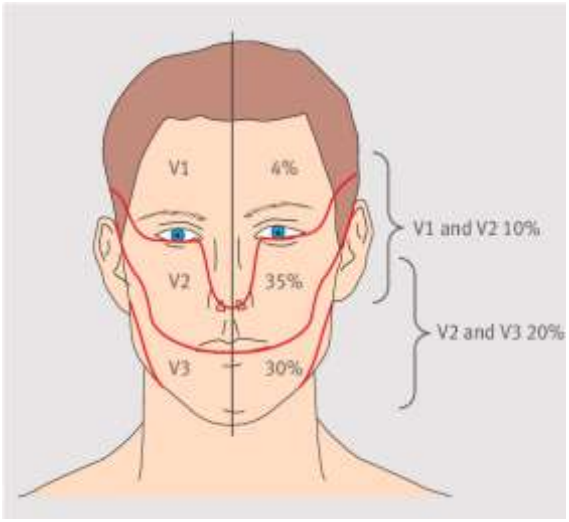

Fig 2 | Distribution of trigeminal neuralgia. In another 1% of patients it also affects all three divisions and rarely it can be bilateral (though paroxysms are not synchronous).[4]

- Right side is slightly , but significantly more often affected than the left side. Bilateral TN is very rare in classical TN and should raise suspicion of secondary TN.[1]
- Almost twice as many women are affected as man. The incidence gradually increases with age and is rare below 40 years of age.[4]
- It is highly characteristic that pain trigerred innocuous sensory stimuli to the affected side of the face.
- Sensory stimuli may be E/O and I/O. Most frequent trigger factors involve normal daily activities such as light touch, talking, chewing, cold wind against the face, brushing teeth, shaving.[1]
- The term 'tic dououreux' is probably applied only when the patient suffers from spasmodic contractions of facial muscles.
- In early stages of the disease the pain is relatively mild but as the attacks progress over a period of months or years they become more severe and tend to occur at more frequently intervals.
- The early pain has been termed 'pre-trigeminal neuralgia' by Mitchell and is sometimes dull aching or burning or resemling a toothache.
- The 'trigger zone' which precipitate an attack when touched are common on the vermilion border of lip, alae of the nose, the cheeks and around the eyes.
- Any portion of the face may be involved by the pain, depending upon which branches of fifth nerve are affected.[7]

DIAGNOSTIC CONSIDERATIONS

The diagnosis of TN is primarily based on patient history, as there are no definitive laboratory or diagnostic tests. TN may be indistinguishable based on pain characteristics.

- **Glossopharyngeal Neuralgia** causes evoked stabbing pain located to the back of the tongue, the pharynx or

deep in the ear. Trigger factors are somewhat different from TN and include swallowing,coughing,sneezing.[1]

- One of the most common conditions mistaken for TN is **migraine or migrainous neuralgia** (Horton's syndrome, histamine headache, histamine cephalgia), but this severe type of periodic headache is persistent atleast over a period of hours and has no trigger zone.
- Tumors of nasopharynx can produce a similar type of pain generally manifested in the lower jaw, tongue and side of the head with an associated middle ear deafness. This symptom complex, caused by a nasopharyngeal tumor has been called **Trotter's syndrome**.
- A condition clinically similar to TN often occurs after attacks of Herpes zoster of the fifth nerve, termed **Postherpetic neuralgia**, the pain usually involves the ophthalmic division of fifth cranial nerve, but commonly regresses within two three weeks.
- **Trigeminal neuritis or trigeminal neuropathy** is a poorly understood condition which has a variety of presumed causes:
- Some dental surgical procedure
- Pressure of a denture on dental nerve
- Surgical or mechanical trauma
- Tumous of head and neck
- Therapeutic use of hydroxystilbamidine isethimate.[7]

INVESTIGATIONS

Investigations are done to :

- Clarify the differential diagnosis for eg: by taking dental x-ray.
- Investigate whether there is an identifiable cause of the disease, particularly with a view to surgical cure. This is best done using Magnetic Resonance Imaging (MRI).[4]

MANAGEMENT

The major therapies for trigeminal neuralgia are drug treatment and surgical intervention.[3]

Medication is the first line treatment for Trigeminal Neuralgia. Phenytoin as the first drug used for TN with reported positive effects.

1. According to recent EFNS guidelines, two drugs are considered as first line therapy in Trigeminal Neuralgia: Carbamazepine[CBZ; 200-12--mg/day] and oxycarbazepine [OXC; 600-1800 mg/day]. CBZ has been found to reduce both frequency and intensity of painful paroxysms and was equally efficacious on spontaneous and trigger evoked attacks.

2. New therapeutic modalities have been tried. More specifically, according to a recent overview gabapentin combined with regular ropivacaine injections into trigger sites improved pain control and quality of life.

Various surgical approaches have been proposed for the treatment of drug resistant TN:

1. **Microvascular Decompression [MVD]** is performed with the objective to resolve the neurovascular conflict between an abnormal vessel and the trigeminal nerve. MVD is based on the assumption that a compression of trigeminal nerve by an abnormal vascular loop is the direct of TN.

2. **Percutaneous Balloon Compressiom [PBC]** was introduced in the clinical setting by Mullan et al and has been extensively used in the treatment of TN due to low cost, simplicity and the advantage of being the only percutaneous procedure performed with the patient under general anesthesia.

3. **Glycerol Rhizotomy** is performed by injecting glycerol in the trigeminal cistern which results in pain relief in patients with trigeminal neuralgia due to demyelination and axonal fragmentation. Dysesthesias, corneal numbness, masseter weakness and herpes labialis have been reported as frequent complications of this procedure.

4. **Radiofrequency thermocoagulation** is based on the attempt to electrocoagulate the trigeminal nerve and the gasserion ganglion rootlets.

5. **Gamma Knife Radiosurgery** has been used as a treatment modalities in several centres for patients with concurrent medical illness who were poor canditates for MVD or who refuse more invasive surgery. Usually, the root entry-zone of the trigeminal nerve is used as target and the dose protocols range from 70Gy to 100Gy.[2]

Prosthodontic considerations

Pain management of the patients can be done as mentioned above but some consideration need to be kept in mind while doing dental procedure.

The relation between dental maloclusions and paroxysms craniofacial pain was first described by Costen. Henderson pointed out that a trigger point in a masticatory muscle may be the single precipitating factor.The muscle is often tender.[8]

It has been suggested that neuralgia may be associated with pain because of myofascial dysfunction or some other temporomandiular disorder (TMD) and that functional and occlusal dental treatment may reduce pain. Physiotherapy with resources such as diathermy, therapeutic ultrasonography and therapeutic laser treatment for painful symptoms is fully acceptable in the treatment of TMD.[3]

Adjustment of dental malocclusion can benefit various forms of craniofacial pain. Dental factors clearly play a more important part in precipitating paroxysmal facial pain than has been previously realised. The pragmatic approach recommended in this investigation depends on the reduction of

trigeminal input below threshold level. Probably the correction of dental abnormalities reduces the sensory inflow from masticatory muscles which were malfunctioning because of abnormal occlusal face height or incorrect occlusal relationship. Dental correction lead to abolition of intraoral triggers and somewhat, unexpectedly, extraoral triggers as well.

- Dentist should examine all patients suffering from trigeminal neuralgia and correct an dental abnormality. Most patients have worn dentures for some years before the onset of trigeminal neuralgia. Often the denture did not preserve the correct relationships between the upper and lower jaws. Indeed, a long period of wearing incorrect dentures may alter the state of the masticatory muscles and establish conditions favouring the development of trigeminal neuralgia.[8]
- Anesthesia should be without epinepherine for the local anaesthetic. Long acting local anesthesia should be used to avoid multiple injections.
- The dentist should inject the local anesthetic at a site as far away as possible from the trigger point for the TN pain.

Conclusion

TN is a rare but debilitating condition that can initially be treated in primary care by both medical and dental practitioners, but ultimately care needs to be shared with specialists who can provide a wide range of treatment options.[9]

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