Trigeminal neuralgia and its management

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Abstract

Trigeminal neuralgia is a disorder of unknown etiology characterized by sudden, recurrent, short paroxysms of high intensity facial pain occurring in later life(50-60 years) and females are affected more than males. Right-sided facial affliction predominates with the maxillary nerve most commonly involved. The condition is generally progressive with the pain worsening at increasing frequency. Medical management is usually the first option. In refractory cases, surgical modalities can be considered. This review gives a brief knowledge about the pathogenesis and management of trigeminal neuralgia.

Introduction

Trigeminal neuralgia, also called as TIC DOULOUREUX, is a common orofacial pain which is characterized by sudden, usually unilateral, severe, brief, stabbing, lancinating, paroxysmal, recurring pain in the distribution of one or more branches of fifth cranial nerve, that is, trigeminal nerve. It is seen in about 4 in 100,000 persons and is usually idiopathic or may occur secondary to a major, recognizable neurological disorder.[1]

It is generally seen in late middle age (5^{th}-6^{th} decade) with a female predilection. The patient complains of sudden, intermittent, shock-like pain (severe, brief, stabbing, and shooting like pain)[2] which is mostly unilateral in nature and follows the sensory distribution of trigeminal nerve primarily affecting the maxillary and mandibular divisions. Trigger zone is an area of facial skin or oral mucosa, where low intensity mechanical stimulation like light touch, an air puff, or during activities like speaking, brushing, smiling, shaving and chewing, a typical pain attack is elicited. The location of trigger points depends on the division of trigeminal nerve involved. In extreme cases, the patient will have a motionless face (mask like face).

Most patients respond well to carbamazepine, however in advanced cases, surgical modalities can be considered, as treatment options.

Etiology

The disease is usually idiopathic in nature. However, certain benign and vascular anomalies may compress the trigeminal nerve root and produce clinical symptoms. Vascular factors like transient ischemia and autoimmune hypersensitivity responses, as well as, mechanical factors like pressure of aneurysms of internal carotid artery, also play a role in initiating the disease.[3]

Various other etiological factors are:

Infections: Various granulomatous and non-granulomatous infections involving the fifth cranial nerve, may produce neuralgic pain.

Ratner's jaw bone cavities: Cavities found in the alveolar and jaw bones may also act as a causative factor for trigeminal neuralgia.

Multiple sclerosis: Presence of sclerotic plaque at the root entry zone of the trigeminal nerve may initiate the disease.

Petrous ridge compression: Trigeminal neuralgia may be caused by compression of the nerve at the dural foramen or over the petrous tip.

Post traumatic neuralgia: Traumatic injuries and certain dental procedures like fractured facial bones, cysts and tumors may involve the trigeminal nerve and cause neuralgic pain.

Intracranial tumors: Lesions like epidermoid tumors, meningioma of cerebellopontine angle and Meckel's cave, arteriovenous malformations, aneurysms and vascular compression may also result in trigeminal neuralgia. Trigeminal neuromas in the middle cranial or posterior fossa may also act as a causative factor for the disease.

Intracranial vascular abnormalities: Compression of the trigeminal nerve or aneurysms of the internal carotid artery may result in trigeminal neuralgia.

Viral etiology: Post herpetic neuralgia is sometimes seen in elderly patients.

Artery anomaly: The superior cerebellar artery lies close to the sensory root of trigeminal nerve and any anomaly of this artery can cause trigeminal neuralgia.

Pathogenesis

The primary pathological factor in trigeminal neuralgia is demyelination of sensory axons due to sustained (static) or pulsatile micro vascular compression of the trigeminal root.[4]

There is ectopic degeneration of spontaneous nerve impulses and erratic transmission of the axonal fibers. When the nerve is injured it becomes 'hyper-excited'. The result is that the nerve fibers inside the nerve itself fire off signals of pain at the minimum provocation. Pain fibers can "short circuit" with touch fibers, due to loss of insulating myelin, and this results in a "sparking" between the two, sending incorrect pain signals to the brain. The myelin tries to heal itself and

this sometimes results in successive periods of remission when no pain occurs.[5]

Diagnosis

The diagnosis of trigeminal neuralgia depends upon the clinician's ability in recognizing a distinctive series of signs and symptoms that define this disorder. The various diagnostic aids which can be used include:

a. **Sweet criteria:** by White and Sweet

The pain:

Table 1: Sweet Diagnostic criteria (Neelima Anil Malik)

1. is paroxysmal
2. may be provoked by light touch to the face
3. is confined to trigeminal distribution
4. is unilateral
5. The clinical sensory examination is normal.

b. **Diagnostic criteria of international headache society (IHS):** given in the year 1998[6]

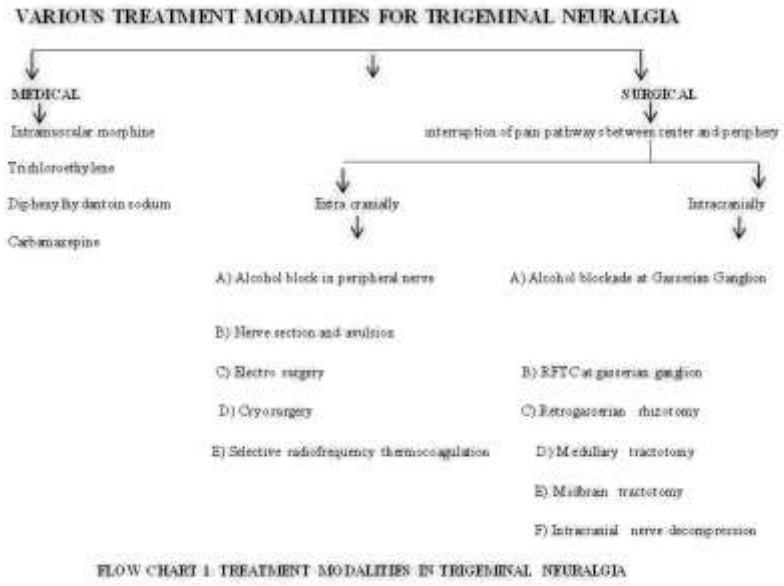
Table 2: IHS diagnostic criteria (Joffroy, Levivier, and Massager)

1. Paroxysmal attack of facial pain which last a few seconds to less than 2 minutes
2. Pain has at least 4 of the following characteristics:
a. distribution along one or more divisions of trigeminal nerve
b. sudden, intense, sharp, superficial, stabbing or burning in quality
c. pain intensity is severe
d. precipitation from trigger areas, or by certain activities such as eating, talking, cleaning the teeth or washing the face
e. between the paroxysms, the patient is entirely asymptomatic
3. Attacks are stereotyped in individual patients
4. No neurological deficit
5. Exclusion of other causes

a. MRI or CT scanning
b. Response to treatment with carbamazepine gives a definitive diagnosis
c. Diagnostic injections of a local anesthetic agent into the patients trigger zone should temporarily eliminate the pain.

Management

- **Peripheral neurectomy**
  - Supraorbital
  - Infraorbital
  - Lingual
  - Inferior alveolar
- **Newer approaches**
  - Physiologic inhibition of pain by transcutaneous neural stimulation
  - Acupuncture
- **Psychological approach**
  - Biofeedback
  - Psychiatric counseling
  - Hypnosis/ autosuggestion



FLOW CHART 1: TREATMENT MODALITIES IN TRIGEMINAL NEURALGIA

a. **Medical management:** Carbamazepine (CBZ) is the drug of choice. Treatmentbegins with 100 to 200 mg two or three times daily. Doses should be increased very progressivelyand titrated to the severity of the patient's pain. In some cases amaintenance dosage of 200 mg or 400 mg per day is sufficient to keep the patient pain-free.Side effects of CBZ include: hypersensitivity reactions, drowsiness, decreasedmental acuity, ataxia (in older patients), and dose-related leucopenia.

Second line of drugs includes Baclofen, Phenytoin, and Lamotrigine.

Table 3: Drug therapy in TN (Neelima Anil Malik)

Drug therapy for trigeminal neuralgia		
Drug	**Initial dose**	**Maintenance dose**
1. Gabapentin	300 mg TID	1800 mg
2. Baclofen	5 mg BID or TID	80 mg
3. Clonazepam	0.5 mg TID	4 mg
4. Lamotrigine	50 mg QD	300-500 mg
5. Oxcarbazepine	300 mg BID	1200 mg BID
6. Toprimate	50 mg QD	200 mg BID
7. Carbamazepine	100 mg BID	1200-2400 mg

b. **Surgical management:** If a patient is not relieved from pain with medical treatment, some form of surgical modality can be considered. Most surgical procedures aim to interrupt or block the electrical activity of the nerve and hence stopthe pain.

But surgical complications include:

- Cranial hemorrhage leading to a stroke
- Eye infections due to loss of sensation
- hearing loss on one side
- Difficulty with eating
- Surgical-wound infection; generalized infection
- Loss of sensation or alteration in sensation on one side of the face
- Anesthesia dolorosa i.e., continuous pain in an area that is numb

Various surgical treatments available include:

Peripheral surgery: This surgery is done very close to where the trigger area is located and includes: cryotherapy, alcohol block, laser, and neurectomy. Most of them are done under a local anesthetic and some require the use of stitches inside the mouth. These give short term pain relief, around 10 months on average, and cause few complications.

Minor surgery[7]: This is a non-invasive surgery as no cutting is done. Instead an instrument is passed just inside the skull under Xray control to enter the Gasserian ganglion. This is done under a short anesthetic and the patient can normally go home the next day.

In order to relieve the pain, nerve tissue is damaged using one of the following procedures:

- **Radiofrequency thermocoagulation:** Passing a current through the nerve which generates heat
- **Glycerol injection[8]:** Bathing the nerve in a toxic substance which over a few days destroys the nerves transmitting pain
- **Balloon micro compression:** The nerve is compressed by a small balloon which is blown up for a few seconds.

All have relatively few side effects but the majority result in numbness of varying degree and extent. The most severe side effect is anesthesia dolorosa.

Major Surgery

This surgery is carried out on the back of the skull behind the ear. It is done under full general anaesthetize.

- **Microvascular decompression[9]:** The only procedure that does not attempt to destroy the nerve but lifts off a blood vessel that is pressing on the nerve inside the brain. It leaves a scar in the hairline behind the ear. It gives the longest pain relief around 8 years for 50% of patients. It is associated with a 0.5% risk of death. Some short term side effects can occur and the most serious one although very rare is a stroke. Unilateral deafness can occur in up to 2% of patients. Numbness is very rare.
- **Rhizotomy:** If no compression is found at surgery the surgeon may partially cut the nerve. It results in loss of sensation on one side of the face.
- **Radiosurgery[10]**

Gamma knife surgery: This is a newer procedure. This procedure aims to damage part of the trigeminal nerve inside the brain. Using MRI and some special equipment, a beam of radiation is directed at the nerve. No surgery is involved so it can be done as day case surgery. Pain relief often does not occur immediately and it may be about three months before complete pain relief is obtained. Pain relief is probably in the order of years.

Conclusion

Trigeminal neuralgia is a severe, unilateral, paroxysmal facial pain. Neurovascular conflict is recognized as the main cause of idiopathic trigeminal neuralgia. However, irritation to the nerve or compression of the nerve by certain tumors may also lead to trigeminal neuralgia. Medical treatment is the first choice of treatment, but over a period of time, the drugs lose their efficacy. Due to advancement in science and technology and the knowledge about the cause of trigeminal neuralgia, various surgical procedures can also be carried out depending upon the severity of the disease.

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