Orofacial pain

**Pain:** - is a sensation of suffering resulting from a noxious stimulus, physical disorder, or mental derangement. It is, an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. The physiologic aspect of pain involves pain receptors stimulation, pain transmission, transduction, modulation and central integration in higher thought and emotional centers. Pain is, an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Acute pain resulting from injury will generally initiate a reflex withdrawal thus ensuring minimal or no tissue damage (nociceptive pain).

Allodynia: the injured region becomes sensitive to even light touch

Hyperalgesia: over reactive to painful stimuli.

**CLINICAL EVALUATION OF PAIN**

Clinical evaluation of pain requires an understanding of the patient's subjective perception of the discomfort. To reach such an understanding the following aspects must be studied

**A- Onset of pain:** A pain of brief duration from its onset to the request for treatment can suggest inflammatory somatic pain and exclude a chronic condition.

**B- Localization of the pain:** Somatic pain of the oral and perioral region nearly always arises from the affected site which is readily identified by the patient. Inability of the patient to localize pain may indicate somatic pain originating from deep tissues or the pain is not somatic. Radiation of pain is the sensation of spreading to the adjacent areas from the primary source which may suggest a neurogenic component to the problem.

**C- Characters of pain:** The descriptive terms chosen by the patient reflect his perception of pain, these could be sharp, dull, aching, burning, stabbing, throbbing, pulsating. The severity of pain can be graded as mild, moderate or severe based on its disruption of normal daily activities like sleeping, eating, working.
**D- Course of pain:** The course of pain often suggests possible causes. Steady increase in the severity of pain is typical of a progressive acute inflammation produced by a bacterial infection. Periods of relief followed by recurrences is a pattern of pain often caused by chronic periapical lesions that episodically undergo acute exacerbations.

**E- Factors that alter pain:** Alteration of pain following exposure to certain agents or conditions can reveal its nature and possible causes. Application of ice can soothe pain from most superficial inflammatory causes, and moist heat usually relieves the deeper discomfort of muscle spasm.

**F- Associated findings:** Certain systemic conditions can cause or influence the nature of pain, and a variety of drugs can accentuate pain perception. Emotional stress may exacerbate somatic pain or suggest psychogenic nature.

**Orofacial pain:**

**Orofacial Pain** is a complaint that around the world affects millions of people on a daily basis. It constitutes any symptom that occurs from a large number of disorders and diseases that result in a sensation of discomfort or pain felt in the region of the face, mouth, nose, ears, eyes, neck, and head. It is the presenting symptom of a broad spectrum of diseases. As a symptom, it may be due to disease of the orofacial structures, generalized musculoskeletal, peripheral or central nervous system disease, or psychological abnormality; or the pain may be referred from other sources (e.g., cervical muscles or intracranial pathology).

**Acute OFP:** is primarily associated with the teeth and their supporting structures. Most frequently, dental pain is due to dental caries, although a broken filling or tooth-abrasion may also cause dental sensitivity. Other oral pains are usually periodontal or gingival in origin.

**Chronic orofacial pain (COFP):** is a term used to describe painful regional syndromes with a chronic, unremitting pattern.

Clinically COFP may be subdivided into three main symptomatic classes

1. musculoskeletal
2. neuropathic
3. neurovascular
Musculoskeletal entities are dealt with Temporomandibular Disorders.

Possible causes of Facial Pain:
- Dental pain
- TMJ
- Neuropathic pain (neuralgias)
- Pathology in related str. (salivary gland, sinus, eyes, cervical spine, nasopharynx)
- Vascular disorder (headaches)
- Intracranial lesions (neoplasm, MS)
- Referred pain (angina pect.)
- Psychogenic facial pain.

Differential Diagnosis of Orofacial Pain
1- **Intracranial pain disorders** Neoplasm, aneurysm, abscess, hemorrhage, hematoma, edema.

2- **Primary headache disorders (neurovascular disorders)** Migraine, migraine variants, cluster headache, paroxysmal hemicrania, cranial arteritis, tension-type headache

3- **Neurogenic pain disorders** Paroxysmal neuralgias (trigeminal, glossopharyngeal, nervus intermedius), Continuous pain disorders (neuritis, post herpetic neuralgia, post-traumatic and postsurgical neuralgia)

4- **Intraoral pain disorders** Dental pulp, periodontium, mucogingival tissues, tongue.

5- **Temporomandibular disorders** Masticatory muscle, temporomandibular joint, associated structures

6- **Associated structures** Ears, eyes, nose, paranasal sinuses, throat, lymph nodes, salivary glands, neck

If the cause is intra-cranial; more than one division may be involved. And in advanced lesion there may be signs of elevation in the intra-cranial pressure (I.C.P). If the cause is intra-cerebral; then there may be neurological deficits to be demonstrated.
Clinical features of raised I.C.P.

- Headache.
- Impairment of conscious level.
- Papilloedema.
- Nausea, vomiting.
- Raised arterial pressure.
- Bradychardia.

Diagnostic Tests: Any test to select is guided by history & physical examination:

1. CT and/or MRI (to rule out intracranial pathology)
2. TMJ radiography
3. Diagnostic occlusal appliance
4. Cervical spine films
5. Labs (ESR)
6. Biopsy

CHRONIC OROFACIAL PAIN

1- musculoskeletal

2- Neuropathic Orofacial Pain

Neuropathic OFP includes a number of clinical entities; the most common are :- Trigeminal Neuralgia (TN), glossopharyngeal neuralgia (GN), geniculate neuralgia, painful posttraumatic neuropathies, burning mouth syndrome (BMS), facial post-herpetic neuropathy, central poststroke pain

3- Neurovascular Pain include:

   cluster headache (CH), migraine, paroxysmal hemicrania (PH), cranial arteritis, tension-type headache

Neuralgias

The classic neuralgias that affect the craniofacial region are a unique group of neurological disorders involving the cranial nerves and are characterized by

(a) Brief episodes of shooting

(b) Trigger zones on the skin or mucosa that precipitate painful attacks when touched
(c) pain-free periods between attacks and refractory periods immediately after an attack, during which a new episode cannot be triggered

Trigeminal Neuralgia:

It is sever recurrent shooting pain, sharp, stabbing or electrical lasting within seconds or minutes and provoked by talking, eating or touching specific areas called the "trigger zone", is an excruciating, short-lasting, unilateral facial pain.

It is characterized by sever paroxysmal pain in one or more branches of trigeminal nerve. Usually affecting the middle aged and elderly and often women are more affected than men. The most common sites involved are the mandibular mental area and the maxillary canine area. The ophthalmic distribution of the trigeminal nerve is rarely affected. There is a period of remission but the condition tends to recur or persist throughout the patient's life. The pain can be also an early manifestation of disseminated sclerosis. TN is characterized by spontaneous remissions lasting weeks to years but approximately 20% of TN patients suffer daily attacks. The most common is the

1- **classical** unrelated to pathology and most probably caused by neurovascular compression of the trigeminal nerve root.

2- **secondary** forms have been classified separately, and these are related to a variety of clear pathologies including tumors, cysts, viral infection, trauma, and systemic diseases such as multiple sclerosis.

The vast majority (>85%) of TN patients are diagnosed with classical TN (CTN).

Recent evidence suggests that most cases of CTN result from the compression of the trigeminal nerve root by a vascular malformation.

Recognized by the current classification are TN cases that present with a continuous background pain in addition to the typical pain paroxysms.

Up to one-third of patients describe typical paroxysmal attacks on a background of dull persistent pain of varying duration.

There are two attack-related phenomena that are particular to TN.

**Latency** refers to the short period of time between stimulation of a trigger area and pain onset.
A refractory period occurs following an attack and during this time pain may not be initiated.

Attacks begin and end abruptly, lasting from a fraction of a second up to 2 minutes. Longer attacks, increasing with disease duration, have been reported. Most paroxysms occur during waking hours, but may awaken the patient. Pain paroxysms are usually accompanied by spasm of the ipsilateral facial muscles (hence the name tic douloureux).

Etiology:

The etiology of neuralgia is unclear and 10% of cases have detectable underlying pathology such as:

1- Tumor of the cerebellar pontine angle,
2- Demyelinating plaque of multiple sclerosis
3- Vascular malformation.

The remainder of cases of TN is classified as idiopathic.

A majority of cases of TN are caused by an atherosclerotic blood vessel (usually the superior cerebellar artery) pressing on and grooving the root of the trigeminal nerve. This pressure results in focal demyelination and hyper excitability of nerve fibers, which will then fire in response to light touch, resulting in brief episodes of intense pain.

Pretrigeminal Neuralgia (PTN)

An early form of TN termed “pretrigeminal neuralgia” (PTN) has been reported in 18% of TN patients characterized by a dull continuous pain (days to years) in one of the jaws. As PTN progresses it becomes more typical with characteristic flashes of pain.

Thermal stimuli may cause triggering at a relatively higher rate, and a throbbing quality to PTN pain is sometimes present mimicking dental pathology. These qualities combined with the success of regional anesthesia have led to misdiagnosis of PTN as pain of dental origin.

PTN is however highly responsive to carbamazepine, and careful dental assessment should help differentiate it.
Diagnosis

The diagnosis of TN is usually based on the history of shooting pain along a branch of the trigeminal nerve, precipitated by touching a trigger zone, and possibly examination that demonstrates the shooting pain.

MRI of the brain is indicated to rule out tumors, multiple sclerosis, and vascular malformations.

Treatment

1- Anticonvulsant; Carbamazepine (Tegretol) remains the drug of choice for TN. Initial low-dose therapy (100 mg with food) and a slow increase (by 100–200 mg) on alternate days will minimize side effects. In responsive cases, therapeutic effects are observed rapidly or within three days. Titration to final dose (800-1200 mg/d) should continue slowly based on response and side effects. Light-headedness, confusion dizziness, vertigo, blurred vision or diplopia, sedation, vomiting, nystagmus, and nausea are very common and request drug cessation.

Main side effects:

a- Transient elevation in liver enzymes may occur

b- Transient leucopenia

c- Aplastic anemia is a serious effect that may occur.

d- Hyponatremia is observed in carbamazepine-treated cases and requires drug withdrawal.

e- Skin rashes occur in patients and may signal the onset of antiepileptic drug hypersensitivity syndrome. This is a life-threatening syndrome (fever, rash, and lymphadenopathy) associated with some antiepileptic drugs (AEDs) and requires immediate drug cessation.

Patients receiving carbamazepine must have periodic hematologic laboratory evaluations because serious life threatening blood dyscrasias occur. Monitoring of hepatic and renal function is also recommended.

Baclofen has a strong synergistic effect with carbamazepine, making it suitable for combined therapy. Newer anticonvulsants have fewer side effects and have been shown to be effective for some cases either as monotherapy or add-on therapy. Lamotrigine is effective particularly as add-on therapy, and gabapentin may be useful in selected TN cases.
2-Surgical: Surgery for TN is directed peripherally or centrally at the trigeminal ganglion or nerve root. Surgical procedures have a better prognosis when carried out on patients with typical CTN; has the best prognosis when performed within seven years of TN onset.

Peripheral Procedures

Peripheral neurectomy carries the danger of inducing traumatic neuropathic pain and is not recommended.

Cryotherapy of peripheral branches may give pain relief for six months. Pain recurrence is at the original site, repeated cryotherapy often produces better results.

Central Procedures

Percutaneous Trigeminal Rhizotomy

Microvascular decompression of the nerve root at the brainstem

Gamma Knife

Historically, alcohol injections have been used but are painful and cause fibrosis. Alcohol may induce herpes zoster (HZ) reactivation and bony necrosis. Pain control after alcohol block lasts just over one year, and there have been reports of post injection neuropathic pain.

Peripheral glycerol injection has been employed, but success seems short term.

Glossopharyngeal neuralgia (GN):

The location of the trigger zone and pain sensation follows the Distribution of the glossopharyngeal nerve, namely, the pharynx, posterior tongue, ear, and intra auricular, retromandibular area. Although similarities with TN are prominent, GN is characterized by a milder natural history with the majority of patients going into remission. Due to its location and features, GN is a difficult diagnosis and adequate treatment is often delayed for years.

Pain is triggered by stimulating the pharyngeal mucosa during chewing, talking, and swallowing. The application of topical anesthetic to the pharyngeal mucosa eliminates glossopharyngeal nerve pain and can aid in distinguishing it from the pain of other neuralgias.

The most common causes of glossopharyngeal neuralgia are intracranial or extra cranial tumors and vascular abnormalities that compress CN IX.
Features

The glossopharyngeal (IX) nerve has two main sensory branches: the aricular (tympanic) and the pharyngeal.

In pharyngeal-GN, the pharynx or posterior tongue-base are involved. Pain radiates to the inner ear or the angle of the mandible, and may include the eye, nose, maxilla, or shoulder and even the tip of the tongue.

In tympanic- GN, pain predominates in the ear but may radiate to the pharynx.

Bilateral pain occurs in up to a quarter of patients. GN is a paroxysmal, unilateral, severe pain that is sharp, stabbing, shooting, or lancinating. Patients often feel a scratching or foreign body sensation in the throat. Pain intensity is usually milder than TN but may vary and attacks last from a fraction of a second up to 2 minutes.

Trigger areas are located in the tonsillar region and posterior pharynx, and these display a refractory period. Swallowing, chewing, talking, coughing and/or yawning, sneezing, clearing the throat, and rubbing the ear activate these areas. Frequency is around 5–12 every hour, and attacks may occur in clusters lasting weeks to months, then relapse for up to a number of years.

Spontaneous remissions occur in the majority of patients, but some have no periods of pain relief.

GN may induce uncontrollable coughing, seizures, and cardiac arrhythmias, particularly bradycardia, and syncope. TN and GN patients should undergo imaging (computerized tomography [CT] or magnetic resonance imaging [MRI]) at least once during diagnosis and therapy.

Imaging techniques such as magnetic resonance tomographic angiography (MRTA) may more accurately identify neurovascular compression. Imaging of the head and neck to rule out pathology is indicated

Pathologies Mimicking GN

1- A significant association between symptomatic GN and multiple sclerosis has been reported

2- Regional diseases such as infectious or inflammatory processes

3- tonsillar carcinoma

4- other regional tumors (tongue, oropharyngeal)

5- Cerebello pontine angle or pontine lesions
Pathophysiology of GN

The pathophysiology is uncertain but is considered to probably be secondary to compression of the nerve root by a blood vessel.

GN cases demonstrate nerve compression on MRI and on surgical exposure, and nerve biopsy shows variable myelin damage and patches of demyelinated axons in close membrane-to-membrane apposition to one another. These morphological changes are similar to those observed in patients with TN suggesting shared pathophysiology.

Treatment

Carbamazepine is usually successful and is the favored medication. Alternatives include baclofen (muscle relaxant), oxcarbazepine, gabapentin, lamotrigine, and phenytoin.

Permanent neurological deficits are rare and may include mild hoarseness and/or dysphagia, or facial nerve paresis

Facial Pain Associated With Herpes Zoster

Post herpetic neuralgia:

Acute Herpes Zoster

Acute HZ (shingles) is a reactivation of latent varicella virus infection that may occur decades after the primary infection. HZ is a disease of the dorsal root ganglion and therefore induces a dermatomal vesicular eruption. Definitive diagnosis may be obtained by identification of viral DNA from vesicular fluid employing the polymerase chain reaction. Trigeminal and cervical nerves are involved in up to a quarter of cases. The ophthalmic branch is affected in more than 80% of the trigeminal cases, particularly in elderly males, and may cause sight-threatening keratitis. The vesicles and pain are dermatomal and unilateral and may appear intraorally when the maxillary or mandibular branches of the trigeminal nerve are affected.

Etiology and Pathogenesis

Herpes zoster (shingles) is caused by the reactivation of latent varicella-zoster virus infection that results in both pain and vesicular lesions along the course of the affected nerve.
In a majority of cases, the pain of herpes zoster resolves within a month after the lesions heal. Pain that persists longer than a month is classified as post herpetic neuralgia (PHN) although some authors do not make the diagnosis of PHN until the pain has persisted for longer than 3 or even 6 months.

**Clinical Features**

Usually begins with a prodrome of regional pain, itching and malaise. Pain precedes typical vesicular eruption by <7 days, usually 2–3 days. The dermatomal vesicular or herpetic eruption will rupture and “dry out” over 7–10 days, but complete healing may last up to one month. Accompanying pain is moderate to severe (VAS 6) and may persist for three to six months. Very rarely dermatomal pain occurs with no rash.

**Treatment**

Therapy is directed at controlling pain, accelerating healing, and reducing the risk of complications such as meningitis, post herpetic neuropathy (PHN), and local secondary infection. Antivirals should be initiated within 72 hours from onset of rash, and will significantly decrease rash duration, pain severity, and the incidence of PHN. This is particularly effective in patients >50 years old.

Fever and pain should be controlled initially by mild analgesics; central analgesics may be used (amitriptyline or gabapentin). Use of glucocorticoids is controversial, but may help reduce acute pain; they should always be used together with antivirals. Amitriptyline may reduce the incidence of PHN. Vaccinating at risk individuals markedly reduces the incidence of PHN among older adults.

**PHN**

Up to one-fifth of acute HZ patients will suffer persistent pain three to six months after acute HZ. By one year however only 5%–10% suffer pain. Advanced age (>50 year), severe prodromal pain (VAS>5), severe acute pain, and severe rash are risk factors for persistent pain. In patients older than 60 years, 50% or more will continue to suffer pain for more than one year.

**Features of PHN**

PHN is a dermatomal disease persisting or recurring ≥3 months after the acute HZ stage. Patients relate a previous herpetic (dermatomal) eruption that was preceded by pain usually two to three days but up to six days prior. PHN is characterized by fluctuations from moderate Back ground pain to excruciating, superimposed lancinating pains. Pain quality is burning, throbbing, stabbing, shooting, or sharp. Burning pain is significantly higher in patients not treated with antivirals for acute HZ. Itching is very common and prominent in trigeminal dermatomes and may be subjectively graded as worse than pain. Pale, sometimes red/purple, scars that are usually hypoesthetic or anesthetic (but with allodynia and hyperalgesia) may remain in the affected area. Patients with PHN experience...
persistent pain, paresthesia, hyperesthesia, and allodynia, months to years after the zoster lesions have healed.

Treatment
Early treatment of established PHN improves prognosis. Ophthalmic PHN per se seems to have the worst prognosis. Evidence-based treatment options for PHN include tricyclic antidepressant (TCA) drugs, gabapentin and pregabalin, tramadol, and topical lidocaine patches.
Invasive therapies include epidural and intrathecal steroids and a variety of neurosurgical techniques. Central nervous system stimulation may also provide some relief.
The best therapy is prevention. Use of antiviral famciclovir 500 mg 3 times daily for 7-10 days or Acyclovir 800mg 5 times 7-10 days.

Short course of systemic corticosteroid during the active phase of the disease. Topical therapy includes the use of topical anesthetic agents, such as lidocaine, or analgesics.
The use of tricyclic antidepressants such as triptyline, nortriptyline, is a well method of reducing the chronic burning pain that is characteristic of PHN.

NERVOUS INTERMEDIUS (GENICULATE) NEURALGIA

Nervous intermedius (geniculate) neuralgia is an uncommon paroxysmal neuralgia of CN VII resulting from herpetic inflammation of geniculate ganglion and nervous intermedius of CN VII characterized by pain in the ear and (less frequently) the anterior tongue or soft palate.
The location of pain matches the sensory distribution of this nerve (i.e., the external auditory canal and a small area on the soft palate and the posterior auricular region). Pain may be provoked by the stimulation of trigger zones within the ipsilateral distribution of the nerve.
The pain is not as sharp or intense as in TN, and there is often some degree of facial paralysis, indicating the simultaneous involvement of the motor root.

Geniculate neuralgia commonly results from herpes zoster of the geniculate ganglion and nervus intermedius of CN VII, a condition referred to as Ramsay Hunt syndrome. Viral vesicles may be observed in the ear canal or on the tympanic membrane. The symptoms result from inflammatory neural degeneration.
Ramsay Hunt syndrome is defined as an acute peripheral facial neuropathy associated with erythematous vesicular rash of the skin of the ear canal, auricle and/or mucous membrane of the oropharynx.

**Treatment**

1- Short course (2 to 3 weeks of high-dose steroid therapy is beneficial.

2- Acyclovir significantly reduces the duration of the pain 200mg 5 times daily for 10-14 days.

3- Patients with geniculate neuralgia are also treated with carbamazepine and antidepressants.

Patients who do not respond to these medications may undergo surgery to section the nervus intermedius.

**Burning Mouth Syndrome (BMS)**

Is a poorly understood pain condition that is most probably neuropathic. The condition is also known as stomatodynia and is characterized by a burning mucosal pain with no significant physical signs and is common in postmenopausal women.

**BMS may be subclassified into:-**

1- “primary” or idiopathic BMS for which a neuropathological cause is likely and cannot be attributed to any systemic or local cause

2-“secondary BMS”(SBMS) resulting from local or systemic pathological conditions.

BMS is characterized by resistance to a wide range of treatments and is one of the most challenging management problems in the field of OFP.

**Clinical Features**

The primary location of the burning complaint is the tongue, usually the anterior 2/3. However, usually more than one site is involved and in addition to the tongue, hard palate, lips, and gingivae are frequently involved. Pain is most commonly described as burning or hot and intensity varies from mild to severe. BMS is typically of spontaneous onset and lasts from months to several years. Pain pattern may be irregular, but some patients may complain that pain increases toward the end of the day.
Although a chronic unremitting pattern is usual, partial remission has been reported in about one half to two-thirds of patients, six to seven years after onset. Spontaneous remission is very rare.

Common aggravating factors include personal stressors, fatigue, and specific foods (acidic, hot, or spicy). More than two-thirds of the patients complain of altered taste sensation (dysgeusia) accompanying the burning sensation, in many cases described as a spontaneous metallic taste. Abnormal sensations, such as feeling of dry mouth, are common but true hyposalivation is less common and should be considered under secondary or symptomatic BMS.

Oral and perioral burning sensation as a result of local or systemic factors or diseases is classified as SBMS.

1. Local factors and diseases known to induce SBMS include oral candidiasis, lichen planus, and allergies.

2. Systemic disorders that induce SBMS include hormonal changes, deficiencies of vitamin B12, folic acid or iron, diabetes mellitus, side effects of medications, and autoimmune diseases.

Successful treatment of the primary disease will usually alleviate the burning sensation in SBMS patients.

Treatment

Topical therapies may be effective and are useful in elderly, medically compromised patients. The most established is clonazepam (tranquilizers) (1 mg) which should be sucked and subsequently spat out three times daily. Topical anesthetics may decrease or increase pain and are therefore unpredictable.

Systemic therapies include paroxetine (antidepressant) (20 mg/d) and sertraline (50 mg/d) or other selective serotonin reuptake inhibitors (SSRIs). These may reduce pain and improve anxiety and depression.

A two-month course of 600 mg daily of alpha-lipoic acid may be beneficial. A combination of alpha-lipoic acid (600 mg/d) and gabapentin (300 mg/d) results in greater improvement of the burning symptoms compared to these medications taken alone.
Pharmacotherapy-resistant BMS has been associated with underlying psychological distress, and these patients may particularly benefit from cognitive behavioral therapy.

**Painful Posttraumatic Trigeminal Neuropathy (PTTN)**

Some patients develop chronic pain following negligible nerve trauma such as root canal therapy or following considerable injury to nerve bundles, such as in fractures of the facial skeleton. Following dental implant surgery 1%–8% and following orthognathic jaw surgery 5%–30% of patients may remain with permanent sensory dysfunction but the incidence of chronic pain is unclear. Third molar extractions may lead to disturbed sensation in the lingual or inferior alveolar nerve for varying periods. Patient complaints of tongue dysesthesia after injury may remain in a small group of patients (0.5%). Persistent pain after successful root canal therapy may occur, also surgical root therapy may resulted in chronic neuropathic pain.

**Features**

Chronic pain issues are possible risk factors for the Features Following identical injuries, onset of neuropathic pain and its characteristics vary from patient to patient. Such variability is probably due to a combination of environmental, psychosocial, and genetic factors.

The presence and duration of pain in the tooth, tenderness to percussion, female gender, previous painful treatment in the orofacial region, and concomitant chronic pain issues are possible risk factors for the development of chronic pain following successful root canal therapy.

Pain is unilateral and occurs in the area of injury, or at the distal dermatome of an injured nerve. Initially pain may be precisely located to the dermatome of the affected nerve, but it may become diffuse and spread across dermatomes. Pain is of moderate-to-severe intensity (VAS 5–9), usually burning in quality but also stabbing during exacerbations.

Positive or negative local neurological signs include clinically demonstrable sensory dysfunction, usually allodynia, hyperalgesia, or parasthesia.

Most cases are continuous, but some report superimposed paroxysmal pain attacks.

Less frequently there may be short-lasting pain with associated mechanical trigger areas, mimicking TN. Rarely, a subjective feeling of swelling, foreign body, hot or cold, local redness or flushing may be reported but these may not always be clinically verifiable.
Treatment

Topical
Topical anesthetics may be successfully employed in the management of painful neuropathies. Some benefits have been observed using topical capsaicin (active component of chili peppers) in patients with oral neuropathic pain. Topical medication as single treatment or in combination with systemic medications can reduce the severity of orofacial neuropathic pain.

Systemic Pharmacotherapy
Available data confirm that antiepileptics AEDs and tricyclic antidepressants TCAs are most effective. For many of the drugs used in the therapy of traumatic neuropathies, response is dose dependent and subsequently accompanied by significant side effects.

Therapy of neuropathic pain with any one of the established drug groups (Antidepressants, anticonvulsants) leads to improved quality of life, sleep, and mood. However, pain intensity is reduced in only a subset of responders and is usually accompanied by significant side effects, particularly at the higher doses often required in neuropathic pain.