

Toothache of Non-Dental Origin: A Review of Its Mechanism and Clinical Characteristics

Abhishek Soni

Senior Lecturer, Department of Oral Medicine & Radiology, Modern Dental College and Research Center, Indore, Madhya Pradesh, India

Abstract

Objective: To review the clinical presentations of the various types of non-odontogenic pains which may be mistaken as dental pain in clinical practice.

Materials and Methods: A search was initiated on web using PubMed/Medline database searching for articles written in English. Peer-reviewed articles were chosen using the key terms "Orofacial pain," and "Non-odontogenic toothache." Available full-text articles published in relevant journals were read, and related articles were scrutinized and finally the search was subsequently refined to articles concerning to "Non-odontogenic toothache."

Results: Non-odontogenic toothaches are frequently encountered in clinical practice and its diagnosis can be challenging to the dental clinician. For appropriate diagnosis, the clinician should be well aware of various causes of the non-odontogenic toothache and be able to differentiate them.

Conclusion: In conclusion, for precise and correct diagnosis of non-odontogenic toothache, understanding of the nature of pain and its specific clinical characteristics is recommended. Knowledge of the various presentations of non-odontogenic pains will ultimately prevent the misdiagnosis and the institution of incorrect and sometimes irreversible treatment to the patients.

Key words: Heterotopic pain, Non-odontogenic toothache, Orofacial pain, Referred pain

INTRODUCTION

Chronic orofacial and head pain are a common clinical problem, and appropriate diagnosis and management are a challenge for health-care professionals. Patients often first seek the care of dentists because of pain localization in the oral cavity and surrounding structures. Most of the toothaches are originated from specific pulpal or adjacent periodontal tissues. The orofacial pain from dental origin was specifically called "odontogenic toothache."^[1] However, some toothaches may non-dental origin. Toothache of non-dental origin is not true dental pathology; rather it is the pain referred to the dentition from distant location.^[2]

The term "non-odontogenic toothache" defined as the pain which is perceived on tooth and adjacent structure but is

not originated from the pulpal and periodontal tissues.^[1] Non-odontogenic toothache is the type of heterotopic pain.^[3] In clinical practice, it is often common for pain in the orofacial region to be mistaken for a toothache, as they mimic odontogenic pain. Therefore, orofacial pain may sometime pose a diagnostic dilemma for the oral physicians and clinicians. Understanding the complex mechanism of odontogenic and non-odontogenic pain and the manner in which other orofacial structures may simulate pain in the tooth is of paramount importance in determining the correct diagnosis and appropriate treatment.

The aim of this article is to provide a brief overview of the various presentations of the non-odontogenic pain which may be mistaken for a toothache with an understanding of mechanism of pain referral and the specific clinical characteristics that have been consider when developing differential diagnoses for pain affecting the orofacial region.

METHODS

To get up-to-date information, a web-based search was initiated by the author using PubMed/Medline database

Access this article online



www.ijss-sn.com

Month of Submission : 03-2018

Month of Peer Review : 04-2018

Month of Acceptance : 04-2018

Month of Publishing : 05-2018

Corresponding Author: Dr. Abhishek Soni, 263-Balaji Villa, Shivom Estate, Station Road, Dewas - 455 001, Madhya Pradesh, India.
Phone: +91-9827511672/9340477983. Tel.: 07272-224403. E-mail: drabhishek_soni@rediffmail.com

searching for articles written in English. Peer-reviewed articles were targeted using the key terms “Orofacial pain,” and “Non-odontogenic toothache” to determine the scope of coverage in well-documented articles. The search was subsequently refined to articles concerning to “Non-odontogenic toothache.” The sites of specialized scientific journals in the areas of oral and facial pain were also assessed. The available full-text articles published in relevant journals such as journal of orofacial pain, journal of oral medicine, and surgery were read, and related articles were scrutinized. The bibliographies were also reviewed to identify additional relevant studies.

Non-odontogenic Toothache

Odontogenic pains are usually inflammatory in origin and arise from either two tissues: The pulp and the periodontal supportive structures. These are considered to be the musculoskeletal type of pain.^[4] But sometimes, orofacial pain that is perceived as toothache does not always originate from the dental structures; therefore to provide accurate diagnosis it is important to distinguish between site and source of pain [Figure 1]. The site of pain is the location where the patient feels the pain, and it is easily located by asking the patient to point out the region of the body that is painful; whereas the source of pain is the structure of the body from where the pain actually originates.^[5]

Primary pain and heterotopic pain

When the site and source of pain are in the same location, it is described as “primary” pain, i.e., the pain occurs where damage to the structure has occurred. On the contrary, when the site and source of pain are different, it is described as “heterotopic” (or “referred”) pain. It is thought to be related to central effects of constant nociceptive input from deep structures such as muscles, joints, and ligament.^[5] Once diagnosed, treatment should be posed at the source of pain, rather than the site. Although the terms heterotopic pain and referred pain are often used interchangeably, there are specific distinctions between these terms. Heterotopic pain can be divided more specifically into three types, namely central pain, projected pain, and referred pain [Table 1].^[3]

Clinical characteristics

The following four clinical characteristics of non-odontogenic toothache (heterotopic or referred pain) help in differentiate it from an odontogenic toothache (primary pain):^[6]

1. Local provocation of the site of pain does not increase the pain.
2. Local provocation at the source of pain increases the pain not only at the source but also increases the pain at the site.
3. Local anesthetic blocking of the site of pain does not decrease the pain.

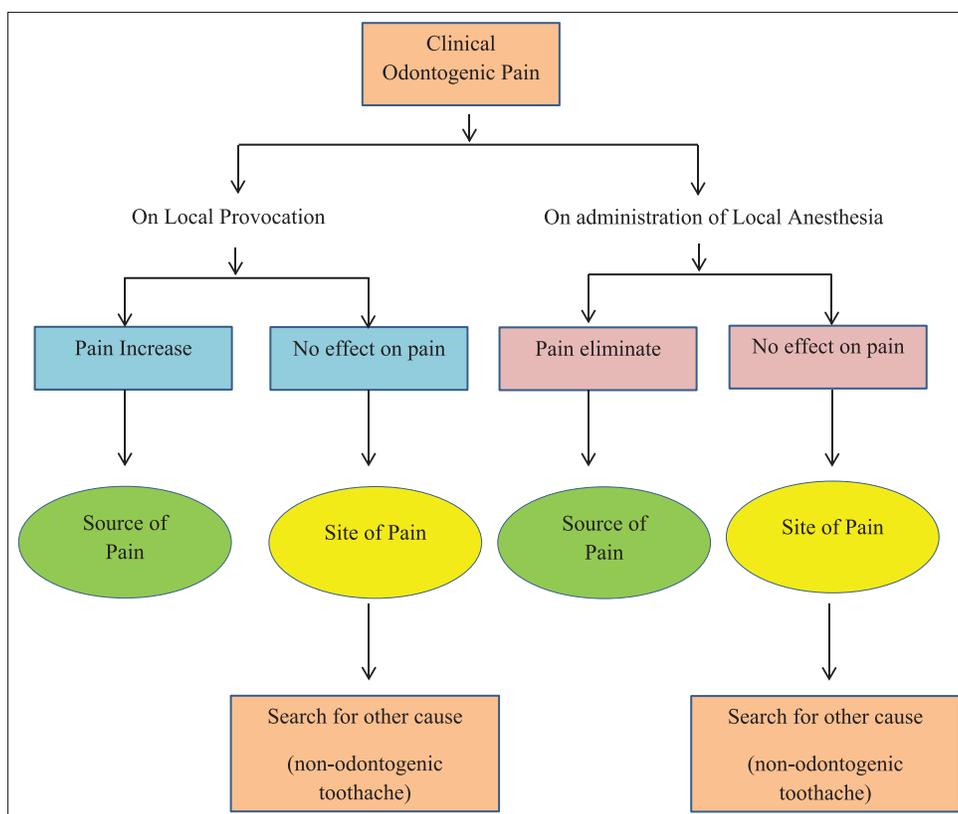


Figure 1: Algorithm to differentiate an odontogenic toothache from non-odontogenic toothache. Therapy should always be directed to treat the source of pain rather than the site of pain

Table 1: Types of heterotopic pain**Central pain:**

Pain derived from the CNS

Pain perceived peripherally

E.g., an intracranial tumor - this will not usually cause pain in the CNS because of the brain's insensitivity to pain, but rather it is felt peripherally.

Projected pain

Pain perceived in the peripheral distribution of the same nerve that mediated the primary nociceptive input

E.g., pain felt in the dermatomal distribution in postherpetic neuralgia.

Referred pain

Spontaneous heterotopic pain felt at a site of pain with separate innervation to the primary source of pain

Mediated by sensitization of interneurons located within the CNS

E.g., pain referred from the sternocleidomastoid muscle to the temporomandibular joint.

CNS: Central nervous system

4. Local anesthetic blocking of the source of the pain decreases the pain at the source, as well as the site.

Types of non-odontogenic toothache

The various types of non-odontogenic pains which may be mistaken as dental pain includes:^[5,7,8]

1. Myofascial toothache
2. Neurovascular toothache
3. Cardiac toothache
4. Neuropathic toothache
5. Sinus toothache
6. Psychogenic toothache.

Myofascial Toothache

The myofascial toothache is a non-pulsatile and aching pain and occurs more continuously than pulpal pain. In such type of pain, patients are often unable to precisely locate the source of the pain and often consider that pain is originating from the tooth.^[4]

Mechanism of pain referral

The theory of convergence supports the mechanism that is thought to cause pain referral to the trigeminal sensory complex from other areas of nociceptive input although it is not well understood. It has been reported that at least half of the trigeminal nociceptive neurons are able to be activated by stimulation outside their normal receptive field.^[5,9]

It is evident from the studies on myofascial pain referral to other regions of the orofacial region that pain is triggered by palpating the strained muscles (source of pain) which may perceive as toothache involving any region of oral cavity and in and around the surrounding structures (site of pain) [Table 2].^[8,10-12]

Clinical characteristics

The clinical characteristics of the toothache of myofascial origin are as follows:^[4,8]

1. Pain is dull aching, non-pulsatile, and typically more constantly aching than that of pulpal pain.

Table 2: Myofascial pain referral: Trigger points in the involved muscle and referred site in the oral cavity

Source muscle	Referred site in oral cavity
Superior belly of the masseter	Maxillary posterior teeth
Inferior belly of the masseter	Mandibular posterior teeth
Temporalis muscles	Maxillary anterior or posterior teeth
Lateral pterygoid muscles	Maxillary sinus region and TMJ
Anterior digastric muscles	Mandibular anterior teeth
Sternocleidomastoid muscles	Oral structures and the forehead
Trapezius muscle	Mandible or temporalis muscle regions

TMJ: Temporomandibular joint

2. There is lack of dental pathology to explain the pain.
3. Pain is not increased by local provocation of the tooth.
4. Pain is increased with the function of involved muscle (trigger point). Pain is increased with extended use of involved muscle or by palpating the affected muscles, and may have tendency to exacerbate with emotional stress.
5. Tooth sensitivity to temperature, percussion, or occlusal pressure may be felt as a result of referred pain from the offending muscle.
6. Local anesthetic of the tooth does not decrease the toothache.
7. Local anesthetic of the involved muscle decreases the toothache.

It has been reported that 37% of patients diagnosed with muscular orofacial pain had previously undergone endodontic or exodontic treatment in an attempt to alleviate their pain.^[13,14] Ehrmann^[15] also reported that 7% of cases were referred for endodontic treatment when the primary source of pain was the muscle of mastication.

Treatment involves elimination of the trigger points found in the involved muscles. Warm or cold compresses, muscle

stretching, massage, and a restful sleep may alleviate both the muscle and tooth pain.^[5]

Cardiac Toothache

Cardiac pain (cardiac ischemia) more commonly presents with substernal pain and radiation of pain to the left shoulder and arm.^[4] It is considered to be the source of referred pain involving the jaw. When pain present is of cardiac origin, most commonly affected areas in the orofacial region are neck, throat, ear, teeth, and mandible.^[16-21]

In patients suffering from cardiac ischemia, sometimes, orofacial pain may be the only complaint. In one study, 6% of patients presenting with coronary symptoms had pain solely in the orofacial region while 32% had pain referred elsewhere. Interestingly, bilateral referred craniofacial pain was noted more commonly than unilateral pain at a ratio of 6:1.^[17]

Mechanism of cardiac referral

- The cause of the cardiac pain referred to the orofacial region can be explained by the convergent mechanisms of the trigeminal complex.^[21] Cardiac afferents and somatic inputs from the upper limbs, chests, and face converge on spinothalamic tract neurons in the central nervous system. This convergence input leads to pain nociceptive input from visceral structures, such as the heart through the spinal cord to the trigeminal region. The information is then projected to the thalamus. Convergence mechanisms into the trigeminal brainstem complex and/or in the thalamus can explain referred pain to the face.
- There may be a physiologic association between vagal stimulation initiated by cardiac ischemia and odontogenic pain.^[5] Based on the anatomic distribution, when the inferoposterior surface of the heart is affected vagal afferent is activated; and stimulation of the anterior portion results in sympathetic response.^[22]
- Another possible mechanism of cardiac pain involves multiple nociceptive mediators which induce a sympathetic response of the heart^[23-25] by evoking a sympathoexcitatory reflex.^[26] The most important nociceptive mediator being the bradykinin. Studies on patients who underwent sympathectomies demonstrated a 50–60% complete relief of angina pectoris, while 40% obtained a partial relief, and 10–20% experienced no relief.^[22]

Clinical characteristics

The clinical characteristics of the toothache of cardiac origin are as follows:^[5,27]

1. The presence of aching pain in the jaw or tooth is cyclic.

2. Pain may be episodic, lasting from minutes to hours, and varies in intensity.
3. The toothache is increased with physical exertion or exercise.
4. The toothache is alleviated with rest.
5. The toothache is associated with chest, arm, or neck pain.
6. The toothache is decreased with nitroglycerin tablets.
7. Local provocation of the tooth does not alter the pain.

Intriguingly, patients experiencing cardiac pain reported the descriptor of “pressure” more often when compared to any other disorder.^[28]

Orofacial pain of cardiac origin is most often relieved by giving sublingual nitroglycerin tablets. An immediate referral to a physician or cardiologist is mandatory.

Sinus Toothache

Sinusitis is a common disease, of which maxillary sinusitis is more prevalent. About 10% of maxillary sinusitis cases are diagnosed as having pain of odontogenic origin.^[11] It has been characterized by constant dull aching pain in and around the zygoma and tenderness of the teeth on percussion due to inflammation of the maxillary sinus. Acute sinusitis can induce referred pain to maxillary teeth particularly maxillary premolar and molar regions because of closeness of the apices of the teeth to the sinus region.^[1] According to a study of the symptoms of acute sinusitis, maxillary toothache was highly specific (93%), but only 11% of patients with sinusitis actually had pain from the tooth.^[29]

Mechanism of pain referral

Due to the proximity of the roots of maxillary teeth with the sinus, it is conceivable that the maxillary dentition could be a potential source of inflammation and infection of maxillary sinus. The final point of growth of maxillary sinus is fortuitous with the growth of the maxillary alveolar process and eruption of the permanent dentition, resulting in a protrusion of roots into the maxillary sinus cavity. Sometimes, the roots may be separated only by the Schneiderian membrane. Since the roots of the maxillary dentition are in intimate contact with maxillary sinus, any infectious process associated with the maxillary dentition or surrounding periodontal tissue may present as acute or chronic sinusitis; conversely, any inflammatory or infectious disease originating in the maxillary sinus may be anticipated as odontogenic pain.^[5]

The sensory innervation of sinus mucosa and maxillary teeth could be responsible for the sinus pain referral. Sensory innervations of the nasal-PNS complex are supplied by the first and second divisions of the trigeminal nerve and secondary interneurons from sinus area shares

with those of teeth. The pain from the sinus complex is typical deep visceral pain, and it can cause central sensitization such as secondary hyperalgesia, referred pain, and autonomic response. In the early stage of sinusitis, facial pain and headache are common. Hyperalgesia on the affected region by central sensitization make the pain more chronic and more complex.^[1]

Clinical characteristics

The clinical characteristics of the toothache of maxillary sinus origin are as follows:^[10,30-33]

1. Dull, constant aching pain is present in several maxillary posterior teeth in one quadrant, i.e., tooth sensitive to percussion.
2. The patient reports pressure or pain below the eyes.
3. The toothache is increased with lowering of the head because of shifting of fluid in the sinus due to the gravitational effect.
4. Pain is experienced with palpation over the involved sinus or infraorbital regions.
5. There is a history of sinusitis or upper respiratory infection which may precede the onset of the toothache.
6. Toothache is increased by stepping hard on to the heel of the foot (e.g., walking down the steps).
7. The diagnosis can be confirmed by air-fluid level seen in appropriate imaging.

A simple maxillary sinus infection may be treated with a 10-day course of amoxicillin and 2- or 3-day use of a topical decongestant. A referral to an otolaryngologist may be indicated if the sinus infection is unresponsive to this therapy.

Rhinogenic Toothache

Non-odontogenic toothache of nasal mucosa origin is a related painful condition affecting the maxillary anterior teeth. This can occur if the nasal mucosa becomes edematous causing swelling of the turbinate and occluding outflow from the maxillary sinus ostium.

This referral pattern has been demonstrated experimentally. Anesthetic blocking by infiltration at the apex of the tooth in question does not completely arrest the toothache; however, the pain may be decreased by applying topical anesthetic to the area of the ostium with a cotton-tipped applicator or spray. If the nasal mucosa is the source of the pain, the toothache should be relieved [Figure 2].^[30]

Neuropathic Toothache

Neuropathic pain can be described as a pain originated from abnormalities in the neural structures and not from the tissues that are innervated by those neural structures.

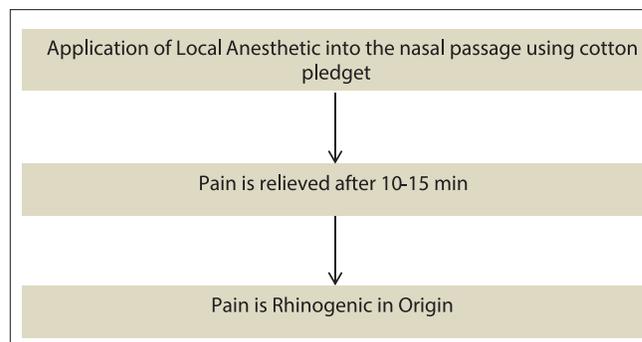


Figure 2: Diagnostic technique to locate pain of nasal mucosa origin

There are two types of neuropathic pains that can be felt in teeth: Episodic and continuous [Figure 3].^[5,34]

Mechanism of pain referral

A number of mechanisms have been suggested for the causation of neuropathic pain in the orofacial region.

Change in excitability of primary nociceptive afferents may be the single most important factor in generation and maintenance of acute chemogenic pain or chronic neuropathic pain in humans.^[35] Demyelination is a degenerative process that is associated with loss of integrity of the myelin sheath that normally protects nerve fibers. This may result in an aberration in nerve impulse generation and conduction. Demyelination can occur peripherally or centrally. Multiple sclerosis is the most well-known example of central demyelinating disease. When the disease affects the trigeminal ganglion, it can present as trigeminal neuralgia.

Neuropathic pain is due to abnormality in components of the nervous system itself rather than to noxious stimulation of otherwise normal neural structures. According to Robinson,^[36] it has been shown that these pathologic entities can cause ectopic discharge or impulse generation from the sites along the axon where the damage has occurred, rather than just at the sensory nerve ending. Recent evidence revealed that there is a result of membrane hyperexcitability along the axon. Studies have recently demonstrated that membrane remodeling, particularly involving Na⁺ channels, is responsible for the ectopic repetitive firing. There are three primary ways in which sodium channels affect a change in membrane hyperexcitability and repetitive firing in damaged axons. First, there is a change in the rate of protein synthesis of various Na⁺ channels as a result of the neuronal injury. More Na⁺ channels mean more sensitivity. The elevated rate of synthesis of these proteins occurs concurrently with axonal ectopic firing and the initiation of allodynia. Second, there is an intracellular regulation of the Na⁺ channels that allow the channels to remain open longer and create more hypersensitivity and even spontaneous

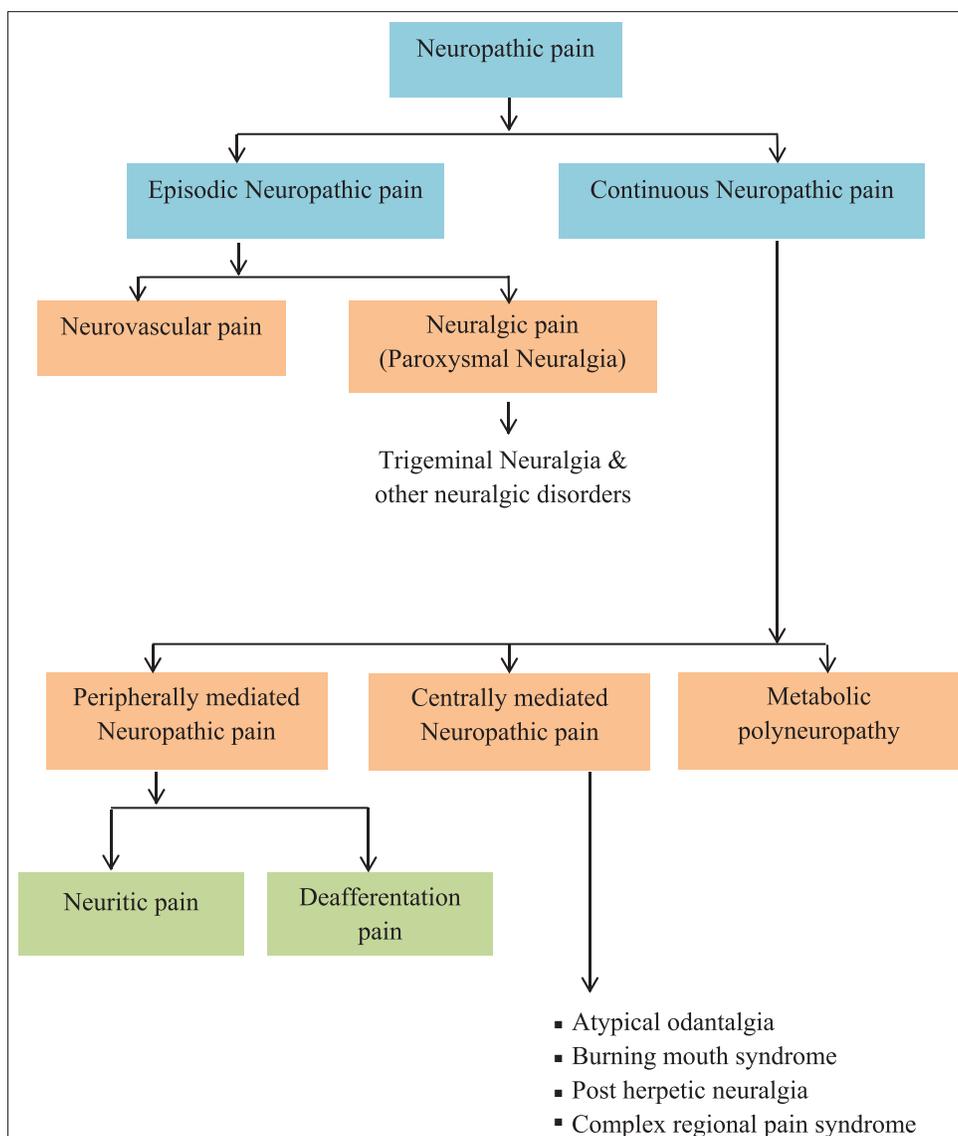


Figure 3: Classification of neuropathic pain (adapted from: Okeson JP. pains of dental origin. In: Okeson JP. Bell's Orofacial Pains: The clinical management of orofacial pain. 6th ed. Chicago: quintessence; 2005; p. 260)

firing of damaged neurons. The third way involves the interruption of axonal transport. If an axon is transected, exposed to certain toxins, or undergoes demyelination, then the axonal transport system responsible for moving Na⁺ channel proteins from the cell nucleus to the axon sensory nerve ending is disrupted. Once damage occurs, may result in the formation of neuroma.^[36]

Episodic neuropathic toothache

Episodic neuropathic pain is characterized by sudden volleys of electric-like pain referred to as neuralgia. The most typical example of this type of pain is trigeminal neuralgia.^[5]

The clinical presentation of an episodic neuropathic toothache involves:^[5,34]

- Pain quality is described as severe, sharp-shooting, and electric-like pain that lasts only a few seconds.

- There are pain-free intervals between the episodes of pain
- Pain is provoked by peripheral stimulation of a “trigger zone.”
- The pain is not always restricted to a tooth but often a broader area.
- The pain is not altered by intraoral thermal stimuli.
- It rarely awakes the patient from sleep, unlike dental pain.
- Often the patient is able to trace the pain radiating down the distribution of the nerve to the tooth.

Continuous neuropathic toothache

Continuous neuropathic pains are pain disorders that appear to have its origin in neural structures and are expressed as constant, ongoing, and unremitting pain. They will often have high- and low-intensity but no periods

of total remission. Patients with continuous neuropathic toothache often report a history of trauma or ineffective dental treatment in the area.^[37]

It is not unusual for patients with continuous neuropathic toothache to have received multiple endodontic treatments or extractions for their dental pain. In many cases, the lack of response to treatment is a key factor in prompting a reassessment of the differential diagnosis.^[38] Ram *et al.*^[39] in their retrospective study involving 64 patients reported that 71% had initially consulted a dentist for their pain complaint, and subsequently 79% of patients received dental treatment that did not resolve the pain. In one case report, the lack of an effect of a local anesthetic injection on reducing the intensity of pain was a significant finding that prompted consideration of non-odontogenic tooth pain.^[40]

In a study of 42 patients with atypical odontalgia, 86% of the patient population was female, and 78% reported maxillary pain. Of 119 reported areas of pain, the most common was the molar (59%), premolar (27%), and canine (4%) regions.^[41,42] The pain may change in location over time; some studies have reported pain shifting location in up to 82% of the subjects.^[43,44] A key to diagnose this is to recognize accompanying neurologic signs that involve other teeth or nearby structures served by the same nerve.^[34]

The common types of neuropathic conditions that can produce continuous pain felt in a tooth are neuritic pain and deafferentation pain [Figure 4].^[34]

The clinical presentation of a neuritic toothache involves:

- Pain is persistent, non-pulsatile, often burning pain felt in a tooth.
- Toothache accompanied by other neurologic symptoms (paresthesia, dysesthesia, and anesthesia)
- Other teeth may feel “dead” or “strange.”
- Associated gingival tissue may get affected.

The clinical presentation of a deafferentation toothache involves:

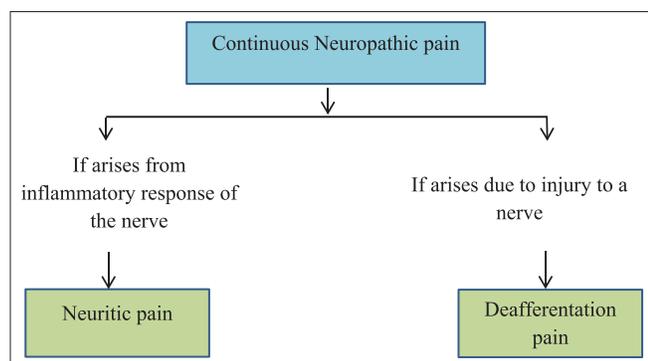


Figure 4: Types of continuous neuropathic pain

- Toothache is continuous, varies in intensity, with no periods of remission.
- Maxillary canines and the premolars are most commonly involved teeth.
- Condition is most commonly reported in middle-aged women with a history of trauma to the painful region.
- Pain is not changed by local provocation.
- Effect of local anesthesia is unpredictable.
- Toothache not responsive to dental therapies.

Continuous neuropathic pains that can be felt in teeth have been referred to as atypical odontalgia^[45,46] or sometimes phantom toothache.^[47,48]

Graff-Radford and Solberg^[42] suggested the criteria for identification of atypical odontalgia:

- Pain in a tooth or tooth site
- Pain is continuous or almost continuous in nature
- Pain persisting for more than 4 months
- No signs of local or referred pain
- Equivocal results of somatic block.

The other clinical characteristics of an atypical odontalgia involves:^[4,42,43,49-51]

- Pain quality is described a dull, aching, throbbing, or burning
- Pain is diffuse in nature and not altered by intraoral thermal stimuli
- Pain not always restricted to a tooth (e.g, the area may be edentulous)
- Pain that may or may not be relieved by a diagnostic intraoral local anesthetic block.

Neurovascular Toothache

Neurovascular pains are the group of visceral pain disorders that are generally characterized by episodic pains accompanied by neurologic, gastrointestinal, and psychological changes. The International Headache Society classifies these pain disorders as primary headaches.^[52]

Mechanism of Pain Referral

Previously, it has been established that pain occurred as a result of dilatation of arteries, which distort and noxiously stimulate the sensory receptors and afferent fibers in vascular and perivascular tissues. However, recent investigations suggested that vascular changes that have been observed to be associated with the pain are merely a result of the condition and not the actual cause of the pain. The actual mechanism appears to be more closely related to a neurogenic phenomenon, and thus termed neurovascular pain.^[52]

It has common mechanism involving the trigeminovascular system. The most common type of neurovascular pain is

migraine; other includes tension-type headache, cluster headache, and other trigeminal autonomic cephalgias.

Clinical characteristics

The clinical characteristics of the toothache of neurovascular origin are as follows:^[53]

1. The pain is usually unilateral, spontaneous, variable, and throbbing-characteristics that stimulate pulpal pain
2. Toothache is characterized by periods of remission and exacerbations over months or years
3. Episodes of pain may possess a temporal behavior appearing at similar times during the day, week, or month
4. There is a lack of reasonable dental cause for the pain
5. The effect of local anesthesia is unpredictable
6. The pain is frequently initially felt in a tooth (the maxillary canine and premolar usually) as a toothache
7. Recurrence is a characteristic of neurovascular pains
8. It may spread to adjacent teeth, opposing teeth, or the entire face
9. If the pain experience is protracted, it may induce autonomic effects manifested as nasal congestion, lacrimation, and edema of the eyelids and face, which may be mistaken for sinusitis or a dental abscess
10. The pain may respond to ipsilateral carotid pressure or ergotamine tartrate.

The chief indicators that help distinguish this toothache from true odontogenous toothache are:

- Absence of adequate dental cause
- Tendency for it to be periodic and recurrent
- Patient's ability to precisely locate the painful tooth.

Psychogenic Toothache

Psychogenic pain can be described as a pain associated with psychologic factors in the absence of any physiologic cause. The American Psychiatric Association has classified this condition as a somatoform pain disorder.^[5,54]

Mechanism

Orofacial pain of psychological origin is explained by "biopsychosocial model." In the biopsychosocial model, the "bio" component denotes the nociceptive input that arises from the somatic tissues; and the "psychosocial" component represents the effect of the interaction between the thalamus, cortex, and limbic structures. The neurotransmission of impulses between all these higher centers is responsible for the psychologic aspects of pain. A biologic factor that contributes to the pain experience includes genetics, fitness level, nutritional status, autonomic balance, and allostatic load. Allostatic load refers to the physical stress on an individual from repeated physiologic

activation and inhibition that comes from responding to life stressors.^[55,56]

Clinical characteristics

The clinical characteristics of the toothache of psychogenic origin are as follows:^[4,5,51,57]

1. Multiple teeth are often involved.
2. The patient reports that multiple teeth are often painful with frequent change in character and location (pain jumps from one tooth to another). Pain can be described as diffuse, vague, and difficult to localize.
3. There is a general departure from normal or physiologic patterns of pain and presents without any identifiable pathologic cause. Pain may be sharp, stabbing, intense, and sensitive to temperature changes, all of which are similar to pain symptoms of odontogenic origin.
4. The patient presents with chronic pain behavior.
5. There is a lack of response to reasonable dental treatment
6. There is an unusual and unexpected response to therapy.
7. There is no identifiable pathology that can explain the toothache.

Given that psychogenic toothache is a somatoform disorder, dental treatment will not resolve symptoms of pain and may potentially elicit an unexpected or unusual response to therapy. Patients should be referred to a psychiatrist or psychologist for further management.^[4,5]

CONCLUSION

In clinical practice, non-odontogenic toothaches may pose challenge to the oral physician in terms of diagnosis. If symptoms and clinical findings do not appear to be consistent with the typical oral disease, or if standard treatments do not alleviate the pain, the oral physician must consider other, more complex orofacial pain diagnosis. For appropriate diagnosis, pain must be considered in terms of quality, duration, referral pattern, exacerbating, and relieving factors. The characteristic clinical features and the perceived origin of the pain may be pathognomonic for specific sites. When patients present with diffuse pain and/or pain radiating to other areas of head and neck, non-odontogenic sources should be given additional consideration. To arrive at the correct diagnosis of toothache of odontogenic and non-odontogenic origin, precise understanding of specific clinical characteristics, and thorough evaluation of the nature of the pain are recommended. This will essential to deliver appropriate treatment and avoid unnecessary procedures that will aggravate the condition.

In summary, it is reasonable to conclude that:

- Therapy must not start without an appropriate diagnosis.
- Consider all pains in the mouth and face to be of dental origin until proved otherwise.
- Management is always directed by the presented symptoms, their course, and influencing factors.
- Successful therapy is achieved by treating the source of pain, not the site of pain.

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How to cite this article: Soni A. Toothache of Non-Dental Origin: A Review of Its Mechanism and Clinical Characteristics. Int J Sci Stud 2018;6(2):26-35.

Source of Support: Nil, **Conflict of Interest:** None declared.