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Master‘s Thesis

Dental Pain Beyond Trigeminal Neuralgia

A Literature Review

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SUMMARY

The correct diagnosis of trigeminal neuralgia is still a clinical challenge for dentists. Therefore, the patients are still witnessing unnecessary dental treatment and procedures like teeth extraction and endodontic treatment before getting the correct diagnosis and a relative treatment. This diagnosis remains clinical, so the description and diagnostic criteria of trigeminal neuralgia and dental pain are presented in the thesis. The clinical case reports when the patients have experienced dental treatment for their misdiagnosed trigeminal neuralgia are correspondingly overviewed.

This literature review emphasizes the misdiagnosis of trigeminal neuralgia and presents the clinical characteristics and management options of this condition versus some common dental pathologies such as pulpitis, odontogenic sinusitis and myofacial pain. The clinical cases present the misdiagnosis of trigeminal neuralgia with dental pains and show that trigeminal neuralgia is a more common complaint than it's reported. It explains that there is a general lack of recognizing the symptoms of trigeminal pain that constantly causes the mischaracterization of the source of the complaint. Dentists are in a great position to detect trigeminal neuralgia and help reach a definite diagnosis. The high occurrence of orofacial pain with dental origin and the clinical symptom parallels between odontogenic pain, neuropathic orofacial pain, and other pathologic pains in the facial region constantly causes incorrect diagnoses. More critically, improper treatment procedures are frequently irreversible and veritably invasive. In connection with etiology, the records show that utmost onsets were connected with special dental treatment or odontogenic symptoms that ended in dental treatment.

Keywords: trigeminal neuralgia, dental pain, endodontic treatment, sinusitis, myofascial pain, misdiagnosis.

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1. INTRODUCTION

1.1 Problem statement

Trigeminal neuralgia (TN) is a neuropathic pain condition in the orofacial region that should be encountered in the daily work of a dentist. Definitive diagnosis of facial pain is a complicated task and requires expertise and awareness. The studies show only 1 of 6 patients with TN condition received a correct diagnosis at the first consultation (38). Misdiagnosis can result in incorrect treatments. Inaccurate interpretations of symptoms, in particular, can negatively impact the scientific conversation. The most common cause of pain in the lower face is a dental cause, for example, pain linking to the teeth and their neighboring structures. However, some dental situations are easy to diagnose with a careful examination using a light, mirror and simple dental instruments; others may need investigations with dental X-rays. Dentists are excellent and well-trained in diagnosing most dental pains, so patients trust them for a check-up and further assessments. Although, awareness should be raised on the dentists doing irreversible dental treatment on a patient based on the medical history alone and symptoms explained by the patients without any clinical signs and X-ray validation. (5) Many patients with trigeminal neuralgia have gone through unnecessary dental treatments for instance endodontic treatments teeth extractions, which has been documented by neurosurgeons (5,38). By the initial presentation of the TN, this confusion can be understandable as the pain is sharp, intermittent, and may be felled for the patient like dental pain. The same patient with neuropathic pain can undergo a complex dental treatment for hours and ultimately find out it does nothing for pain relief. The most challenging dental diagnosis can be completed by more sophisticated imaging like cone beam CT; also, injection of local anesthesia and analgesics can be helpful while diagnosing and differentiating between TN and usual dental pain (23). However, the correct diagnosis of trigeminal neuralgia is still a clinical challenge for dentists.

1.2 AIM

While TN is frequently misidentified and treatment is often delayed, the research **aim** is to analyze the misdiagnosis of trigeminal neuralgia with dental pain.

2. OBJECTIVES

The **main objectives** were formulated:

1) To overview the modernized knowledge of the pathophysiology and clinical features of trigeminal neuralgia.

2) To describe the most common dental pain pathologies that can be mimicked by trigeminal neuralgia

3) To advise dental caregivers with trigeminal neuralgia's clinical image and diagnostic approach.

3. METHODOLOGY

A PubMed search was conducted by inserting the keywords "dental pain", "endodontic treatment", and "extraction," each separately in combination with the term "trigeminal neuralgia". To obtain only the most recent data, a timeframe only including articles from the last thirteen years (2010 – 2023) was set within the PubMed database. Sorted by best match, the search "dental pain" and "trigeminal neuralgia" yielded 21 matches, the search "endodontic treatment" and "trigeminal neuralgia" 70 matches, and the search "extraction" and "trigeminal neuralgia" 160 matches, potentially giving 261 articles to choose from. The actual number was lower as there was a considerably high number of cross–matches between the searches of the mentioned keywords. Nonetheless, inclusion and exclusion criteria were set apart from the timeframe to create a relevant literature review on the research topic.

Regarding language, only articles published in English were considered. Some additional papers found by reviewing the reference list of articles meeting the inclusion criteria have been included. To not start this literature review with a preconceived opinion, reviews of other authors on the misdiagnosis of trigeminal neuralgia were neglected on the first search attempt. In later stages of the work process, narrative studies, exclusively focusing on the diagnosis and misdiagnosis of trigeminal neuralgia and dental pains and systematic reviews were included.

3.1 Structure

This thesis includes an introduction, methodology, literature review, discussion, results and conclusion. It contains 36 pages, four figures, two tables, and 60 references.

4. LITERATURE REVIEW

4.1 Definition and Classification of Neuropathic Pain

TN is a neuropathic pain condition in the orofacial region. Understanding all neuropathic disorders is crucial to identify the pathology when symptoms are presented besides underlying diseases. If the correct diagnosis is made, the patient can be referred to the responsible specialist for further investigations and treatment (19). But how can tooth pain mimic neuropathic pain and vice versa? The International Association for the Study of Pain (IASP) has defined neuropathic pain as pain "initiated or caused by a primary lesion or dysfunction in the nervous system." The indications of neuropathic pain may be an abnormality in the function or structure of the peripheral or central nervous system (PNS)or (CNS). There are a variety of etiologies that can cause neuropathic pain. Some etiologies include trauma, infections, radiation, chemotherapy, surgery, metabolic abnormalities, neurotoxins, nerve compression, inflammation, and tumor infiltration (1). Neuropathic pains can be a real struggle for doctors at this time due to difficult management conditions. Thus they mostly have been reported as chronic pain situations. In recent years, there has been massive progress in acknowledging the causes and the etiology of neuropathic pain. The diagnosis can be made by the physical examination and the patient's medical history. Patients show symptoms such as hypoesthesia, hyperesthesia, paresthesia, dysesthesia, neuralgia, or burning, typical symptoms of neuralgia. These symptoms can occur spontaneously or can be elicited (19). Neuropathic pain can be divided into episodic and continuous neuropathic pain. Episodic neuropathic pain has two subgroups: paroxysmal neuralgia pain and neurovascular pain. Ongoing neuropathic pain is divided into three subgroups: peripheral-mediated pain, central-mediated pain, and metabolic polyneuropathies (7).

4.2 Trigeminal Neuralgia

4.2.1 Clinical characteristics

TN is one of the most common neuralgias in the face. TN is also known as TN *tic douloureux*, and its clinical symptoms are generally unilateral with an unforeseen, severe, pecking, shock-suchlike onset. Typically, they are provoked during mastication, talking, when cold air is on the face, touching, eating, and tooth brushing. Generally, the pain is allocated at the maxillary and mandibular jitters, the alternate and third branches of the trigeminal whim-whams (5). Indeed, though TN is a rare pain complaint, TN is one of the most common neuralgias. Only cases are

estimated by the Institute of neurological diseases and Stroke, and women are more current than men (9).

Moreover, most of the cases are after the age of 50. The individual criteria of the International Bracket of Headache Diseases (ICHD- 3) for TN include pain that lasts from a bit of an alternate to 2 twinkles with the symptoms mentioned ahead. They must be touched off by an inoffensive vexation within the affected trigeminal whim-whams (15). Besides that, the ICHD-3 helps us classify TN further into two groups: classical TN and secondary TN. Secondary TN can be subdivided into idiopathic TN and characteristic TN. Classical TN can be purely ferocious. Then the case doesn't have pain between the attacks or classical TN with patient attacks, whereby the issue continues to witness constant pain (25). Atypical signs, similar to patient bilateral pain, in the background of cases under 50 and with sensitive impairments and an abnormal neurological examination, dizziness, or impassiveness indicate characteristic TN.

According to this bracket, expected TN results from a serious neurological complaints similar to a space-enwrapping excrescence/ lesion of the cerebellopontine angle, multiple sclerosis, herpes zoster contagion, or, indeed, trauma. Incipiently, the bracket of idiopathic TN has the same symptoms as classical TN, but it happens without an egregious reason. Also, the radiographic and electrophysical examination doesn't reveal different pathology between these two (25). The exact etiology of TN is unknown due to its complexity, but the proposition of vascular contraction is the most likely bone. The predominant pathophysiological process is the demyelination of the primary sensitive trigeminal afferents in the root entry zone. In utmost cases, vascular conflict with the trigeminal root changes in the morphology similar to contraction will initiate the jitters' demyelination, enabling ectopic impulses and ephaptic crosstalk. Most, you will likely see morphological changes in cases with classical TN. In contrast patients with secondary TN will have pathological changes caused by neurological diseases or space-enwrapping lesions which affect the trigeminal whim-whams (27). To diagnose TN, we must first predicate it on the case's information and history. If we suspect a case to have TN, it's recommended to search for the newest bracket criteria on the internet initially. The alternate step should be to perform an MRI. MRI is preferred over CT because it can help reveal small lesions conterminous to the whim-whams and multiple sclerosis or cerebellopontine angle excrescences. Some individual criteria were developed to help during the personal procedure (38). The A criterion is that the case should have intermittent attacks of facial pain in the area of the

trigeminal whim-whams and should fulfill both of the following two criteria B and C. B criteria includes that the pain should last one second over to two twinkles, that it should be with severe intensity (firing pain or electric shock- suchlike). C order is that inoffensive stimulants should provoke pain. The last order is D, where we can divide the TN into three orders. Classical TN, which was provoked by neuromuscular contraction. Secondary TN is caused by underpinning conditions, and idiopathic TN is where we don't have any pathological changes visible (41). To get a better isolation of the opinion, the croaker should pay special attention to the history of the case, how the case describes the onset of the pain, what he says about former treatments and the position of the pain. When the symptoms of the case are analogous or common to the short-continuing once of a herpes zoster rash, it should be taken into account that the cause of the trigeminal neuropathy could be an acute outbreak of herpes zoster. However, we should remember that the cause for the neuropathy is post-traumatic because of the former treatment. If the case mentions an invasive dental treatment, the pain has passed on the same side analogous to TN but with loss and gain of functions. Incipiently, the position of the pain. Bilateral pain, especially in the temporomandibular region, can signify common temporomandibular diseases. If the pain only occurs during chewing, we should consider dental pathologies such as a fractured tooth or malocclusion (43,45). If sudden, brief attacks of unilateral pain in the orbital region, the periorbital, or temporal region is present, this can be a sign of short-lasting unilateral neuralgiform headache attacks (SUNA) and short-lasting unilateral neuralgiform headache with conjunctival injection and tearing (SUNCT) (50).

4.2.2 Management Considerations

For TN, we have three options: medications, surgery, and complementary approaches. The first choice for the initial treatment should be medical pharmacotherapy. In medical therapy, carbamazepine is the medication of choice and is used as first-line medication. carbamazepine (600-1200mg/day) is normally used for treating patients who have epilepsy. This medication is used to stabilize the sodium channels when they are inactive. But like all medications, it has some side effects like concentration problems of the patient, and the patient can feel dizzy and tired. Also, some more severe complications are known, such as agranulocytosis or aplastic anemia (51,26). A study in 2015 showed that oxcarbazepine (600-1800mg/day) could also be used as a second option for first-line medication, as it has the same efficiency results as

carbamazepine but fewer side effects (45,40). Both medications showed a decreased interaction with other drugs and better tolerability (11). The second line of medications are lamotrigine (400mg/day) and baclofen (40mg-80mg/day). Lamotrigine is a sodium channel inhibitor normally used to treat patients with bipolar disorders. Baclofen, which depresses the excitation of the neurotransmission, can be combined with carbamazepine or alone. Pregabalin and levetiracetam, newer medications, should be used as third-line medications. Still, they should be considered critically since insufficient scientific evidence exists for treating patients with this kind of non-anticonvulsants (41). As the second step, surgical treatment can be taken into consideration. Indications for surgical therapy are two failed treatment attempts with medications. Surgical interventions can be percutaneous procedures on the Gasserian ganglion, balloon compression, gamma knife radiosurgery, and microvascular decompression, which has the best outcome. More than 90% of the patients are pain-free after this procedure, and more than 80% are after the first year of treatment. Many studies were revealed within the last years about surgical interventions and when it is the right time to start with them. Two parties have built up. One recommends starting early with surgeries; the others recommend starting with it after two failed treatment attempts. The scientific evidence is missing for both hypotheses to determine which is right. But to start with surgical treatment right after failed medicinal treatment seems important (40,45). The third option is to use neuromodulation to treat TN. Neuromodulation uses advanced chemical and electrical technologies to target neural stimulation or inhibition to recreate a normal function. Still, as before, strong recommendations cannot be made due to its early stage. However, this part of modern technology is an exciting field with a lot of promise and should be considered (51).

4.3 Dental pain

4.3.1 Etiology

Epidemiologic studies have shown that toothaches are the most common form of orofacial pain, with about 12 to 14% of the population reporting a history of toothache over six months (6). However, it should be noted that toothache may not always be originated from the dental structure. Thus, pain management starts with developing a precise differential diagnosis of the pain's origin. It is an essential first step in managing pain because adequate treatment must be used for controlling or eliminating the underlying causes. The unique characteristic of the teeth

structure is that they are visceral tissues that function as a part of the musculoskeletal system. It explains some of the uncertain behavior of dental pain. The tooth pulps are served as an isolated organ; therefore, each individual in their rights. The sensory ability of dental pulpal tissue is like that of other visceral structures, and pulpal pain is like other visceral pains (7). However, the connection of the teeth to the bone is an authentic joint. It, therefore, constitutes a musculoskeletal structure called periodontal ligaments that convert the mastication forces to traction on the alveolar bone. The unique characteristic of the teeth and their connections to the alveolar bone is that visceral structures function as an elemental division of the musculoskeletal systems. The sensory attitude, for example, dental pain, includes a combination of visceral characteristics and musculoskeletal ones.

The teeth are connected to the alveolar bone by the fibrous joints, and the periodontal ligaments complete the fibrous, flexible parts. The collagen fibers that unify the alveolar cortex and the cementum are obliquely arranged to convert the masticatory pressures into forces of traction on the bone. When the sensory receptors that consist of the mechanoreceptors and free nerve endings are triggered by masticatory stress, the mechanoreceptors apply inhibitory effects on the action of the masticatory elevator muscle. (22,23) This reaction is known as the nociceptive reflex. It is important to note that sensory innervation of the teeth is ipsilateral; except for pain initiating from structures of mid-face, the sensory demonstrations from the oral cavity are not showing bilaterally or contra-laterally (28).

4.3.2 Dental pain behaviour and quality

It is essential to know that dental pains are very adaptable and can mimic almost all pain disorders. The severity of these kinds of pains can differ from mild soreness to unbearable intensity. They can be spontaneous or can be generated in many different ways. They might be intermittent, meaning there are periods of no pain between the pain attacks (49). They can be uninterrupted but periodic by acute worsening attacks radiating all over the head and neck. The wide range of toothache is an important rule for any clinician to count all pains in the face and oral cavity to have a dental origin until it is proven with a different source. As mentioned before, an adequate dental examination is the crucial first step in managing all pains, including face, oral cavity and dental pains. Despite this variability, dental pains present clinical characteristics that put them in the category of deep somatic pain (48). Dental pain generally is explained as an

aching feeling, occasionally throbbing. In the case of mild discomfort, it can be felt only as soreness or tenderness. When it is severe, it can have a burning quality. The essential background aching pain may be punctuated by short, repeated acute pains that diffuse widely. Dental pains can happen spontaneously or be associated with stimuli like sensibility while eating sweets and other food or liquids, thermal changes, or applying pressure or touch. Dental pains can be spontaneous or protracted, while they can be intermittent or continuous (16).

4.3.3 Localization of dental pain

Usually, patients have difficulty showing the location of the pain arising from the dental pulp. It is often hard for them to confirm whether the involved tooth is located in the maxilla or mandible, or even less likely which teeth it is. The pain is felt diversely in the head, face, jaws and teeth. However, the patients show pain from periodontal structures quite well, especially when the involved tooth is pressed or touched. This characteristic can be a plus to outspread radiate pain within the jaws and face and may need diagnostic administration. It is not uncommon for patients to question the localization of the pain when it is widely spread, and they can also feel the pain in any other structures (44). They can make mistakes while the clinician and point are doing percussion to a wrong tooth as an offended tooth. However, localizing the pain from periodontal structures is accurate for making the diagnosis based on that. Localization of dental pain can depend on whether the pain originates from the pulpal structures, periodontal sources, or both (30).

4.4 Odontogenic Toothache

As mentioned above, odontogenic toothaches have their originating sources in dental structures. These structures can be either the tooth (mostly pulp) or the periodontal structures. Odontogenic toothaches are the most common reason patients seek dental treatment when in pain. These toothaches are also divided into subgroups of pulpal origins and periodontal origins (20).

4.5 Dental pain with pulpal origins

Healthy pulp tissue can be recognized by visceral pain. The pain is the type of threshold pain compared with the promoted response of musculoskeletal pain. Thus, with healthy pulp tissue, no response happens until the threshold level is touched. Pulpal pain responds to a pernicious

provocation unrelated to the usual movements of mastication. It usually reacts to direct exploration, shock, chemical, and thermal irritants but not general mastication forces (16). As mentioned before, it is often a struggle for the patient to show the location of the pain. These pains can be sub-classified as chronic, acute, recurrent, or mixed with periodontal elements. A primary clinical characteristic of pulpal pain is that it does not stay the same over time. Often it resolves, changes to chronic, or progresses to include the periodontal structures by direct extension through the apex of the teeth. In rare cases, it may remain unchanged longer (24).

All in all, pulpal pain characteristics are explained by the following:

• The quality of the pain is throbbing, dull, aching, and may occasionally be sharp, depending on the pulp condition.

• Unilateral pain.

• Presence of clinical etiology for odontogenic toothaches like deep decay and restoration leakage.

• There is the ability to reach the peak of the pain complaint during the clinical examination.

• The affected tooth is sensitive to thermal stimuli and percussion.

• The pain tends to get better or worsen, rarely remaining in the same condition over time.

• The pain can be eliminated or reduced by injection of local anesthesia to the affected tooth region (22,24,50).

4.6 Acute pulpal pain (Pulpitis)

4.6.1 Clinical characteristics

The most common of all visceral pains is acute pulpal pain, known as pulpitis. It is usually complicated to show the location, and the patient cannot help in finding the origin of the pain. Some internal objectives like erosion into the pulp chamber or root canal, very deep caries, chipping, splitting, and fracture can directly find the affected tooth. The reason for pulpitis pain is pernicious stimulation of the receptors in the pulp (18). Generally, the tooth structure inhibits all the external stimulation that may reach the nerve endings; thus, only extreme surface simulators like electrical and thermal stimuli are sensed as pain. Dental pulpitis can occur due to infection from deep caries near the pulp or by inflammation raised from thermal or chemical stimulation after dental treatment. It can be reversible or irreversible. Intermittent sharp, throbbing, or shooting pain attacks are also the pain symptoms of TN, so clinicians have to be

careful about the differential diagnoses (22). Pulp inflammation may happen by bacterial infiltration when the teeth are destructed due to severe caries.

Nevertheless, iatrogenic destruction, such as restoration close to the dental pulp or trauma, can cause inflammation in the pulp. It may lead to an extreme sensitivity to sweet and cold with short and sharp 'neuralgic' pain. To minimize the risk of acute pulpitis turning into chronic irreversible pulpitis, the exposure of pulp to chemical and bacterial irritation has to be considered by dietary and salivary content. The pulpitis will become irreversible if the original cause persists and is not eliminated (16,22).

The elevation in pulpal vascularity may result in intra-pulpal pressure rise, which associates with sensitivity and ischemia, prolonged pain reaction to thermal stimuli. Generally, the situation may lead to pulpal necrosis. When this occurs, pain from the pulpal tissue ceases. This transition might be highly variable. If an infection is the cause of pulpal inflammation, the change is usually fast and involves pulpal and periodontal aching at first, which terminates in an acute periodontal abscess (22). If the pulpal inflammation is closed and sterile, all pain may discontinue, and a radicular cyst or painless periapical granuloma can develop. The moment pulp necrosis has happened, the infection advances through the apical portion of the tooth into the surrounding alveolar bone and the periodontium, starting periodontal inflammation, which has the potential to be followed by a dental abscess that causes spontaneous prolonged pain and pain while occlusion or chewing (17).

Generally, the pain arising from an abscess is defined as a spontaneous pain attack or throbbing that may last long, from hours to days. Swelling in the jaw, lymphadenopathy, or trismus may be associating indicatives of an acute infection spread which shows that different stages of the infection process may have other clinical characteristics (16).

4.6.2 Management Considerations

care for dental pulpitis is to dig all the tooth decay postdated by pulp disposal, called pulpectomy or root canal treatment, and restoration. However, if the acute infection occurs, e.g., periostitis, it might require drainage intraorally via drainage of involved tissues (24).

4.7 Chronic pulpal pain

4.7.1 Clinical Characteristics

As mentioned, under some circumstances, injured/ infected pulp tissue can guide to a chronic inflammatory order, and it undergoes changes that develop neither to agree nor to necrosis. Still, it stays indefinitely as what is generally defined as chronic pulpitis. The status that may bring this transition is trauma to a primary tooth, particularly the ones with an open apical portion which are less likely to ease necrosis and congestion of the pulp (18). These teeth reply positively to vitality testing, although at a significantly lower position than teeth with typical and undamaged pulps. Intermittently, internal resorption of the tooth can do. When chronic pulpitis occurs, the pain answers alter from the relatively variable symptoms of acute pulpal pain to a minor variable discomfort that cannot be outlined as pain. Nevertheless, the tooth can become symptomless and only show the response if more injuries occur (17).

4.8 Recurrent pulpal pain

4.8.1 Clinical Characteristics

Severe acute pulpal pain can be recurrent in some rare cases because the inflammation generally progresses to chronicity, resolution, or necrosis. When acute pulpal pain occurs recurrently, it usually is the sequential pattern of recurrent inflammation. Real recurrent pulpal pain usually is not causing toothache, but it can be sensed as a recurrent hypersensitivity (17). These kinds of conditions are generally associated with changes and dis-balanced in fluid balance or vascular pressure, which can also be called menstrual toothache; furthermore, a high-altitude toothache is also in this category. The affected tooth is usually slightly inflamed, and the pain threshold can be lowered in suitable environmental conditions. Other examples of recurrent pulpal pain hypersensitive responses of the tooth to stimuli such as sweets, thermal changes, or occlusion abuse can be noted (23).

4.9 Dental pains with the periodontal origin

Periodontal pain is a musculoskeletal deep somatic pain, meaning that the pain's localization is more visible than pulpal pain. It is directly related to mastication forces (biomechanical function). It reacts to the pain stimuli proportionally and gradually rises rather than being felt as a threshold pain, like pulpal pain. The periodontal ligament receptors can localize the stimulus more precisely; thus, periodontal pain can rarely be problematic diagnostically because the affected

tooth is already identified (11). Localization is determined by applying pressure to the affected tooth laterally and axially and usually under a load of mastication forces and occlusal pressure while chewing; the tooth feels oversized or elongated and sore. The pain and discomfort can be felt when the biting pressure is released rather than while biting. The reasons for periodontal pain are different. It can happen as a condition of periodontal inflammation for a local cause like occlusal overloading, trauma, or contact with an adjacent tooth. It can sometimes occur due to dental prophylaxis, orthodontic treatment, endodontic treatment, prosthetic tooth restoration, inadequate occlusal contact with opposing tooth, stresses to abutment teeth, occlusal interference, over-contoured or over-contoured or under-contoured proximal contact points, or surgical interference of any kinds (20). When periodontal pain includes few teeth, especially antagonists, occlusal overloading should remain in mind. Overloading may happen from abnormal heavy occlusal contacts during jaw movements, bruxism, and clenching.

In general, periodontal pain is described by the following points:

• The quality of the pain is dull, aching, and sometimes throbbing.

• There is a noticeable periodontal situation such as furcation, abscess, or pockets.

• Pain is proportional to the degree of local.

• Biting on the tooth may raise the pain.

• Release after biting may also increase the pain.

• Injection of local anesthesia in the affected tooth region may reduce or eliminate the pain.

To sum up, dental pain of periodontal origin has an obvious source or visual characteristics that can be seen in a routine dental X-ray or a simple intraoral examination. Nevertheless, in case of an abscess, an extraoral objective like swelling can be observed (11,23).

4.10 Toothache with the myofascial origin

4.10.1 Etiology

Myofascial pain is an indigenous myogenous pain condition characterized by original areas of establishment, hypersensitive bands of muscle tissues known as sensor points. It's a muscle pain complaint diagnosed in farther than 50 cases reported to a university pain center (53). In this pain condition, truly localized areas in muscle tissue and/ or their tendinous attachments (sensor point)

are felt as tense bands when gentled, which elicits pain. The exact nature of a sensor point has yet to be discovered (38). It has been suggested that certain nociceptors in the muscle tissues may come adapted by algogenic substances that produce a localized zone of perceptivity (55). A sensor point is a circumscribed region where, numerous motor units feel constricting. Because a sensor point has only a select group of motor unit exertion, no overall shortening of the muscle results. The unique particularity of sensor points is that they are a source of constant deep pain and, therefore, can produce appertained pain to various facial structures, including the teeth. The etiology of myofascial pain is complex. Unfortunately, we do not entirely understand this myogenic pain condition (56).

4.10.2 Clinical characteristics:

Myofascial pain is usually explained as a dull, deep, hurting muscle pain that might be associated with dental pain. Some studies have reported that three muscles of mastication, the masseter, anterior digastric, and temporal muscles, generally direct the pain to the teeth (44). Masseter's superior belly sends the triggers to the maxillary posterior teeth. The inferior belly refers to the pain in the mandibular posterior teeth, anterior digastric to the mandibular anterior teeth, and temporal muscle sends the triggers to the maxillary anterior or posterior teeth. In a series of 230 cases with an opinion of temporomandibular complaint, 85 demonstrated appertained pain with palpation of muscles or detector points, with 11. Six of the cases had pain appertained to the teeth (33). Importantly, molars were the teeth that most constantly entered appertained pain from muscle or detector point palpation, with the masseter muscles being the most common source. The temporalis and the TMJ itself also generally appertained pain to the teeth. Thus, the clinician must flick these muscles for implicit sources of pain referral. An intriguing clinical point of a detector point is that it may present in either an active or an idle state. In the active state, it produces central excitatory goods. Thus, a toothache is generally felt when a detector point is active (54).

Because appertained pain wholly depends on its source, clasping an active detector point (original stimulation) frequently increases similar pain. Although it isn't always present when this characteristic appears, it has significant individual value. In the idle state, a detector point is no longer sensitive to palpation and thus doesn't produce appertained pain. When inactive,

detector points cannot be set up by palpation, and the case does not complain of toothache pain. In this case, the history is the only data that leads the clinician to diagnose myofascial pain (55).

In some cases, the clinician should consider asking the patient to return to the office when the toothache is present so that evidence of the pain referral pattern can be vindicated and the opinion can be verified. It's allowed that detector points don't resolve without treatment. They may come idle or dormant, temporarily relieving the appertained pain. Detector points may be actuated by colorful factors similar to increased muscle use, the strain on the muscle, emotional stress, or an upper respiratory infection. When detector points are actuated, the toothache returns. It's a common finding with cases who complain of a regular late autumn toothache following a veritably searing and stressful day (55).

A summary of the clinical characteristics of a toothache of myofascial origin follows

• The pain is fairly constant, dull, hurting, and not pulsatile.

• The original stimulation of the tooth does not alter the pain.

• Examination reveals the presence of localized establishment and hypersensitive bands within the muscle tissues (detector points).

• Other heterotopic pains are frequently reported (e.g.," pressure-type" headache).

• The toothache is increased with the function of the involved muscle (detector points).

• Palpation and stimulation of the detector points increase the toothache.

• Verified anesthesia of the tooth does not alter the toothache.

• local anesthetic injection of the involved muscle (detector points) reduces the toothache (53,55).

4.10.3 Management considerations

Several treatment modalities have been suggested for myofascial pain. A partial list includes relating and closing out contributing factors, mild painkillers, spray and stretch therapeutic, deep massage, biofeedback, deep heat, and injection of the detector points (32).

4.11 Muscular Toothache

Heterotopic pain displaying as a simple toothache or a kind of apparent tenderness of the teeth (secondary hyperalgesia) can happen due to myofascial trigger points in the orofacial muscle system Trigger points in the muscle temporalis can direct the pain to maxillary teeth. The superficial masseter muscle trigger points may cause discomfort in the maxilla and mandible's molar areas (46). The pain that can be felt in anterior mandibular teeth can be from digastric

muscle trigger points. A muscle-induced heterotopic toothache may be differentiated from an odontogenic toothache because of insufficient dental cause. Also, local anesthesia administration will not eliminate the pain (49).

Myofascial pain is generally explained by dull, aching, and regional muscle pain and the localized trigger points in a tendon, muscle or fascia. However these symptoms are the most typical characteristics of muscle pain presentation, but there are some cases in pain severity and the quality of pain is similar to TN. The pain severity of myofascial pain attacks can be ranged from agonizing to some painless attacks. Sometimes the pain can be reported as high or even higher than pain with neurological origins. The pain in the myofascial region can be aggravated by mandibular function, which involves the masticatory muscles like the TN episodes that sometimes can show up by fascial and tongue movements or chewing (48,30).

4.12 Toothache with sinus and nasal mucosal origin

4.12.1 Etiology

Pain arising from the nasal mucosa responding from viral, bacterial, or antipathetic rhinitis may be expressed as related pain throughout the maxilla and maxillary teeth in the form of a toothache. It may also display autonomic signs wrong for maxillary sinusitis symptoms. The sinus headache may cause handling problems due to the associated non-odontogenic tooth pain. The etiology for the non-odontogenic pain is likely to arise from inflammation of the ostium, which compresses a significant number of nociceptors and frequently refers pain to the maxillary teeth (43).

4.12.2 Clinical characteristics

In discrepancy with odontogenic pain, non-odontogenic dental pain that occurs by sinusitis is commonly defined as pain not only in a single tooth (for instance, pain can contain the malar and maxillary alveolus areas), pain that can be relatively relieved by a diagnostic local anesthetic block intraorally, pain that escalates after percussion, and casual thermal perceptivity to cold wave. (43,44) Patients may also describe sensing wholeness or pressure in the infraorbital region on top of the infected sinus. A particular and bold character is the company of several maxillary posterior teeth sensitive to percussion with a positive response to the pulpal vitality test.

Applying a lidocaine spray intranasally can be helpful in diagnosis if it leads to downgrading the pain (48). As an optional system, a swab impregnated with five lidocaine may be applied in the middle meatus for 30 seconds to impose pain reduction (35). Bacterial sinusitis pain is described as an intense, throbbing, stabbing pain with applying pressure. More than 70% of cases of bacterial sinusitis are *Streptococcus pneumoniae* and Hemophilus influenzae (32). if the patient complains of moderate to severe pain, the "head-dip" test will be positive. For example, pain increases when the patient lies down or places the head below the knees, and purulent nasal discharge is reported. Radiographic observations, especially a water view of the sinuses' computed tomography (CT) scan, can show a fluid accumulation in the sinuses (43). Allergic sinusitis can depend on the seasons and colder weather temperatures but can occur in any climate and season. Most of the time, the pain is described as a chronic dull pain in the posterior region of the maxilla with a positive response to percussion in the premolars and molars. Rarely do some patients also report an "itching" feeling in the maxillary teeth. A brief clinical characteristic relating to a toothache that has sinus and nasal mucosal origin follows:

• The patient feels pressure below the eye(s).
• The pain is escalating by applying pressure over the sinus, which is involved.
• Tooth-positive response to percussion.
• Increasing the toothache by lowering the head.
• The toothache can get worse by stepping hard onto the heel of the foot, for instance, walking down the way.
• Local anesthesia of the tooth may be useful only incompletely for pain elimination or can also fail to drop the pain.
• The diagnosis can be confirmed by sinus imaging, like using a Computed Tomography scan or Waters view (51).

4.12.3 Management considerations

Bacterial sinusitis is mostly treated by antibiotics with β- lactamase–resistant like trimethoprim-sulfamethoxazole or the more known ones like amoxicillin with clavulanic acid. (44) Allergic sinusitis is usually treated by decongestants or/and antihistamines. The reduction of atmospheric pressure may also stimulate pain in the maxillary sinuses and the maxillary and mandibular teeth. Some reports have shown odontogenic and non-odontogenic pain in patients' teeth during or after

flights or scuba diving. (43) It is recommended that dental treatments and root canal obturations be completed more than 24 hours before exposure to atmospheric pressure reduction. (32) Knowing that a few other inflammatory situations can lead to misdiagnosis is essential. These contain pain related to another tooth, eruption sequestrum of bone, (35) impacted third molars, foreign bodies impacted into periodontal tissues during mastication or retained under surgical tissue flaps and otitis media (34,37). The exact diagnosis needs a detailed medical history, clinical examination including X-ray and MRI, and interpretation of findings.

5. DISCUSSION

5.1 Trigeminal neuralgia and pupal pain

Fundamentally, TN is categorized into classical, secondary, and idiopathic subgroups. The lifetime prevalence of TN is approximately 0.16 % to 0.3 %. It is more prevalent in women than men by the ratio of 3:2. Its incidence increases with age, so most of the patients are in the age group of 24 to 93 years old however a recent pediatric headache clinic identified five children with TN with the age between 9.5 to 16.5 years (57,58). As for pulpal pain, the prevalence may vary depending on various factors such as age, oral hygiene, socioeconomic status, and access to dental care. In countries with better access to dental care and oral hygiene education, the prevalence of pulpal pain may be lower compared to areas with limited resources. It's important to note that pulpal pain, such as pulpitis, can occur in individuals of any age. However it is more commonly seen in adults due to the cumulative effects of dental caries over time. Additionally, the prevalence of pulpitis can vary between different populations and demographic groups. The pain of TN is very sharp but episodic and can be aggravated by daily movements and routines or even a light touch to the affected site. However pulpal pain is a dull pain that can have specific trigger points, such as thermal triggers or percussion on the affected tooth, and can cause facial swelling.TN diagnosis stays clinical. Dental pain is normally provoked by percussion directly to the affected tooth or cold or hot application (6,16). Permanent or episodic pain that radiates toward the ear or eye can be a sign of TN. The most characteristic sign differentiating dental pain from TN is aggravation at night and sleep disturbances, while TN acts the opposite (17). Most of

the time, a dentist's injection of local anesthetics into the periodontal area may lead to pain relief. The local anesthetic injection on mucosa leads to numbness in the local area, which lasts until the effect of the anesthetic medication is gone. Thus, local anesthesia can relieve any pain but not TN pain. However, if local anesthesia is performed in the case of TN, only numbness would be present with no significant pain relief unless a nerve block is given instead of local anesthetics. One of the most diagnostic tests of TN is the positive or negative response to oxcarbazepine and carbamazepine (7). The pain relief is to the point that if the patient rejects any pain improvement by using carbamazepine at any time, the clinician should reconsider the diagnosis that was made (18). In many cases of TN, the pain relief moderately decreases over a period that may take months to years, and eventually, the medical management may fail (20). Many patients can have recurrent dental issues because of poor oral hygiene or other causes that can aggravate the characteristic signs of TN. It might be incredibly relatable because brushing can trigger severe pain in patients with TN. Among adults with continuous dental pain, mostly suffering from periodontal diseases rarely, TN can also pose a nuisance from trigger point areas in the mouth (6). Elicited allodynic pain has been found in reversible pulpitis and cracked tooth syndrome in response to percussion and cold stimuli, which can mimic TN. In these kinds of cases, a patient should always be examined by a dentist too and should get dental treatment in case of any apparent characteristics sign. Nonetheless, it is unfortunately common to see patients with dental-alveolar, non-odontogenic, or idiopathic orofacial pain getting treated with many different dental procedures, such as root canal treatments and tooth extractions which can often associate with temporary or partial pain relief (6,19,21).

In one study that has been done by de Siqueira *et al.* (10), which is almost 65 % of the 48 reviewed patients had undergone a total of 83 dental treatment procedures: thirty-nine experienced tooth extractions, thirty-two with single tooth extractions, and seven patients with multiple tooth extractions. Six patients had become edentulous due to dental treatments that had been done for TN. In this study analysis, intraoral trigger points for TN had no connection with dental procedures that have been done compared to extraoral trigger points. A very impressive finding was an analytically expressing correlation between some patients undergoing dental treatment procedures within the duration of TN, with 100 % of patients receiving some treatment and pain relief if the pain has occurred for more than ten years (10). Another study by von

Eckardstein *et al.* reported that forty-one patients, who count as 82 percent of the total 51 evaluated patients, had consulted their dentists initially. They received invasive dental procedures, including root canal treatments, extractions, and relative implants. Most patients in that study reported that their dentist could not distinguish the exact issue. Fifty-four teeth had been extracted in thirteen patients with at least two and a maximum of 20 extractions. Of all the patients that were referred to a dentist, 70.6 % had undergone extractions within four weeks of the onset of clinical signs. Still, more than two years passed before neurological or neurosurgical help-seeking. The authors had raised attention to the point that the critical difference in these studies done ten years apart was that fewer teeth (at least two vs. 10) were extracted (6). A recent survey done by Tripathi M et al. showed the same results as previously conducted studies [see Table 1]. About 65.8 % of the patients had appointed a dentist for their pains and clinical symptoms, and around 41.8 % had undergone some dental procedures during their appointments. About 19 % of patients reported that their pain progressed after dental treatment. Forty-nine patients had 78 teeth extracted, ranging from at least 1 to 4 in number, and the most extracted tooth was the first maxillary premolar (8).

Study	Patient with the extracted tooth	Number of extracted teeth
de Siqueira *et al.* (2004)	39 of 48 patients (81%) had extractions	Total of 83 dental extractions
von Eckardstein *et al.* (2014)	13 of 51 patients (25%) had extractions	54 teeth were extracted in 13 patients, and at least 2
Tripathi M et al. (2020)	49 of 117 patients (41.8%) had extractions	Extraction of 78, meaning 1.6 teeth extracted per patient

Table 1. Distribution of patients with trigeminal neuralgia undergoing teeth extraction.

The other case report by J Clin Exp Dent (22) shows the misdiagnosis of pulpal pain for a TN case. The dentist confused the clinical symptoms and the advanced anomalies of the tooth. Some points can explain these issues and could be of interest to practitioners. Symptomatology of the pain meets the diagnostic criteria for TN in the ICHD3 classification. Still, the patient was not in the usually affected group of women over 40 times old, which is in 90 of TN cases. It was

explained as electrical shocks, needles, and legs, severe mechanical allodynia, and critical neuropathic pain symptoms. Dental pulpal pain, as mentioned above, is generally described as palpitating, dull, and sharp (15), which reflects seditious processes in an enclosed area. Although neuropathic clinical symptoms similar to electric shock- suchlike attacks, firing, legs and needles, chinking, deadening, and burning, have also been reported at the same time, toothache shows that neuropathic mechanisms can commit to typical dental pain (25). Figure 1 and Figure 2 show the case report (22).

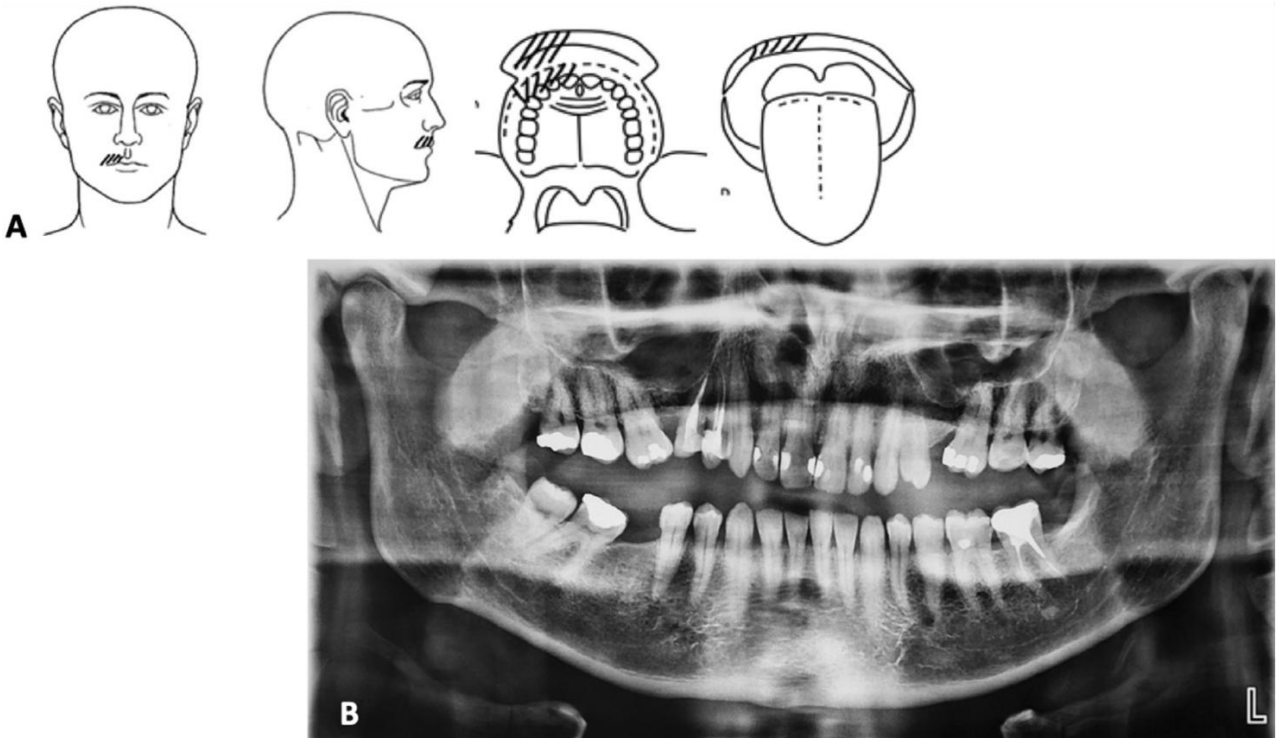

Fig. 1: A. Pain drawing showing the location of pain described by the patient. B. Panoramic X-Ray. Pay attention to the bilateral diastema between teeth numbers 16 and 15 and 24 and 26 with the mesial rotation of 24. Teeth 14 and 15 were endodontically treated many years ago (adapted from 22).

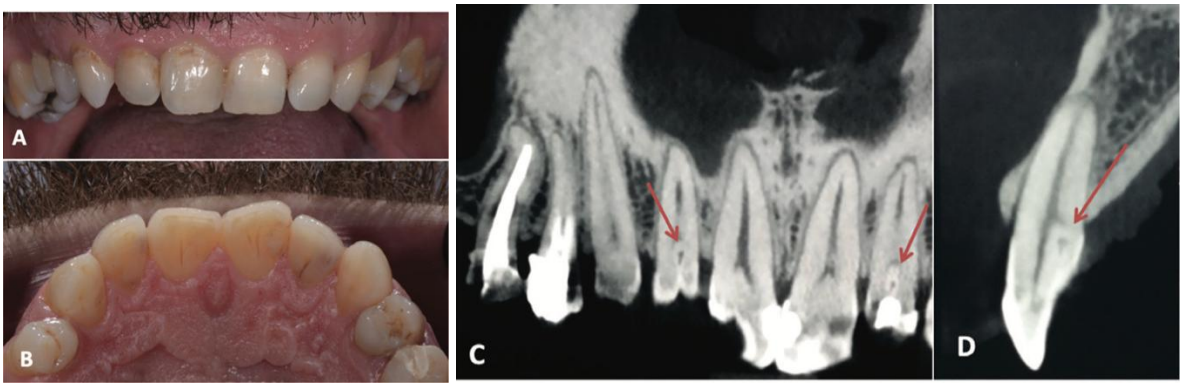

Fig. 2: Photographic view of buccal (A) and palatal (B) view of maxillary teeth. CBCT images C. frontal view, D. sagittal view of maxillary teeth (adapted from 22).

In summary, patients with TN may face pain attacks that can last from a few seconds to some minutes, and also, the series of TN attacks can be continued for days, weeks or months, and the progress of pain can be constant or get worse. The pain can be like jabbing or shooting pain or, as mentioned, "stabbing" or "electric shock-like" pain (20). TN pain attacks can be generated by brushing teeth, swallowing, talking, chewing, head movements, shaving or putting on makeup, or even a light touch. However, dental pain is a very dull pain, it can be sharp or shock, or shooting-like, but it has some different symptoms, or it is triggered by specific points like swelling around a tooth or swelling of your jaw, hot and cold sensitivity, bleeding or discharge from around a tooth or gums, dull pain with chewing and also injury or trauma to the area (25).

5.2 Trigeminal neuralgia and Toothache of periodontal origin

According to global estimates from the World Health Organization (WHO), severe periodontal disease affects 10-19% of adults, with over 1 billion cases worldwide. However, it is essential to note that these estimates may vary across different populations and geographic regions. In some populations, the prevalence of periodontal pain may be higher due to factors such as poor oral hygiene habits, tobacco use, systemic health conditions like diabetes, and genetic predisposition. There is a lack of articles or clinical cases of the misdiagnosis between periodontal pain and TN, as periodontal pain has a particular cause and symptoms and the clinical characteristics of the TN may not mimic the pain with periodontal origin.

5.3 Trigeminal neuralgia and myofacial pain

Myofascial pain is generally explained by dull, aching, and regional muscle pain and the localized trigger points in a tendon, muscle or fascia. The prevalence of myofascial pain in the general population is around 30% to 85%, and it is usually found in patients in the age gap of 27 to 50 years. The gender differences and muscular dental pain prevalence remains unclear (59). However, The symptoms are the most typical characteristics of muscle pain presentation, but there are some cases in pain severity and the quality of pain is similar to TN. The pain severity of myofascial pain attacks can be ranged from agonizing to some painless attacks. Sometimes the pain can be reported as high or even higher than pain with neurological origins. The pain in the myofascial region can be aggravated by mandibular function, which involves the masticatory muscles like the TN episodes that sometimes can show up by fascial and tongue movements or chewing (48,30).

5.4 Trigeminal neuralgia and sinusitis

Sinus pain can be shown as an ongoing dull pain in the area of maxillary teeth with sensitivity to mastication forces, thermal stimuli, and percussion (30). However, TN might be detected as a sharp, electric shock-like aching, differing from moderate to severe pain (31). The prevalence of sinus-related toothache or odontogenic sinusitis is 10 to 40%, with 11% of pain in the maxillary premolar and molar reports (60).

In a clinical case presented by Hyung-Joon Ahn et al., maxillary sinusitis was confused for TN due to the patient description of the pain. The patient reported electric-shock-like pain in the left upper premolar, triggered by brushing teeth and body movements, significantly lowering the head or cold wind. After taking the panoramic X-ray, dental X-ray, and oral examination patient was diagnosed with TN, and carbamazepine was prescribed (Figure 3). However, after two years, the patient returned with severe episodic pain attacks. At this time, a cone beam CT was performed, and left maxillary sinusitis was revealed (Figure 4). As a treatment, Endoscope-assisted surgery was performed, and the pain disappeared after a month (31).

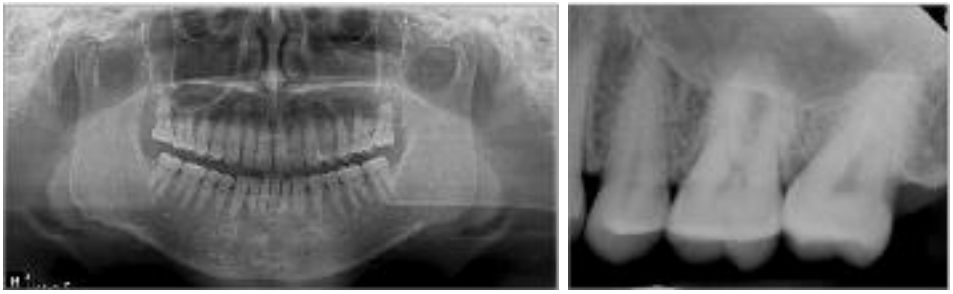

Fig.3. Panoramic and dental Xray that was performed in the first visit (Adapted from 31).

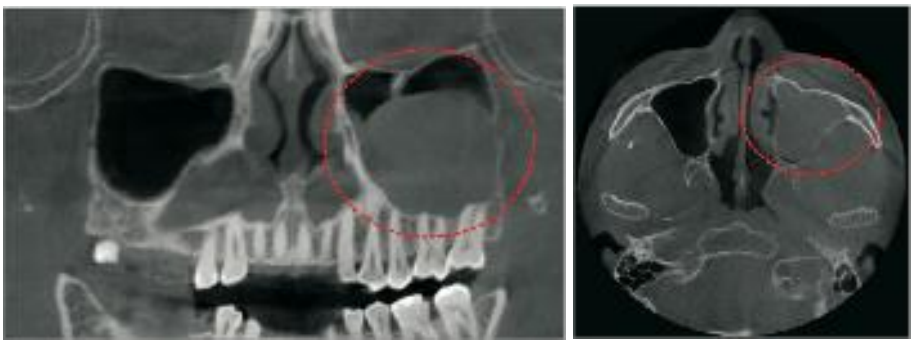

Fig.4. CBCT after two years. The circled area shows thickening of the mucosa in the left maxillary sinus (Adapted from 31).

Although, only a few reports describe a pain like TN for sinusitis (35). It may be explained by the anatomical variation of the canal in the bony wall of the sinus and the route of the posterior superior alveolar nerve (36). In two studies that have been done on dry skulls, around 17 to 20 % of cases showed fragmented canals along with superior alveolar nerve. So, the lesions in the maxillary sinus can demyelinate and press the nerve exposed to the sinus wall and mimic the pain like TN (36,37).

6. RESULTS

When the patient is experiencing the pain of TN, they usually need clarification about whether they have to consult with a doctor or a dentist. Usually, general practitioners or medical students struggle with diagnosing TN because they often do not get to think about the mouth and the structures around it in depth (43). Conversely, dentists need to gain deep knowledge of the biopsychosocial approach in the head and neck area. Every TN diagnosis will take a long way to reach the MRI and a definite diagnosis. Thus the diagnosis of TN may need a multi-professional team that consists of neurologists, psychiatrists, neurosurgeons, oral and maxillofacial surgeons,

and pain specialists, as the clinical management is different among all types of orofacial pains (47). The first line of treatment for TN will remain carbamazepine and oxcarbazepine, but the pharmaco-resistant cases may refer to a professional team of neurologists and neurosurgeons (57). According to this study, a table of the reviewed oral conditions was made, including the characteristics and treatment options for each pathology (ANNEX 1).

	Trigeminal neuralgia	Toothache of pulpal origin	Toothache of periodontal origin	Toothache of myofascial origin	Toothache of nasal/sinus origin
Epidemiology	Rare	Common	Common	Common	Common
Onset	Sudden	Spontaneous	Slow but steady	Acute or insidious	Spontaneous and sudden
Duration	Intermittent second to minutes	Seconds to daily	Variable	Second to hours	Variable
Periodicity	Refractory periods, day to months	Variable	Daily	Variable	Daily
Location	V2,V3 most common intraoral, extraoral	Tooth	Tooth/gingiva/bone	Facial muscle trigger points	Mid-face area or upper teeth
Character	Sharp, shooting, lightening, maybe a dull ache, burning after pain	Throbbing and sharp aching	Tender and aching Low intensity	Dull, deep hurting muscle pain	Aching dull pain
Severity	Moderate to severe	Mild to severe	Mild	Mild to severe	Mild
Aggravating factor	light touch, eating, some attacks are spontaneous	Electric and thermal stimulation, tooth percussion	Lateral and apical tooth pressure	Mandibular functions	Application of pressure on the affected sinus
Examination	Light touch, evoked pain, rarely sensory changes	Tender to percussion, caries, gingival swelling	Mobile teeth, erythematous gingiva, deep pockets	Tender to palpation in trigger points	Congestion or nasal drainage, pain elevation when the head is lowered
Management	carbamazepine/oxcarbazepine, neurosurgical procedures	Endodontic treatment/ extraction	Analgesic/injection of local anesthesia to the affected tooth region	Mild painkillers, stretch therapy, deep massages	Antibiotics with β-lactamase–resistant, decongestants or/and antihistamines

Annex 1. The main characteristics of the most common dental pain and trigeminal neuralgia with their management.

6. CONCLUSIONS

The pathophysiology of trigeminal neuralgia is primarily compression or irritation of the trigeminal nerve. The exact mechanism behind this compression is not fully understood, but it is thought to involve the blood vessels that come into contact with the nerve near its root entry zone in the brainstem. The compression can lead to demyelination and abnormal firing of the nerve. The clinical features are paroxysmal (sudden and recurrent) episodes of intense, stabbing, or shock-like facial pain. The pain is usually unilateral and localized to specific areas innervated by the trigeminal nerve, such as the cheek, jaw, teeth, gums, or less commonly, the forehead and eye region. The episodes typically last from a few seconds to a couple of minutes but can occur in rapid succession. The most common dental pathologies that can be mimicked by trigeminal neuralgia are pulpitis, odontogenic sinusitis and myofacial pain which their clinical characteristics and management options are mentioned shortly in ANNEX 1. Although the correct diagnosis of trigeminal neuralgia is still a clinical challenge for dentists and oral caregivers, it is very important to raise the awareness about the correct diagnosis of this pathology by providing the correct diagnostical methods followed by accurate clinical characteristics. Finally, the significance of a multi-professional team approach is to provide correct diagnosis accompanied by patient pain management.

7. ABBREVIATIONS

TN: trigeminal neuralgia, ICHD-3: International classification of headache disorders-3rd edition, CNS: central nervous system, PNS: peripheral nervous system, CT: computed tomography, TMJ: temporomandibular joint, IASP: International Association for the Study of Pain, SUNA: short-lasting unilateral neuralgiform headache attacks, SUNCT: short-lasting unilateral neuralgiform headache with conjunctival injection and tearing.

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