Original Article

Differential diagnosis of orofacial pain III: associated with neuropathic disorders

Diagnóstico diferencial del dolor orofacial III: asociado a desórdenes neuropático

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ABSTRACT

This is the last of three articles that have the purpose of presenting a literature review of the conditions that have been considered to establish the differential diagnosis of different orofacial pain conditions (OFP). In this article, neuropathic disorders both episodic (trigeminal neuralgia, glossopharyngeal neuralgia, occipital neuralgia) and continuos (neuritis, postherpetic neuralgia, deafferentation pain, and atypical pain) are discussed. Throughout these articles, the main clinical characteristics of the different orofacial pain conditions have been presented. Those characteristics are used to make a systematic comparison with the clinical finding of the patient, which will provide the basis to establish the correct differential diagnosis. Therefore, independent of knowing the treatment, the clinician should be familiar with as many clinical characteristics of the different orofacial pain conditions as possible.

Keywords: *pain, orofacial pain, differential diagnosis, headache, neuropathic pain, neuralgias, traumatic neuralgia.*

RESUMEN

Este es el último de tres artículos que tienen como propósito presentar una revisión de la literatura sobre las condiciones que se han considerado para el establecimiento del diagnóstico diferencial del dolor orofacial (DOF). En este artículo se discuten los desórdenes neuropáticos de tipo episódico (neuralgia trigeminal, neuralgia glosofaríngea, neuralgia occipital), y continuo (neuritis, neuralgia posterapéutica, dolor por deaferentación y los dolores atípicos). A través de estos tres artículos, las principales características clínicas de las diferentes condiciones de DOF han sido presentadas. Esas características se usan para realizar una comparación sistemática con los hallazgos clínicos del paciente, lo cual proveerá las bases para establecer el correcto diagnóstico diferencial. Por lo tanto, independiente de conocer el tratamiento, el clínico debe estar familiarizado con la mayor cantidad posible de características clínicas de las diferentes condiciones de DOF.

Palabras clave: *dolor, dolor orofacial, diagnóstico diferencial, dolor neuropático, neuralgias, neuralgia traumática, dolor atípico.*

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INTRODUCTION

This is the last article in a series of three describing the different pathological conditions that should be considered to establish the differential diagnosis of orofacial pain[1,2]. Episodic and continuous neuropathic disorders will be discussed.

NEUROPATHIC DISORDERS

Neurogenic or neuropathic pain is caused by functional disorders of the nervous system and the symptomatology indicates a structural abnormality in the peripheral or central nervous system. Neurogenic disorders can be classified according to the duration of the pain episodes: episodic and continuous.

Episodic or intermittent neuropathic disorders are characterized by the paroxysmal onset of pain. They are usually reported as "electrical shocks", which is an important aid in the diagnosis of this condition. This category includes neuralgias coming from different nerves (trigeminal, glossopharyngeal, etc.).

Continuous neuropathic disorders feature burning, sharp, dull, high or low intensity pain, but rarely with total relief of the painful symptomatology. This category mainly includes neuritis, postherpetic neuralgia, deafferentation pain and atypical pain.

EPISODIC OR INTERMITTENT NEUROPATHIC DISORDERS

Trigeminal neuralgia (TN)

TN is a rare condition, with an estimated prevalence of 1% in general population and an incidence of 4.3 per 100 000 people[3,4]. It occurs more in women than in men in a ratio of approximately 2:1 and there is a slight genetic association[3,4]. Onset age is generally after 20 years of age, reaching the highest incidence after the fifth decade. Pain paroxysms are very intense, 95% of the time are unilateral and although they can occur bilaterally, pain episodes do not occur simultaneously on both sides. Painful attacks have periods of remission, which vary from weeks to months, even years: this causes that in many cases patients easily forget previous painful episodes.

The pain is reported as "electric shocks", sharp, "stabbing-like" and usually lasts between 10 and 30 seconds; occasionally it lasts for more than a minute. Pain follows the distribution of the involved trigeminal branches, occurring more frequently in the maxillary and mandibular branches and with a slight tendency to be felt more often on the right side of the face[4,5]. Pain can be initiated by manipulation of a specific area of the face, which is called a "trigger zone" and it is usually located in the distribution of the involved nerve branch. The trigger zone may be located on the skin of the lips, cheeks, gums or other intraoral or extraoral areas. Activities such as talking, eating, whistling, shaving or simply rubbing the skin may be the onset factors of the painful episode. For this reason, it is common to find that patients point to the trigger zone, but avoid contact with it. Other activities such as a particular head tilt or lying in a specific position have also been identified as pain

onset factors[7,8]. After painful episodes there is a refractory period during which the trigger zone remains inactive. However, the patient may report a dull pain that usually lasts a couple of minutes and is apparently caused by the rigid protective reflex produced by the involved muscles. This reflex can be controlled by stretching exercises and the use of cold vapor sprays (fluor methane). On the other hand, it is to be noted that in the muscles there are certain spastic reflexes that accompany the pain on the affected side and hence it is called tic douloureux[9].

Table 1. Neuropathic disorders classification

Episodic (neuralgia - n)	Continuous
Trigeminal and pretrigeminal n Glossopharyngeal n Occipital n Other neuralgias • Sphenopalatine ganglion n • Facial nerve n • Intermediate nerve	Neuritis Postherpetic n Deafferentation pain • Posttraumatic n • Neuroma Atypical pain

Source: by the authors

Table 2. General clinical characteristics of neuropathic disorders

Episodic (neuralgia - N)	Continuous
Electric, sharp pain	Burning, dull pain
Unilateral	Uni or bilateral
Duration is short.	Pain is always present
Severe intensity	Mild to moderate intensity
Positive response to local anesthetics	Variable or equivocal response to local anesthetics

Source: by the authors

Occasionally, during a careful neurological examination, mild or moderate loss of sensation of the involved nerve branch (sensation that is not perceived by the patient) and/or corneal reflex dysfunction is found. However, if not present, this should not discourage the clinician from carefully looking for other signs. In some cases the patient may report certain premonitory symptoms just before the painful episode. These symptoms are displayed with tingling in the trigger zone and in this case an anesthetic block provides temporary pain relief while its effect lasts, thus being an important diagnostic aid.

During the onset of the painful episode, the condition can easily be mistaken for acute odontogenic pain[10,11], which is why many patients receive unnecessary dental treatment. This situation occurs especially when there is a state of "pretrigeminal" neuralgia[12,13], which is a painful prodrome of typical trigeminal neuralgia. This can last from weeks to years and is characterized by the presence of dull, burning, sharp pain, moderate or severe, usually located in a small area of one of the alveolar quadrants and base of the tongue, creating confusion in patients, who refer to the condition as toothache or sinus pain[12,13].

Table 3. General characteristics of episodic neurogenic disorders

Neuralgia types	Trigeminal	Glossopharyngeal	Occipital
Average onset age	50 years	50 years	35 years
Pain type	Electric	Electric	Sharp
Predominant gender	Women 3 to 2	Not sharp	Slightly more frequent in women
Location	Unilateral	Unilateral	Uni or bilateral
Pain triggering factors	Trigger zones palpation	Swallowing, mastication	Hyperextension of the head
Associated symptoms	Localized loss of sensibility	Throat clearing	Limitation of neck movement
Drugs used as diagnostic treatment	Carbamazepine (Tegretol)	Carbamazepine (Tegretol)	Local anesthetics with corticosteroids
Response to blocks with local anesthetics	Positive	Positive	Positive

Source: by the authors

TNs have been divided into idiopathic (there is no evidence of a structural abnormality) and symptomatic (its etiological source are factors such as compression of the trigeminal nerve by arteriovenous vessels, acoustic neurilemoma, aneurysms and multiple sclerosis (MS) among others)[14-18]. One of the conditions frequently associated with symptomatic TN is MS. It is estimated that 1% of MS patients present TN, while 3% of TN are caused by MS[15]. On the other hand, it has been established that some brain tumors may resemble the symptomatology of typical TN, with positive responses to baclofen and/or carbamazepine[13]. For this reason, diagnostic imaging should be considered, especially in young patients[15,16].

Glossopharyngeal neuralgia (GFN)

GFN is less frequent than TN and occurs with an estimated incidence of 0.7 per 100.000 inhabitants, with a sex- and age-adjusted ratio between TN and GFN of 5:9:1[3,19,20]. However, estimated values of occurrence ratios vary from 1 to 13 in GFN per 75 to 100 cases of NT[20-21]. Similar to TN, NGF appears after the second decade and with an onset peak after the age of 50 years[16-19]. Certain differences between these 2 neuralgias can be highlighted: in GFN there is no differentiation between sexes and there is predominance of lateralization on the left side. In addition, it should be mentioned that in a small number of patients (8%) TN and GFN can occur simultaneously[20-22].

Pain is similar to the one reported in TN, but it generally displays less intensity and a longer duration in the episode following the painful paroxysms. These episodes are usually accompanied by sensations of clicking, throat clearing or foreign body sensation in the throat. Pain localization is more diffuse and involves, in decreasing order of appearance, the ear, pharynx, palatine tonsils, base of the tongue, ear canal, throat, lower neck and post mandibular area. Trigger points are less frequently reported because they are possibly located deep in the mouth, pharynx and ear. However, activities such as swallowing, chewing, conversation and the ingestion of cold drinks and spicy foods have been described as precipitating factors. Anesthetic blocks in the trigger zones can help to establish the diagnosis, for this reason a relief of more than 2 hours after the application of topical anesthesia (cocaine) in the tonsils or pharyngeal areas is considered an important diagnostic aid in the assessment of GFN. It is therefore fair to recognize that some associated symptoms such

as cough or bradycardia, followed by syncope or convulsions, may also manifest in this type of patient (2%)[20-22]. This is related to the fact that GFN is frequently accompanied by vagus nerve involvement and hence this condition has also been called Vagoglossopharyngeal neuralgia.

Likewise, GFN has been also divided in idiopathic and symptomatic. Symptomatic GFN is associated with tumors, infections and vascular lesions, and should be especially differentiated from Eagle's and Trotter's syndromes, which are causes to a compression of the throat tissues by an elongated styloid process and to a tumor growth in the nasopharynx, respectively. In the latter case, the pain is referred to the jaw, tongue and lateral area of the head. Moreover, there may be secondary deafness due to closure of the Eustachian tube and/or asymmetry in the movement of the soft palate[23].

Occipital neuralgia (ON)

ON may appear with intermittent, "stabbing-like" and sharp pain, simultaneously accompanied by a continuous dull aching sensation, usually located unilaterally over the upper nape of the neck and radiating to the orbital, retro-orbital, frontal, auricular, mandibular angle and/or upper neck and shoulders[24-26]. Patients frequently report changes in skin sensation, decreased neck rotational movement and other associated symptoms such as nausea, vomiting, nasal congestion, vertigo and visual disturbances. Trigger points are not present, but due to the similarity of symptomatology, this condition is easily confused with posterior neck area myofascial pain (MFP) and can be misdiagnosed, especially when there is referred pain to the orbital area (from the neck muscles). Therefore, a good differential diagnosis is important, especially if surgical therapy is going to be performed.[27]

Other neuralgias

Other less frequent neuralgias also must be considered. The description of pain is quite similar; however, the location varies according to the nerve involved.[28-29] Sphenopalatine ganglion neuralgia (SGN)[30,31] displays piercing pain, of variable duration, located unilaterally in the orbit, base of the nose and/or mastoid process region. Occasionally it involves the neck, moves to the shoulder area following the path of the elbows and finally reaching the fingers. During pain attacks, some autonomic symptoms such as swelling of the nasal membrane, nasal drainage and lacrimation may be associated.

Table 4. General characteristics of the main continuous neurogenic disorders

Condition	Neuritis	Postherpetic neuralgia	Post-traumatic neuralgia	Neuroma	Atypical pain	Glossodynia	NICO
Average onset age	Variable	Fifth decade	Variable	Variable	Fifth decade	Fifth decade	Fourth decade
Pain type	Burning	Burning	Burning and sharp	Sharp	Variable	Burning	Variable
Predominant gender	It does not exist	Women 3 to 2	Variable	Variable	Women	Women 5 to 1	It does not exist
Location	Variable	Uni or bilateral	Unilateral	Variable	Unilateral	Bilateral	Unilateral
Pain worsening or triggering	Mechanical stimulation	Skin rubbing	Cutaneous stimulation	Pressure	Variable	Variable	None

factors							
Associated symptoms	Paresthesia, anesthesia	Sensitivity changes	Hyperesthesia	Dysesthesia	Nonspecific	Dry mouth, taste disturbances	None
Response to blocks with local anesthetics	Positive	Negative	Positive	Positive	Confusing	Negative	Positive

Source: by the authors

An anesthetic block of the sphenopalatine ganglion, via the nasal route, is definitive to establish the diagnosis; a negative result should lead to the search for another related pathology. Because of the similarity with cluster headaches (CH), some authors consider SGN to be a variant of this disorder and only when medical treatment for CH has failed should the SGN diagnosis be considered[32]. Vidian neuralgia[31] (VN) is considered a variant of SGN and because of their similar clinical features, it is reckoned that both conditions correspond to the appearance of the same disorder, associated with the sphenopalatine ganglion region and adjacent structures[31-32]. Facial nerve neuralgia (FN) is characterized by unilateral paroxysmal pain accompanied by spasm in the face muscles, which makes it be easily confused with TN. This has also been described as intermediate nerve or geniculate ganglion neuralgia[33] and is characterized by paroxysmal, piercing pain in the ear area that may involve the tympanic membrane, walls of the ear canal, external auditory meatus, and adjacent structures. Some nasal, lacrimal, salivary and taste disturbances may be reported during painful attacks that can last from seconds to a couple of minutes. Upper laryngeal nerve neuralgia[34] displays clinical features similar to those of SGN, causing piercing, paroxysmal pain of momentary duration, located unilaterally in the submandibular region and anterior part of the sternocleidomastoid muscle. This pain commonly radiates to the eyes, ears, palate, cheeks and shoulders. Pain onset factors include swallowing, voice straining (shouting, singing), head rotation, coughing, sneezing, yawning and nose wiping. Some trigger zones can be found within the anatomical distribution of the nerve, especially in the anterolateral neck area. Because this nerve is responsible for the motor innervation of the vocal cords, the patient may have hoarse or weak voice accompanying pain episodes. Nasal nerve neuralgia[35] is characterized by a sharp paroxysm of severe pain, localized between the inner corner of the eye, root and nostril, which may be accompanied by redness of the eye, ipsilateral profuse nasal discharge, and congestion. Nowadays, this condition is included as a variant within CH diagnosis.

In general, most neuralgias respond positively to non-surgical treatment with carbamazepine or baclofen. In cases where results are not positive and there is no change in the patient's symptomatology, the clinician should consider other conditions within the differential diagnosis.

CONTINUOUS NEUROPATHIC DISORDERS

Neuritis

This condition occurs as a result of an alteration in the afferent fibers of a nerve branch and is presumably the result of an inflammatory process usually caused by trauma (laceration), bacterial/viral infection or toxic substances[36-38]. The pain is described as burning, stimulating, and is localized precisely at the site of the involved nerve branch. Pain intensity is temporarily persistent

according to the duration of the process causing the inflammation, and increases, although not necessarily proportionally, with mechanical stimulation. Other symptoms associated with neuritis will depend on the type of nerve involved. When a sensory nerve is affected, symptoms such as paresthesia, anesthesia, hyperesthesia, hypoesthesia and dysesthesia can appear along the nerve distribution. Symptoms such as paralysis, muscle weakness and tics accompany motor nerve neuritis. If there is involvement of nerve fibers of the autonomic system, there might be changes in temperature, color, swelling, tearing, congestion and nasal secretion, among others. Any nerve can be involved, and the diagnosis of neuritis essentially requires a good understanding of neuroanatomy and an adequate association of the features found in the medical records and physical examination.

Trigeminal nerve neuritis is usually caused by trauma or viral infection. Therefore, it is not uncommon to find that after exodontia of impacted third molars or root canals (involving alveolar nerves, lower or upper), patients display paresthesia, anesthesia of the lower lip or other symptoms localized on the previously treated tooth (or supporting tissues), which differ from typical odontalgia. [28,36-38] Another type of neuritis frequently found in the trigeminal nerve is the one caused by viral infection due to herpes and varicella-zoster virus (VZV), which is characterized by a scalding, burning pain, usually followed (one week after the onset of painful symptoms) by vesicles that appear along the involved trigeminal branch (the ophthalmic branch is the most common one) and that will corroborate the diagnosis. Approximately 20% of VZV neuritis develops into a chronic pain condition called postherpetic neuralgia, which will be discussed later in this section.

Glossopharyngeal nerve neuritis[23-28] causes sore throat and appears in post mandibular and auricular areas, which is worsened by throat and/or jaw movements. These characteristics make it be easily confused with pain of the masticatory apparatus. A frequent reason for neuritis caused by trauma is elongation of the stylohyoid process or calcification of the stylohyoid ligament, commonly known as Eagle's syndrome[39]. Facial nerve neuritis (Bell's palsy)[28] features burning pain involving the auricular area, accompanied by marked unilateral weakness or paralysis of the facial muscles. Simultaneously, autonomic sensory symptoms and changes in taste, among others, may occur.

Inflammation of the ocular, trochlear or abducens nerves is associated with Tolosa-Hunt syndrome[40-41]. This is a poorly understood condition and usually associated with inflammation of the structures related to the cavernous sinus. In its initial phase it causes intermittent pain that progressively becomes constant, intense, stabbing and piercing in the orbital, frontal or temporal region, with ophthalmoplegia (inability to move the eye): This condition is generally self-limiting, lasts approximately eight weeks, and has a good response to corticosteroid treatment. Other similar conditions also associated with ophthalmoplegia, such as optic neuritis, should also be ruled out. This is mainly characterized by being accompanied by a decrease in visual acuity.

Environmental neuralgias are caused by toxic agents[42] such as heavy metals (mercury, arsenic, etc.), certain drugs (vincaalkaloids, nitrofurantoin, etc.) or other adverse stimuli (excessive noise or prolonged vibration). In this type of conditions it is common to find associated symptoms such as skin changes, gastrointestinal disturbances and/or neurological changes. For the diagnosis it is important to evaluate the patient's habits and lifestyle, type of work and area where they live as

this might reveal the presence of some environmental factor causing the neuralgia. The elimination of the factor itself is a fundamental part of treatment.

Postherpetic Neuralgia (PHN)

One of the complications in neuritis caused by herpes zoster (acute state) is to evolve into a chronic condition or PHN, which can occur weeks after the first viral eruption. This condition can affect any body area and has an estimated incidence of 40 to 160 per 100 000 person-years in people under 20 years of age and 450 to 1100 in 100.000 person-years in adults[43,44]. Its frequency of occurrence increases in adults generally after the sixth decade, with a slight tendency to occur more in men than in women. Approximately 10 to 30% of acute cases progress to PHN, and 10 to 20% affect the cranial nerves: the first division (ophthalmic) of the trigeminal nerve is the most frequently affected and involves the cornea and frontal area of the face. Diagnosis is relatively easy and is basically made using the pain-related history and vesicular eruption, which may leave scars on the patient's face, and sometimes analgesia and hyperesthesia occur in the involved areas. Prognosis is not very good and the literature reports that drugs such as prednisolone, acyclovir, or amantadine, while they do not reduce the probability of generating PHN, can accelerate the healing process, shortening the painful stage.[45]

Deafferentation pain

These types of conditions are not unusual in the orofacial region and given their difficult management they become very relevant disorders. Deafferentation means loss of normal afferent information to the central nervous system[28]. This category includes post-traumatic neuralgia (including pain maintained by the sympathetic system) and atypical pain (facial and dental).

Post-traumatic neuralgia (PTN) is a condition that appears after aggressions to peripheral nerves caused by cut, laceration, surgeries (orthognathic, dental extraction, pulp removal or dental implants) or accidental trauma. This situation can onset several symptoms such as anesthesia, hypoesthesia, paresthesia, dysesthesia, hyperesthesia, hyperalgesia, spontaneous pain, and other autonomic symptoms such as swelling, changes in skin color and/or temperature[46-49]. The pain in PTN is characterized by the combination of constant neuritis pain, interrupted by the sudden painful paroxysms of neuralgia. This combination is the reason why some authors call it atypical trigeminal neuralgia (when it involves the V pair). There is great difficulty in classifying these disorders because the symptoms do not constitute a stereotyped condition. On the contrary, it shows different responses of the nervous system depending on the components involved. Gregg[50] based on a study of 84 patients with trauma history on the maxillofacial region, divides these disorders into four categories. These categories include painful anesthesia (PA), hyperalgesia (HA), hyperpathia (HP) and sympathetic mediated pain (SMP). PA is characterized by pain in an area that the patient refers to as "anesthetized" or deprived of all sensation. In HA (synonymous with allodynia and hyperesthesia) the patient experiences pain sensation in response to stimuli that are not normally painful. PH is a delayed pain response to a pressure stimulus in the nerve distribution of the involved nerve, which then increases rapidly, reaching a high intensity and occurs in addition to "shooting pain". SMP [51,52] (synonymous with causalgia) includes causalgia disorders and reflex sympathetic dystrophy. SMP is defined as pain resulting from nerve damage, which is worsened by

increased sympathetic system tone, cold or emotional stimuli and is reduced by the use of alpha-adrenergic or stellate ganglion blockade. This painful condition is usually reported in the limbs and although infrequently, it has also been reported to occur in the orofacial region[52]. Pain is characterized as burning, continuous or hot and is not limited to the area of the involved nerve. It is often associated with hyperesthesia and is exacerbated by movement, cutaneous stimulation, stress and relieved by immobility. Certain physical signs of vasomotor and/or sudomotor dysfunction (vasodilatation) are frequently present, with increased temperature, edema and hyperhidrosis, skin atrophy, coldness and redness, among others 28. In a second research study, Gregg[53] reported the correlation between anomalies found in the nerve branches during microsurgical intervention and the appearance of the different clinical conditions with which the patients were diagnosed. Some of these anomalies are foreign bodies, extraneural abnormalities, collateral nerves and traumatic neuromas, the latter being the most frequently found within all categories.

Traumatic neuromas[54,55] are nerve tissue masses formed in an attempt to regenerate a peripheral nerve after receiving an aggression or a total or partial cut. The pain is described as deep, dull, burning and may be induced by compression or stretching of the neuroma. Additional symptoms such as dysesthesia or abnormal sensation accompanied by numbness may be associated with pain. Although neuromas have been considered as a separate condition with its own characteristics, they can be associated with different symptoms and may not necessarily be an isolated condition[53]. An injection with local anesthetics at the site of the neuroma relieves pain while its effect lasts and becomes a key tool for establishing the diagnosis[28,54,55], especially when placed in the area sensitive (painful) to palpation of the neuroma. The response to local anesthetics of PTN and traumatic neuromas depends on the involvement of the sympathetic system, psychological factors or factors of central origin.[49,50,53]

Atypical Pain (AP) (Facial and dental)

AP [56-61] refers to idiopathic pain symptoms that do not correspond to any of the above categories or to the anatomical distribution of a nerve branch and are sometimes also considered a deafferentation problem. Symptoms may have characteristics of vascular, muscular and/or neurogenic pain, but with generally vague descriptions and changes in anatomical location. Once other OFP disorders have been excluded, the search for specific evidence to support the diagnosis of AP should be pursued. Atypical facial pain (AFP) occurs more frequently in women (in a ratio of 6 to 1) and the onset age is usually in the fifth decade[60,61]. AFP occurs unilaterally (70% of cases) with diffuse, deep pain, with intensity varying from moderate to severe. The type of pain may vary between dull, bothering and sharp. Duration may be intermittent or continuous throughout the day, it has spontaneous onset and does not interfere with sleep. Allodynia, hyperalgesia, or dysesthesia occur sometimes, there are usually no worsening factors and trigger points are rare; but when they do occur, they are multiple and varied[60-61]. Edema, erythema (reddened oral mucosa) and increased temperature are reported by some patients and the onset may be associated with a history of trauma (exodontia, accidents, sinus surgery)[60,61]. Management therapies are generally unsuccessful, and some patients even report a significant increase in pain (especially if the therapy is surgical). Unfortunately, in many cases the AP diagnosis is established when there are repeated failures in clinical treatment, because the expected relief is not achieved, or because the results are

confusing due to the use of analgesics (including narcotics), nerve blocks, surgery or psychotherapy.

In many cases, the AP diagnosis becomes a "disposal bin" for those patients whose clinical treatment has not had satisfactory results or when the clinician is unable to establish an adequate diagnosis.[60-61] Blasberg[59] conducted a study in 102 patients referred to a specialized pain treatment clinic with AP diagnosis (given by physicians and dentists). After being evaluated by the specialist of that clinic, the examination could determine the specific cause of pain complaint and maintain the AP diagnosis only for 14.7% of patients (15). Others estimate that in specialized pain clinics, only 5% of patients are diagnosed with AP[62].

Some authors have suggested that certain psychological aspects could play a role in the case of these patients[62,63]; however, this point is controversial because some researchers have reported that there are no differences in the levels of depression and/or anxiety when compared with other chronic pain conditions[64]. Burning mouth syndrome is perhaps a variant of AP and it has been generally established that in order to consider BMS as an atypical pain, its diagnosis must be made after ruling out the myriad causes that may generate burning mouth symptoms. It should also be remembered that AP is idiopathic[60,61].

Atypical odontalgia (AO) is a variant of atypical pain and its general characteristics are similar. Different names have been used to refer to AO, including: phantom tooth pain, atypical facial neuralgia, migrainoid neuralgia and idiopathic tooth pain. This variation has ultimately contributed to the confusion that exists regarding this condition.[65-69].

The diagnosis of AO is usually established after the repeated implementation of unnecessary dental treatments (exodontia, root canals), which in some cases temporarily work, creating even more confusion for the treating clinician and the patient.

Suggested diagnostic criteria for AO are[70] constant pain in a healthy tooth without an obvious cause or local pathology; normal radiographic findings; repeated therapies without major success (root canal therapy); pain lasting 4 months or more, which may improve or worsen over time; and associated hyperesthesia. Relief with nerve blocks often has equivocal results.

A condition that should be considered whenever AP is suspected is neuralgia-inducing cavitational osteonecrosis (NICO).[71-74] NICO generally occurs between the ages of 35 and 65 years with no gender predilection. Pain progresses slowly increasing in intensity, frequency and extension of the involved area, which is usually reported as episodes of severe neuralgic pain of variable duration. The most involved teeth are the posterior teeth, especially the mandibular teeth. Therefore, there is a history of trauma or surgical or endodontic treatment. The use of anesthetic blocks, depending on the location of the lesion, may aid in the diagnosis. In the area of pain, radiographically, cavities or radiolucent shadows or areas of metabolic activity can be found in 80% of cases with the use of bone scintigraphy. Although the pathophysiology of this condition is not clear, it appears to be caused by coagulation disorders, which interfere with normal bone healing, causing an ischemic osteonecrosis that can be revealed by a biopsy.

DISCUSSION

Diagnosis is the foundation of clinical treatment and consists of determining what diseases or disorders are causing pain to the patient, with the respective contributing factors. An accurate diagnosis is the best tool to be successful in treating patients suffering from orofacial pain. Thus, it is necessary to know the clinical characteristics of the pathologies that occur in this region. Using this information, a systematic individualized comparison is made with the clinical characteristics of the patient, which will offer the first hypotheses within the differential diagnosis. It is necessary for the clinician to approach the whole process with shrewdness, experience, knowledge and clinical judgment: using an inferential and deductive reasoning model. This reasoning consists of ruling out diagnoses that do not apply to the patient, leaving aside the diagnostic options that the clinician prefers. It is important to note that it is difficult to diagnose something that is not known and although "common things happen normally" it is not uncommon to find patients with "rare" pathologies, of little occurrence, that the clinician has forgotten or does not know about. Failure of the clinician to consider all these aspects during the clinical evaluation can easily lead to confusion, which will ultimately point to misdiagnosis, clinical mismanagement and possibly disastrous consequences for patients. Therefore, regardless of whether or not the clinician is familiar with the treatment of the pathologies, they should be familiar with the clinical characteristics corresponding to each possible pathology that may affect the orofacial region. Finally, it is advisable to keep in mind that, due to the complexity of this field, the dentist often requires to consult with other colleagues in health areas such as neurologists, ENT specialists, psychiatrists, psychologists and physiotherapists, among others. Oftentimes patients receive adequate care and treatment only as a result of the simultaneous collaboration of different specialists.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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