Views and Perspectives

Atypical Odontalgia: A Review of the Literature

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Objective.—To review previous reports of cases of atypical odontalgia to examine its epidemiological and clinical characteristics and to explore the etiology and pathophysiology of the disease.

Background.—Atypical odontalgia is one of many painful conditions that affect the oral cavity and is often overlooked in the differential diagnosis.

Methods.—A search of the literature was performed for all cases of atypical odontalgia reported from 1966 to the present.

Results.—The typical clinical presentation of atypical odontalgia that has been reported involves pain in a tooth in the absence of any sign of pathology; the pain may spread to areas of the face, neck, and shoulder. The existing literature suggests that this condition occurs in 3% to 6% of the patients who undergo endodontic treatment, with high female preponderance and a concentration of cases in the fourth decade of life. Deafferentation seems to be the most likely mechanism to initiate the pain, but psychological factors, alteration of neural mechanisms, and even an idiopathic mechanism have been implicated. Not all reported cases were preceded by trauma to the teeth or gums.

The treatment of choice is a tricyclic antidepressant, alone or in combination with a phenothiazine. The outcome is usually fair, with many patients obtaining complete relief from pain. Especially in the absence of overt pathology, particular attention should be paid to avoiding any unnecessary and potentially dangerous dental intervention on the teeth.

Conclusion.—Atypical odontalgia is surprisingly common, of uncertain origin, and potentially treatable.

Key words: atypical odontalgia, phantom tooth pain, deafferentation pain, neuropathic pain, atypical facial pain

Abbreviations: AO atypical odontalgia, CNS central nervous system

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Atypical odontalgia (AO) is probably one of the most frustrating conditions that challenge dental clinicians. It was reported for the first time by McElin and Horton in 1947, and since then there have been many clinical reports in the literature, especially in relation to endodontic treatment.[1]

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It presents as tooth pain or pain in a site where a tooth was extracted, in absence of clinical and radiographic evidence of tooth pathology. Unfortunately, the occurrence of AO is common; it occurs in 3% to 6% of patients who undergo endodontic treatment.[2,3] There is a female preponderance with a concentration of cases of women in their mid 40s.[2-18] Except for children (no reports have been found in the literature), all ages can be affected. Molars and premolars are more frequently involved,[10,15,18-21] with the maxilla being affected more often than the mandible.[10,15,18,19]

DIAGNOSTIC CRITERIA

To date, there are not universally accepted "official" classification and diagnostic criteria for the

diagnosis of AO; yet, many have been proposed. According to the "Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain" of the International Headache Society (IHS),[22] AO is included, together with atypical facial pain, in diagnosis 12.8 "facial pain not fulfilling criteria in groups 11 and 12" (11: "headache or facial pain associated with disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cranial structures," 12: "cranial neuralgias, nerve trunk pain and deafferentation pain").

It is a diagnosis of exclusion, based on ruling out all other pathologies that originate from the teeth and adjacent structures. The diagnostic criteria are listed in Table 1. In the "Comment" below the diagnostic criteria listed by the IHS, it is specified that "Pain may be initiated by operation or injury to face, teeth or gums but persists without any demonstrable local cause."

The American Academy of Orofacial Pain separates AO from facial pain corresponding to diagnosis 12.8, stating that advancements in understanding neuropathic pain allow us to better explain conditions such as AO, and avoid including it in the "waste basket" of "facial pain not fulfilling criteria in groups 11 and 12."[16]

Graff-Radford and Solberg suggested instead a different collocation of AO (they proposed the term *idiopathic toothache*) in the IHS classification, within the diagnosis 11.6, headache or facial pain associated with disorder of "teeth, jaws and related structures."[19] They suggested expanding this classification to:

11.6.1 Pulpitis

11.6.2 Periodontitis

Table 1.—Atypical Odontalgia: Diagnostic Criteria*

12.8 Facial pain not fulfilling criteria in groups 11 and 12
A. Is present daily and persists for most or all of the day.
B. Is confined at onset to a limited area on one side of the face. May spread to the upper or lower jaws or a wider area of the face or neck. Is deep and poorly localized.
C. Is not associated with sensory loss or other physical signs.
D. Laboratory investigations including X-ray of face and jaws do not demonstrate relevant abnormality.

*Data from the Headache Classification Committee of the International Headache Society.[22]

Table 2.—Atypical Odontalgia/Idiopathic Toothache: Proposed Diagnostic Criteria of Graff-Radford and Solberg[19]

11.6.1 Idiopathic toothache (atypical odontalgia)
A. Pain in a tooth or a tooth site.
B. Continuous or almost continuous pain.
C. Pain persisting more than 4 months.
D. No sign of local or referred pain.
E. Equivocal somatic nerve block.

11.6.3 Dentinal

11.6.4 Cemental

11.6.5 Idiopathic toothache (atypical odontalgia)

They also suggested the diagnostic criteria reported in Table 2. Those criteria were followed by Okeson in his symptom-based classification of orofacial pains, including AO in the category of deafferentation pain, a subcategory of continuous neuropathic pain.[23]

In 1993, Marbach introduced other diagnostic criteria based mainly on the clinical characteristics of the pain (Table 3), and called AO *phantom tooth pain* (PTP).[24] A few years later, he stressed again points 5, 6, 7, 8, and 10 from his 1993 classification (see Table 3) as diagnostic criteria for AO.[25] In a recent review, Marbach and Raphael presented a revision of the criteria with the purpose of aiding differential diagnosis rather than describing the syndrome (Table 4).[26] Merskey and Bogduk, in the *Classification of Chronic Pain*, defined AO as "severe throbbing pain in the tooth without major pathology."[17] They also introduced these simple diagnostic criteria: "patient with history of tooth pain associated with endodontic therapy and/or extractions," and "remaining teeth while clinically sound and vital are tender to thermal stimuli and to percussion."

In 1995, Pertes and colleagues revised Graff-Radford and Solberg's criteria (Table 5); what is notable is the inclusion of the nonresponsiveness of the pain to treatments (point 9).[21]

All the above-mentioned criteria for the diagnosis of AO differ in the details that are included by some authors yet overlooked by others; this results in slight variations in the precision of the differential diagnosis. Yet, it seems clear that AO is characterized by chronic pain that is usually continuous, and clinical,

Table 3.—Atypical Odontalgia/Phantom Tooth Pain (PTP): Proposed Diagnostic Criteria of Marbach[24]

1. The onset of pain is usually associated with an injury to a peripheral nerve. The injury often occurs in the course of routine dental and medical surgical procedures. Injuries also occur as the result of physical trauma to the face.
2. The onset of the pain does not necessarily coincide with deafferentation at the tooth site. Pain may be delayed for days, weeks, months, or perhaps years.
3. The pain may endure long after healing of the injured tissues and spread to the adjacent healthy tissue. Spreading can follow synaptic reorganization of an injured afferent nerve with resulting structural and functional changes in associated areas.
4. PTP is more likely to develop in patients who have suffered pain in the tooth or face in the period immediately before the peripheral nerve section or endodontic treatment.
5. The pain is described as constant, dull, deep ache with occasional spontaneous sharp pains. There are no refractory periods.
6. Sleep is undisturbed by pain. Many cases report a brief pain-free period upon awakening. This period lasts from seconds to about 1 hour.
7. Peripheral stimuli can momentarily exacerbate the pain but have no prolonged influence. Percussion over the site of the injured nerve may result in Tinel's sign.
8. These stimuli can be of a type normally not nociceptive. There appears to be a lowered pain threshold (allodynia).
9. The pain is often worse at the site of the original trauma; although in chronic cases patients have difficulty in localizing the pain, which is in part the result of pain spreading to the adjacent tissues. Additionally, precise localization of tooth pain is difficult. The treatment of neighboring teeth obscure the original condition. Non-painful phantom phenomenon also confound accurate perception and localization of the pain site.
10. Radiographic and laboratory tests are negative.
11. Without early intervention, the pain is often permanent once it is established.
12. PTP occurs in both sexes.
13. PTP has been reported in adults but not in children.
14. There is no evidence currently that PTP is characterized by a premorbid personality. Whether affective states such as major depression are a cause or a consequence of chronic pain remains to be determined.

radiographic, and laboratory examinations that fail to reveal any organic pathology in the area where the pain is felt.

Our suggestion for the classification of AO is that it could be included in the IHS 12.1 classification "persistent (in contrast to tic-like) pain of cranial nerve origin," subdiagnosis "cranial neuralgias, nerve trunk pain, and deafferentation pain." The simplest diagnostic criteria, that include all the information needed for a correct diagnosis, are the criteria proposed by Pertes and colleagues.[21] They comprehend the clinical description of the disease (presence or absence of signs and symptoms), response to diagnostic tests (radiographs, local anesthetic injection), and response to treatment (analgesics, surgery, dental procedures), neglecting other features that may be present, but are not essential for the diagnosis. Furthermore, these criteria do not assume that we know the etiology of AO, leaving the question open to future research as hypotheses remain different and controversial.

Table 4.—Atypical Odontalgia/Phantom Tooth Pain (PTP): Revised Criteria of Marbach*

1. Pain is located in the face or described as a toothache.
2. The pain is described as a constant dull, deep ache. (Less than 10% of sufferers report occasional spontaneous sharp pains that overlay the ache. Sharp pain is not essential to meet criteria.)
3. A brief (seconds to minutes) pain-free period is reported upon awakening from sleep. There are no other refractory periods.
4. Pain develops (or continues) within one month following endodontic treatment or tooth extraction, or other trauma or medical procedure related to the face.
5. Overlying the area of dental (or other) treatment (usually on the surface of the face but occasionally intraorally) is a location with a much lowered pain threshold (hyperalgesia), often surrounded by a larger area with less severe hyperalgesia.
6. Sleep is undisturbed by pain or other phantom sensations.
7. No radiographic or laboratory tests suggest other sources of pain.

*Data from Marbach and Raphael.[26]

Table 5.—Atypical Odontalgia: Proposed Diagnostic Criteria of Pertes et al[21]

1. Aching, burning or throbbing.
2. Moderate intensity.
3. Continuous or almost continuous pain.
4. No obvious local cause.
5. Normal radiographs.
6. Pain present more than 4 months.
7. Increased sensitivity to pressure.
8. Somatic block equivocal.
9. Non-responsive to analgesics, surgery and dental procedures.

PATHOPHYSIOLOGY

The mechanism through which pain is generated in AO is far from being clear. Some authors suggest an idiopathic origin of this condition because no other hypotheses have proven responsible for precipitating and perpetuating the pain,[20,27,28] while others propose a psychogenic origin of the disease highlighting the evidence of association between AO and several psychological conditions.[7,8,10,11,18,24,26,29,30-32] Most of the reports, however, identify AO as a neuropathic pathology characterized by deafferentation.

A possible alternative hypothesis could be based on the Melzack theory of the "neuromatrix"—a neural network whose composition and connections are determined genetically and are later influenced by multiple inputs coming from different parts of the body.[33]

Woda and Pionchon stress that many facial pain conditions occur without any clear explanation or evidence of organic lesions, and this is why they grouped these pathologies under the term *idiopathic orofacial pain*.[20,27,28] Their approach underscores the unknown origin of the problem, although different hypotheses have been suggested.

One hypothesis suggests a major role of psychological diseases in the development of AO. Many studies indeed support a strong correlation between AO and different psychological conditions such as depression,[7,8,11,18,29,30,32] somatoform pain disorder,[24,26,30,31] anxiety,[18] demoralization,[18,30] introversion,[10] or hypochondriacal psychosis.[17] Rees and Harris examined 44 patients with AO and found that 29 (66%) had a history of depression or depressive symptoms and others had personality disorders.[11] Brooke and Schnurr also reported depression in 41% of 22 patients they examined with AO.[7]

Thus, as in other chronic pain conditions, we do not know if such conditions are the cause or the result of the pain.[19,23,28,34,35] Other authors criticize this hypothesis and question the relevance of the psychological component on pain.[13,19,23,26,27,34-37] Graff-Radford and Solberg evaluated 19 patients with AO using the Minnesota Multiphasic Personality Inventory (MMPI), and compared them with 19 patients with headache.[37] The profiles of the patients in the study were essentially unelevated, and there was no difference between the patients with AO and those with headache. This may suggest that psychological factors are not significant in the genesis of AO.

The most accredited theory currently is the hypothesis that trauma to the orofacial structures (traumatic injury, periodontal surgery, pulp extirpation, endodontic therapy, apicoectomy, tooth extraction, implant insertion), or even minor trauma (crown preparation, inferior alveolar nerve block) might alter the neural continuity of the tissues creating deafferentation.[21] This falls into the category of neuropathic pain, in that after the wound has healed the neural tissue is responsible for the pain and other related symptoms (paresthesia, dysesthesia).[23,24,38,39]

Multiple mechanisms are involved in the pathogenesis of pain: (1) sensitization of nociceptive fibers,[27,39-43] (2) sprouting of somatic afferent fibers from adjacent intact nerves,[39,40,42,44,45] (3) activation of afferent fibers by sympathetic efferents,[2,14,19,27,39,40,42,44-49] (4) cross-activation between injured afferent fibers (ephaptic crosstalk),[27,40,45,50,51] (5) phenotypic switching of afferent neurons,[27,39,40,42,52,53] (6) neuroma formation,[2,9,40,44] (7) changes induced in the central nervous system (CNS),[9,24,39,40,42,45,50,54-58] and (8) loss of inhibitory mechanisms.[9,27,39] Following a nerve injury, afferent fibers may become sensitized showing a lower activation threshold and developing spontaneous ectopic activity as a result of increased expression or redistribution of sodium channels.[39,41] This could explain some of the clinical manifestations of AO such as mechanical or thermal allodynia and persistent spontaneous pain.

Trauma to a nerve also has been associated with the formation of nerve collaterals from other non-injured nerves or from other parts of the same nerve.[45] When this occurs in the dorsal horn of the spinal cord, second-order neurons within lamina II, which usually receive nociceptive inputs, will also receive collaterals from non-nociceptive Aβ-fibers,[39,42] that might bring information misinterpreted as pain even when non-noxious stimuli such as hot, cold, or pressure are applied to the tooth (allodynia).[17,39,40]

Nerve collaterals have been observed sprouting from sympathetic fibers and reaching sensory afferents and sensory neurons of the dorsal root ganglia.[39-41,48,49] In addition, α-adrenergic or β-adrenergic receptors become expressed in those cells which develop sensitivity to catecholamines.[27,40-42] The role of the sympathetic system in the perception of pain in AO seems to be confirmed by the fact that sympatholytic procedures (stellate ganglion block, phentolamine infusion) usually significantly reduce the pain.[14,19,21]

Cross-activation of nerve fibers also has been reported by the formation of ephapses between injured sensory neurons.[27,40,51] This anatomical and functional connection might be responsible for enhanced ectopic and natural firing of the involved neurons,[27,40,50] leading to increased pain perception.[40]

Nerve fiber lesion, as well as other factors (hormonal, degenerative, traumatic, or psychological), has even been theorized to induce phenotypic changes in sensory afferent neurons causing altered expression of sodium/potassium channels, receptors, and neurotransmitters.[27,39,40] Non-nociceptive first-order neurons start releasing substance P and calcitonin gene-related peptide, activating second-order neurons in the dorsal horn of the spinal cord.[40,42,52,53]

Another phenomenon that may occur after a nerve has been damaged is the formation of a neuroma—a heterogeneous formation containing axoplasmatic elements, myelin, Schwann cells, and connective tissue elements that originates from the injured nerve growing in a disorganized fashion.[9,23,40,50,59] Neuromas are extremely sensitive to mechanical stimulation (pressure and tension) and norepinephrine, and produce continuous or episodic pain that can be spontaneous or triggered by external stimuli.[9,23,40,60] Similar characteristics appear in demyelinated axons, even in absence of a true neuroma.[40,50,61-63]

The CNS is also involved in the perception of pain. There is evidence that nerve lesions occurring peripherally lead to central changes at the cellular level and to functional changes in the CNS.[40,64-67] Similar to first-order neurons, the neurons in the receptive zones of the brain stem, within the CNS, demonstrate ectopic activity after trauma.[40,57] Some second-order neurons are normally intrinsically rhythmogenic, but the magnitude of discharge increases after nerve injury.[40,68-72]

A major role in the perpetuation of chronic pain by the CNS is attributed to *N*-methyl-D-aspartate (NMDA) receptors located on central nociceptive neurons.[27,39,40,42,73,74] Their activation is subordinated to activation of amino-3-hydroxy-5-methylisoxazole-4-propionic acid (AMPA) receptors or metabotropic receptors. These receptors cause the removal of the magnesium block from NMDA receptors and render them available for binding the neurotransmitter, glutamate, which has high affinity for this type of receptor.[39,40,73] The effect of this interaction is increased sensitivity to external stimuli and chronic pain that becomes independent of peripheral inputs, and these are the characteristics of AO.[40,73,74]

As a significant part of the CNS, we think inhibitory control of afferent stimuli deserves to be discussed separately. Since the amount of impulses that reaches the brain from the periphery is the result of excitatory and inhibitory modulation existing within the CNS,[74] we can achieve the same effect of increasing pain perception by either increasing the excitatory component or decreasing the inhibitory component.

A major role in the modulation of nociceptive stimuli in the CNS is performed by the reticular formation of the brain stem, which is part of the paleospinothalamic tract that carries "slow pain" impulses.[75] Marbach hypothesized that deafferentation by, for example, dental pulp denervation or tooth extraction, reduces the amount of inputs into the reticular formation, decreasing its inhibitory influence.[9] Yet, disinhibition can occur in other sites of the CNS such as the dorsal horn of the spinal cord. This effect

might be due to inhibitory interneurons' death following deafferentation,[27,39] down-regulation of inhibitory neurotransmitters within the same interneurons, or down-regulation of presynaptic inhibitory receptors on primary sensory neurons. These changes would enhance primary afferent excitability.[39]

According to the Melzack theory, a matrix of neurons exists that is genetically predisposed and is successively modified in response to inputs coming from several sources. These include somatic receptors, visual and other sensory inputs that influence the cognitive interpretation of the situation, phasic and tonic cognitive and emotional inputs from other areas of the brain, intrinsic neural inhibitory modulation inherent in all brain function, and the activity of the body's stress regulation system, including cytokines as well as the endocrine, autonomic, immune, and opioid systems.[33] The summation of these factors leads to the output of the neuromatrix that is called *neurosignature*, and is the processing and synthesis of all the nerve impulses in the brain. Body sensations that are normally elicited and modulated by sensory stimuli can be felt also in absence of these stimuli, because of processing occurring in the brain.[33] This is why previous pain experience could lead to change in existing neuromatrix and, in turn, in neurosignature; persistent input from altered neuromatrix can make the change persist after the pain event and become chronic, even without true deafferentation.

These changes in the peripheral and CNSs are common to many neuropathic diseases as it is evident reading the original articles. This is why AO probably shares the same pathophysiological mechanisms of other pathologies such as complex regional pain syndrome (CRPS) and traumatic neuralgia. It is also assumable that inferior alveolar or lingual nerve lesions, that are moderately common during dental procedures, can start a symptomatology resembling symptoms of either AO, CRPS, or traumatic neuralgia, even though CRPS seems to be very rare in the head and neck region.[40]

Nevertheless, all the mechanisms reviewed in this section could possibly play a role in the precipitation and perpetuation of pain in AO. For this reason, both diagnosis and management of this condition are still a challenge even for expert clinicians.

SIGNS AND SYMPTOMS

Since the diagnosis of AO is essentially clinical, special attention needs to be given to the clinical manifestation of the disease. The most prominent and sometimes the only symptom is pain. It is more commonly described as a continuous and spontaneous dull ache localized in a tooth.[12,20,21,23,25,36,76] The location may change to an edentulous area or entire parts of the maxilla or mandible.[4,20,21,36,76] The pain also can be described as burning, sharp, or throbbing.[4,12,17,20,21,25,36,76] It usually persists for months or years being continuous and persistent, but oscillating in intensity with episodes when the pain is more acute and severe.[5,12,25,76] Sleep is not disturbed by the pain which starts again after awakening.[20,24,25]

Accompanying symptoms that have been reported are headache (migraine or cluster headache),[12,77-79] hyperesthesia in the site of the pain,[76] allodynia,[20,25] exacerbation of pain evoked by temperature, palpation, and percussion.[11,17,21,25] No local signs of pathosis are usually present.

DIFFERENTIAL DIAGNOSIS

For a diagnosis of AO, other pathologies characterized by tooth pain need to be ruled out. Several have been listed: pulpal toothache,[8,16,23-25,30,80] trigeminal neuralgia,[7,21,24-26,30] temporomandibular joint disorders,[24,25,30] myofascial pain,[7,8,21,26] pretrigeminal neuralgia,[24,76] sinusitis,[7,8,21,25,30] ear and eye problems,[7] cracked tooth syndrome,[21] migrainous neuralgia,[7,8] temporal arteritis,[7,8] cranial neuralgias,[7,8] acute herpes zoster,[24-26,30] postherpetic neuralgia,[24-26,30] geniculate neuralgia,[24-26,30] arthritis of the temporomandibular joint.[24,25,30]

Probably the most difficult task is to distinguish between AO and toothache from pulpal origin. To help clinicians, 5 characteristics that are common to AO, but not common to pulpal toothache have been listed[16,23]: (1) constant pain in the tooth with no obvious source of local pathology; (2) local provocation of the tooth does not relate consistently to the pain. Hot, cold, or loading stimulation does not reliably affect the pain. (3) The toothache is unchanging over weeks or months. Pulpal pain tends to worsen or improve with time. (4) Repeated dental therapies fail to resolve the pain. (5) Response to local anesthesia is equivocal.

Thermography has been suggested by Graff-Radford et al as an additional aid in the diagnosis.[80] Patients with pulpal pain showed no thermographic difference in the territory of the pain complaint when compared to the opposite nonpainful side. Conversely, patients suffering from AO presented with either hot or cold thermograms.

It is usually easier to differentiate AO from trigeminal neuralgia because of the typical presentation of the latter. Marbach and Raphael highlighted the clinical and epidemiological characteristics of trigeminal neuralgia as follows:[24-26,30] (1) paroxysmal, unilateral, sharp, sudden, electrical, stabbing, recurrent pain confined to the distribution of one or more branches of the trigeminal nerve. Atypical odontalgia pain is dull and continuous. (2) Age of onset after the fourth decade, with a peak in the fifth and sixth decades. Atypical odontalgia is more frequent in women in their mid 40s. (3) Presence of trigger points that, stimulated by touch, elicit the pain. In addition, AO is usually preceded by a traumatic event to the tooth (root canal treatment, extraction, etc).

Other conditions that might be misdiagnosed as AO are temporomandibular disorders, including temporomandibular joint disorders and myofascial pain. In these pathologies, the pain is rarely limited to a tooth; it involves the preauricular and temporal regions, face, neck, and shoulder. Movements of the mandible (chewing, talking, yawning) commonly exacerbate the symptoms.[24] Trigger points that can elicit referred pain spontaneously and on palpation also characterize myofascial pain.[81]

Patients affected by pretrigeminal neuralgia have reported symptoms that are similar to those of AO:[82] burning, throbbing, and/or aching pain with no obvious dental pathology. In addition, pain frequently is reported to start after a dental procedure.[76] Nevertheless, we must note that the diagnosis of pretrigeminal neuralgia is a debatable issue, and that the name implies a pathophysiologic link to trigeminal neuralgia without scientific evidence.[24]

It is also important to recognize patients with migraine or cluster headache because AO has been reported to be associated with these types of headache. It is notable that pain increases during the headache episode in these individuals, and that the administration of some typical migraine (isometheptene,[12] methysergide,[12,78] flunarizine,[78,79] β-blockers[78]) or cluster headache (lithium[79]) medications often gives relief from the pain.[12,77-79]

Other diseases localized primarily in regions other than the teeth, such as eye, ear, and sinus, present usually with other accompanying symptoms that allow us to make a correct differential diagnosis.[7]

Attention should be paid when differentiating AO from the other conditions that could sometimes mimic such a disease,[7,8,21,24-26,30] knowing that the diagnosis can be difficult even after a thorough examination.

TREATMENT

Once the diagnosis of AO has been made, appropriate treatment needs to be established avoiding any further dental procedure that could only aggravate the pain. Most of the medications that are used are formulated for the treatment of neuropathic pain and seem to be effective in the majority of patients with AO.

In many of the articles reviewed,[4,5,8,9,13,16,21,23,24,26,76,83,84] tricyclic antidepressants have been prescribed with good results, alone or in association with phenothiazines (perphenazine or trifluoperazine).[4,9,21,24,26,83] More than their action on the mood, their analgesic effect seems to be responsible for the clinical results,[16,23] and phenothiazines potentiate the analgesic effect of the antidepressant. Amitriptyline has been used more frequently, in doses starting from 25 mg and going up to 100 mg daily.[2,4,5,8,9,16,21,23,24,83,84] Other tricyclics that have been suggested are imipramine,[4,21,83] nortriptyline,[21,84] and dothiepen.[11] What limits the use of these medications is the occurrence of side effects. Tricyclics may cause dry mouth, weight gain, constipation, and urinary retention, and are contraindicated in patients with angle-closure glaucoma or high intraocular pressure and in patients taking other medications such as monoamine oxidase (MAO) inhibitors, CNS depressants (alcohol, barbiturates, narcotics), anticholinergics, and sympathomimetics, because of drug-to-drug interactions. It is usually possible to avoid or minimize the side effects by adjusting the dose, or switching to a different medication within the same category (eg, nortriptyline has less

anticholinergic effect compared to amitriptyline and imipramine).[4,83,85] Phenothiazines need to be used more carefully because of potentially permanent adverse effects in the nervous system causing tardive dyskinesia. For this reason, when possible, their use should be limited and once the symptoms subside, the clinician should try to taper and stop the medication. Other side effects include drowsiness, dizziness, skin reactions, rash, dry mouth, insomnia, amenorrhea, fatigue, muscular weakness, anorexia, endocrine disturbance, blurred vision, and neuromuscular reactions; also, phenothiazines interfere with vasopressors, oral anticoagulants, thiazide, anticonvulsants, and CNS depressants.[4,83,86]

Some results have been reported with other medications such as gabapentin,[26] clonazepam,[24,26,76] baclofen,[24,26,76] doxepin,[4,8] α- and β-blockers,[76] aspirin,[9] phentolamine infusion,[14] cocaine,[24,26] and MAO inhibitors,[11,13,24] especially in cases where tricyclic antidepressants were not tolerated. Opioid narcotic analgesics (oxycodone, meperidine, controlled-release morphine, fentanyl, ketamine, and methadone) have been tried, but they are usually only moderately effective for neuropathic pain and AO.[24,26]

Sometimes a more "peripheral" intervention can give relief, especially when the local component of the pain is significant. Injections of local anesthetics and corticosteroids (dexamethasone), eventually repeated more than once, have been found effective especially in early treatments;[24,26] sympathetic and parasympathetic nerve blocks, through the stellate or sphenopalatine ganglia, have been reported with similar results.[9,76]

Medications applied topically to the site of the pain sometimes give good results, such as capsaicin at the concentration of 0.025% for 4 weeks, eventually associated with a topical anesthetic if the treatment is too painful,[14,23] and eutectic mixture of lidocaine and prilocaine bases (EMLA) cream at the concentration of 5%.[14]

Based on the results, once the diagnosis is made, we recommend starting treatment with low doses of amitriptyline (20 to 25 mg) or another tricyclic antidepressant if side effects are tolerable, and slowly titrate the dose evaluating effect on pain and side effects of the medication. Addition of phenothiazines should be avoided if not needed. If pain relief is obtained, the dose of the drug should be progressively reduced and finally discontinued, unless the symptoms return during the taper. In this case, the patient should be administered the lowest dose of medicine sufficient to control the pain.

CASE REPORTS

A MEDLINE search was performed for all AO cases reported and described in the literature since 1966. We found that descriptions of the cases (Table 6) generally confirm the clinical and epidemiological characteristics of AO as they have been reported in the literature.

As previously mentioned, all ages (except children) may be affected. In the literature, ages ranged from 13 to 82 years. The prevalence of females was high (4.6:1) confirming previous reports.[2-17] The major clinical symptom reported was usually tooth pain, frequently spread to involve other areas of the head and neck, including entire quadrants of the mouth, cheeks, temple, temporomandibular joint, eye, ear, neck, and shoulder. The quality of the pain is aching and dull, or burning and continuous, at times exacerbated by cold, touch, talking, eating, chewing, menses, or stress. Even though many cases occurred after dental treatment (endodontic treatment, tooth preparation, placement of crowns and bridges, tooth extraction, periodontal surgery), in other cases apparently there was not a precipitating factor. This fact is puzzling if we accept only deafferentation as the cause of symptoms.

Most of the patients were treated at first by dental therapy (root canal treatment, apicoectomy, tooth extraction, surgery, oral appliance) with no or minor results. Only after a diagnosis of AO was made and medical treatment was prescribed, did the patients receive relief from the pain. The most effective medications were tricyclic antidepressants and phenothiazines; however, good results were obtained using other medications such as injections of dexamethasone and lidocaine, anti-inflammatories, sphenopalatine blocks with cocaine hydrochloride, ethyl chloride vapocoolant sprays, trazodone, methysergide, isometheptene, MAO inhibitors, topical application of EMLA, phentolamine infusion, and topical application of capsaicin. Prognosis seems to be fair in most

Table 6.—Literature Review of Atypical Odontalgia*

Source, y	No. of Cases	Age at Diagnosis, y	Male: Female Ratio	Chief Complaint	Precipitating Factors	Concomitant Pathologies	Treatment	Outcome
Marbach,[9] 1978	25	2-77	3:25	Tooth pain Severe unremitting pain in upper R quadrant of mouth, radiating to R ear & R side of face & neck	Dental procedure Unknown	None	Intramuscular injections of dexamethasone & lidocaine; Tricyclic antidepressants; Anti-inflammatory medications Sphenopalatine ganglion blocks with cocaine hydrochloride Ethyl chloride vapocoolant sprays	52% of patients reported relief from pain, 32% no change, 0.8% increase of pain, 0.8% some exacerbations & remissions of pain
Brooke,[7] 1980	22	3-74	0:22	Ache in all upper teeth, intermittent sharp, jabbing pains in isolated teeth, exacerbation of pain by cold food, chewing, touching teeth Pain in all parts of mouth from time to time	Dental procedure (tooth preparation for bridge) Unknown	Migraine, depression, bruxism, tension-type headache, thyrotoxicosis, cystic fibrosis, asthma	Antidepressants, tranquilizers	50% of patients had permanent relief from pain with use of antidepressants & tranquilizers
Kreisberg,[8] 1982	2	39, 41	0:2	Constant, severe dull ache in R zygomatic & facial region Intermittent dull pain in L facial region lateral to nose, precipitated by talking, eating, chewing	Dental procedure (full mouth reconstruction) Emotionally upsetting events	Anxiety, depression	Amitriptyline, doxepin hydrochloride, psychotherapy	Complete relief from pain

(*Continued*)

Table 6.—Continued

Source, y	No. of Cases	Age at Diagnosis, y	Male: Female Ratio	Chief Complaint	Precipitating Factors	Concomitant Pathologies	Treatment	Outcome
Reik,[12] 1984	8	3-56	1:7	Pain in R mandibular first & second premolar, spread to R maxillary first premolar Localized to one tooth or several adjacent teeth Continuous aching & throbbing discomfort in mandibular teeth Throbbing pain in R mandibular first molar, spread to R temple & TMJ, neck, & shoulders, worsened during menses	Unknown	None Depression	Amitriptyline, fluphenazine, trazodone, methysergide, isometheptene	3 patients reported complete relief from pain, 2 reported partial relief, 3 reported no changes
Bates & Stewart,[4] 1991	30	22-82	3:27	Burning, throbbing pain in maxillary & mandibular L quadrants Intense pain with "pulling" & "swelling" sensation in maxillary R tuberosity area, spread to entire R maxillary quadrant Tooth pain in maxillary L molar become pain & "itching" in whole L maxillary quadrant	Dental procedure (placement of crown & bridge) Unknown	None Myofascial pain	Amitriptyline, trifluoperazine, perphenazine, imipramine, doxepin	Almost complete relief from pain
Schnurr & Brooke,[13] 1992	120	13-80	23:97	Pain in teeth, jaws, & gingiva, at times spreading involving face, cheeks, areas around eyes, & ears	Dental procedure (restorative procedure, tooth extraction)	None Migraine	Tricyclic antidepressant, MAO inhibitor	Temporary relief from pain

(*Continued*)

Table 6.—Continued

Source, y	No. of Cases	Age at Diagnosis, y	Male: Female Ratio	Chief Complaint	Precipitating Factors	Concomitant Pathologies	Treatment	Outcome
Pertes et al,[21] 1995	2	28, 52	1:1	Pain in upper R posterior teeth Pain in several teeth, jaw bones, jaw joint	Dental procedure (periodontal surgery) Unknown	None	Amitriptyline, fluophenazine	Complete relief from pain
Battrum & Gutmann,[5] 1996	1	47	0:1	Continuous pain in area of maxillary L first premolar	Dental procedure (tooth extraction)	Mild hypertension	Amitriptyline	Complete relief from pain
Lilly & Law,[84] 1997	2	54, 71	0:2	Pain within maxillary & mandibular anterior region Burning pain distal to mandibular R canine	Unknown	Non-insulin dependent diabetes mellitus, osteoarthritis, ankylosing spondylitis, asthma, allergy to penicillin	Amitriptyline, nortriptyline	1 patient reported complete relief from pain with use of amitriptyline, 1 reported partial relief with use of nortriptyline
Vickers et al,[14] 1998	50	21-82	16:34	Tooth pain, pain in entire quadrants	Dental procedure Unknown	TM disorders	Topical application EMLA, phentolamine infusion, topical application of capsaicin	14% of patients reported complete relief from pain with use of EMLA, 60% reported partial relief, 2% reported no change 18% of patients reported partial relief from pain with use of phentolamine, 6% reported no change 4% of patients reported complete relief from pain with use of capsaicin, 42% reported partial relief, 14% reported no change

*R indicates right; L, left; TM, temporomandibular.

cases with many patients completely pain-free after treatment and some reporting only partial relief. Only a few did not obtain satisfactory results.

CONCLUSIONS

We do not have enough data to draw definitive conclusions on the etiology and pathophysiology of AO, but our opinion is that neuropathic mechanisms are involved primarily, perhaps not necessarily induced by trauma, since not all the patients report such an event, and psychological disturbances are probably more the consequence than the cause of chronic pain.

In light of the information extrapolated from cases of AO reported in the literature, the major issue in the treatment and prevention of the pain is establishing a correct diagnosis. Even though not all the cases of AO seem to originate from trauma due to dental procedures, almost all the patients underwent several dental treatments without obtaining any relief from the pain. Attention should be paid to any toothache in absence of evident signs of dental pathology in order to avoid unnecessary treatments that could originate or perpetuate the problem. Misdiagnosis can lead sometimes to frustrating outcomes. Significant is a panoramic radiograph showed by Marbach in several articles where ALL THE TEETH of a 22-year-old woman were treated by root canal treatment and apicoectomy![3,24-26]

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