

Diagnosis and Treatment of Atypical Odontalgia: A Review of the Literature and Two Case Reports

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Abstract

Aim: This report presents two cases diagnosed with atypical odontalgia (AO) and successfully treated with amitriptyline as well providing a review of the current literature on the subject.

Results: The literature indicates the most important issue is an accurate differential diagnosis to distinguish between AO, pulpal pain, myofascial pain, and trigeminal neuralgia.

Conclusion: Once the correct diagnosis is made the prognosis of AO is usually fair and the administration of tricyclic antidepressants often resolves symptoms. An effort should be made to avoid any unnecessary dental treatment that would only aggravate the problem.

Keywords: Atypical odontalgia, AO, phantom tooth pain, differential diagnosis, treatment, amitriptyline, tricyclic antidepressants

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Introduction

Atypical odontalgia (AO) has been defined by Merskey¹ as “severe throbbing pain in the tooth without major pathology.” In fact the primary symptom is pain, usually located in a tooth or a tooth site,²⁻⁹ that may spread with time involving the entire maxilla or the mandible.^{2,4,6-10} The pain is frequently continuous and persistent, but fluctuating in intensity^{4-5,7,11} with no signs of pathosis present. The purpose of this manuscript is to present two cases of AO, mainly focusing on the diagnosis and treatment of the disease, and to describe these cases who were patients suffering from AO that were successfully treated pharmacologically.



Literature Review

A MEDLINE search was performed selecting the articles on AO written in the English language and published from 1966 to 2004. The term “phantom tooth pain” was also included in the research since it has been frequently employed in the literature. Several articles were found on the topic with ten^{2,5,10-17} involving cases clinically evaluated and treated.

Pain is described as the main characteristic of the disease, usually located in a tooth or a tooth site,²⁻⁹ that can spread to a wider part of the face^{2,4,6-9,10} and usually starts after a dental or surgical procedure.¹⁸⁻²⁰ The pathology is more frequent in female patients in their mid-40s,^{1,9-10,21-25} affecting predominantly maxillary molars and premolars.^{2,6,9,25-26} Typically no clear tooth or periodontal pathoses are evident, no radiographic

signs are present, and anesthetic block of the involved tooth provides equivocal results.^{2,9,18-20,26} The pain also stops during sleep.^{9,18-20}

The pathophysiology of AO is not clear, yet idiopathic,^{6,20,27-28} psychogenic,^{20,29-31} and neuropathic^{3,19-20,32} hypotheses have been suggested. Probably the most accepted theory is deafferentation of the nerves involved is caused by a traumatic injury with changes occurring at the level of the peripheral, central, and autonomic nervous systems.^{20,33-34}

There are no universally accepted guidelines for the diagnosis of AO while several authors have suggested their own.^{1-3,7,18-19,21,35} In our opinion the diagnostic criteria best describing the disease are the ones proposed by Graff-Radford and Solberg²⁶ in 1992 (Table 1). They are very simple but focus on all of the typical characteristics of AO. The criteria describe the pain which is the principle characteristic of the disease in terms of location, timing, and duration. Moreover, the criteria help with the differential diagnosis by indicating the absence of clinical signs of pathology, and local anesthesia does not resolve the pain.

Differential Diagnosis

Particular attention to the diagnosis and treatment of AO was paid during the literature review. As a result, the authors propose some guidelines for the differential diagnosis of AO based on the reports examined.

Since a patient with AO might present with pain in the gingiva, mandible, maxilla, a single tooth, or other parts of the face,²⁻¹⁰ other pathologies characterized by similar symptoms need to be considered. The most common source of dental pain is pulpal in origin which can be confused with AO. However, several factors help to differentiate between the two diseases. They relate to the characteristics of pain and its

Table 1. Diagnostic criteria for Atypical Odontalgia.²⁶

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 four months.
D. No sign of local or referred pain.
E. Equivocal somatic nerve block.

duration, the evidence of pathology in the tooth, and the response to treatment and diagnostic tests.^{3,9,21} The details of this diagnostic approach are shown in Table 2.

Another condition frequently a cause of pain symptoms in the orofacial region is myofascial pain. The principle test to help differentiate between AO and myofascial pain is palpation of the orofacial muscles. In a patient affected by myofascial pain palpation elicits pain not only at the site of palpation but can be referred to other areas of the face.^{19,36} Conversely, AO pain is not affected by muscle palpation. The details of this approach are shown in Table 3.

A less common disease that can be confused with AO is trigeminal neuralgia. It is characterized by sharp pain in the absence of any signs of pathosis, and the pain can be localized in a tooth. However, the characteristics of the pain and the frequent presence of trigger zones allow us to differentiate between the two pathologies.^{7,9,18-19,35,37} The details of this approach are shown in Table 4.

Treatment

The most important evidence to be deduced from the articles reviewed on this topic is dental procedures should be avoided in the treatment of AO. Furthermore, the literature indicates pharmacologic treatment is often successful.

Table 2. Differential diagnosis between Atypical Odontalgia and pulpal pain.

Atypical Odontalgia	Pulpal Pain
1. Pain is constant and unchanging over weeks or months.	1. Pain is oscillating and tends to worsen or improve with time.
2. Local provocation of the tooth (hot, cold, pressure) does not relate consistently to the pain.	2. Local provocation (hot, cold, pressure) exacerbates the pain.
3. No clinical or radiographic signs of pathology (decay, fracture) are present in the tooth.	3. Clinical or radiographic signs of pathology (decay, fracture) can be detected in the tooth.
4. Repeated dental therapies fail to resolve the pain.	4. Dental therapy resolves the pain .
5. Response to local anesthesia is equivocal.	5. Local anesthesia resolves the pain.

Table 3. Differential diagnosis between Atypical Odontalgia and myofascial pain.

Atypical Odontalgia	Myofascial Pain
1. Pain in a tooth or a tooth site.	1. Pain is rarely limited to a tooth, it involves the preauricular and temporal regions, face, neck, and shoulder.
2. Mandibular function does not affect the pain.	2. Movements of the mandible (chewing, talking, yawning) commonly exacerbate the symptoms.
3. There are no trigger points, and muscle palpation does not affect the pain.	3. Trigger points can elicit referred pain spontaneously or on muscle palpation.

Table 4. Differential diagnosis between Atypical Odontalgia and trigeminal neuralgia.

Atypical odontalgia	Trigeminal Neuralgia
<ul style="list-style-type: none"> • Pain is dull and continuous. • More frequent in women in their mid 40s. • No trigger zones are present. • Symptoms are usually preceded by a traumatic event to the tooth (root canal treatment, extraction, etc .). 	<ul style="list-style-type: none"> • Pain is paroxysmal, unilateral, sharp, sudden, electrical, stabbing, recurrent ; confined to the distribution of one or more branches of the trigeminal nerve. • Age of onset after the fourth decade, with peak in the fifth and sixth decades. • Presence of trigger zones when, stimulated by touch elicit the pain. • No history of trauma.

Administration of several medications has been tried and reported to achieve control of the pain in patients affected by AO. These medications include:

- * gabapentin¹⁸
- * clonazepam^{4,18-19}
- * baclofen^{4,18-19}
- * α and β -blockers⁴
- * aspirin¹²
- * phentolamine infusion¹³
- * cocaine¹⁸⁻¹⁹
- * doxepin^{10,15}
- * monoamine oxidase inhibitors^{14,19,38}
- * opioids¹⁸⁻¹⁹
- * injections of local anesthetics and corticosteroids¹⁸⁻¹⁹
- * sympathetic and parasympathetic nerve blocks^{4,12}
- * topical capsaicin^{3,13}
- * eutectic mixture of lidocaine and prilocaine bases¹³

The most efficacious medications that have been described are tricyclic antidepressants^{2-4,10-16,18-19,21,39} alone or in association with phenothiazines.^{2,10,12,18-19,39}

A report by Marbach¹² described 25 cases of AO, out of which 17 were treated with tricyclic antidepressants. Good results were obtained in 69% of the cases (11 out of 16) with relief from the pain. Similar results were achieved by Brooke¹⁷ who reported permanent resolution of the symptoms for 50% of 22 patients after administration of tricyclic antidepressants.

Kreisberg¹⁵ described two cases of AO, the first case was treated with amitriptyline and the pain subsided completely after two months, the second case was also treated with amitriptyline and the patient experienced a gradual reduction of the symptoms. The treatment of anxiety was necessary to achieve complete relief.

Another eight cases were described by Reik.⁵ Five out of eight patients were treated with amitriptyline in doses ranging from 25 mg to 200 mg. One patient discontinued the medication because of side effects (excessive drowsiness) and one never took the medicine. Among the other three patients, 75-100% of pain relief was obtained.

Bates and Stewart¹⁰ described three cases, out of 30 treated, specifying management of AO was started using amitriptyline associated with perphenazine in one patient and amitriptyline associated with trifluoperazine in a second patient. In both patients pain relief was achieved but the medication could not be tapered. In a third patient successful results were obtained using imipramine followed by doxepin.

Schnurr and Brooke¹⁴ presented results after treatment of 120 patients with AO and reported partial relief from pain after treatment with antidepressants. However, long-term follow-up (5 years or more from first visit) revealed 19 out of 28 patients that could be reached still had pain.

In two cases described by Pertes et al.² amitriptyline alone or in association with fluoperazine either eliminated or significantly reduced the pain. Similar results were achieved by Battrum and Gutmann¹¹ who reported a single case of AO completely resolved after administration of amitriptyline.

Lilly and Law¹⁶ describe two cases where tricyclic antidepressants were used to treat the pain. In the first case amitriptyline was prescribed and the patient had complete relief. In the second case nortriptyline was prescribed and the patient obtained significant, but not complete relief from the pain.

Although amitriptyline is the medication that has been reported more frequently in the literature,^{2-3,10-12,15-16,19,21,39-40} other tricyclic antidepressants have been used (imipramine,^{2,10,39} nortriptyline,^{2,16} and dothiepin³⁸) and probably have the same effect. Treatment starts with a low dose of 20-25 mg of amitriptyline that needs to be adjusted according to two factors: (1) pain control and (2) adverse reactions. The dose is titrated until acceptable pain level is achieved, usually reaching up to 75 mg per day,^{2-3,15,21,39} but the appearance of side effects can prevent the clinician from increasing the dosage. In this case the first option is to switch to a different drug within the same category, like imipramine and nortriptyline. Common side effects of amitriptyline include: dizziness, drowsiness, headache, xerostomia, constipation, increased appetite and weight gain, nausea, weakness, hypotension,

arrhythmias, tachycardia, nervousness, sedation, diarrhea, and many others that are less frequent.⁴¹ Other antidepressants share similar effects,⁴²⁻⁴⁴ however, imipramine and more markedly nortriptyline cause less incidence of drowsiness, orthostatic hypotension, and cardiac arrhythmias.⁴⁵ In the event symptoms cannot be controlled by the use of tricyclic antidepressants alone, phenothiazines can be included in the therapeutic regime.

Nonetheless, particular attention should be paid to patient response to antidepressant medications because adverse reactions include tardive dyskinesia, which is a permanent extrapyramidal movement disorder.⁴⁶ Their use should be limited to the cases that cannot be resolved otherwise, and the dose should be tapered and discontinued after pain control is achieved.

Case Reports

Case One

A 38-year-old female presented for evaluation of pain in her upper left first molar. She reported the pain started one year earlier right after the tooth “exploded” while chewing a piece of toast. The tooth was endodontically treated by her dentist, but the pain did not stop and spread to the entire zygoma and the left side of the neck below the ear. The pain was described as continuous and dull, occasionally throbbing. It was exacerbated by warmth such as sitting beside the fireplace or staying in the sun. It was typically absent during sleep but resumed about one hour after awakening.



Non-steroidal antiinflammatory drugs such as ketoprofen and diclofenac reduced but did not eliminate the pain. The patient was previously treated for anxiety with alprazolam but that treatment did not affect the pain. A clinical examination revealed the following: malocclusion (loss of vertical dimension of occlusion; mandibular midline shift to the left) and tenderness on palpation of the left masseter and right lateral pterygoid muscles and the temporalis tendon. In addition, the left trapezius, levator scapulae, and scalene muscles were tender. The range of motion of the temporomandibular joints was normal, yet the left joint was tender and a reciprocal click was heard on opening and closing of the mouth.

A diagnosis of pulpal pain and trigeminal neuralgia (Table 2 and Table 4) was ruled out because of the duration, location, and the characteristics of the pain. Percussion of the tooth did not affect the pain, and there was an absence of caries, periodontal, and trigger zones to elicit the pain. The differential diagnosis was limited to AO and myofascial pain because the patient reported some muscle ache both spontaneously and on palpation. As a result, the diagnostic effort focused on determining if her main complaint was caused by muscle dysfunction or a neuropathic condition. Following the criteria listed in Table 3, a diagnosis of AO was made. The patient's tooth pain was not affected by chewing, talking, yawning, or moving the mandible and was not increased by muscle palpation. The fact palpation of some muscles of the neck and shoulder elicit pain was used for a diagnosis of myofascial pain is considered comorbid with AO but not related to the tooth pain. Furthermore, many of the other features of AO were present such as: involvement of maxillary molar, female patient, age of about 40 years, previous endodontic treatment, and no pain during sleep.

A conclusive diagnosis of disc displacement without reduction of the left temporomandibular joint, myofascial pain, and AO was made. Treatment for AO was started prescribing amitriptyline 25 mg once a day before sleep. After two days, the patient complained of severe drowsiness in the morning and the dose was gradually reduced to a quarter of a tablet (6.25 mg). After two additional days, the pain was

reduced and no side effects were reported. The following week the patient reported complete relief from the pain with no side effects from the medication. The dosage was maintained for six weeks and then the dose of antidepressant was decreased (more for psychological reasons since the dose was already very low) and then discontinued. At a one-month follow-up visit, the patient was free of tooth pain and she chose to discontinue further treatment for her muscle pain she described as mild. At six and 12-month follow-up visits, the results obtained previously were stable and no further treatment was needed.

Case Two

A 39-year-old female patient with a chief complaint of pain in the left side of the maxilla presented for evaluation. She reported the pain started as a mild discomfort limited to the cervical area of the teeth which later spread to the entire half of the maxilla and occasionally to the mandible. The pain was dull and persistent and apparently started spontaneously about one year earlier. It was milder in the morning while waking up and moderately increased when the patient clenched her teeth during the day.

Several treatments had been attempted including the administration of various medications (amoxicillin, gabapentin, tramadol, nimesulide, ketoprofen, rofecoxib, and other anti-inflammatory drugs) but the results were unsatisfactory.

Upon examination the patient showed a class II malocclusion with loss of vertical dimension of occlusion and mandibular midline shift to the right. The range of motion of the temporomandibular joints was normal and asymptomatic, and the trapezius muscle was bilaterally tender to palpation.

Similarly to the previous case, the characteristics and the duration of the pain together with the absence of any clinical and radiographic signs of dental and periodontal disease excluded a diagnosis of pulpal pain (Table 2). In addition, the muscles of the face and neck, except for the trapezius, were not tender to palpation, and palpation of the trapezius elicited only local tenderness without affecting the complaint of the patient. Therefore, diagnosis of myofascial pain was excluded as well (Table 3). The timing

of the pain and the absence of trigger zones is completely different from the typical presentation of trigeminal neuralgia and such a diagnosis was also excluded (Table 4).

The patient demonstrated some of the usual features of AO such as: involvement of the maxillary molar, female gender, and an age of about 40 years. However, the pain was also present at night, although milder than the rest of the day, with an absence of a history of trauma. These two points are not necessary for the diagnosis according to the proposed criteria²⁶ (Table 1), therefore, a conclusive diagnosis of AO was made.

Treatment was initiated by prescribing amitriptyline at a dosage of 10 mg once a day before sleep. The dose was gradually increased one month later to 30 mg with moderate improvement and further increased to 60 mg (20 mg three times a day) during the following month. At that time, the patient reported complete resolution of the symptoms. The dosage was kept stable for 30 days without relapse of pain. The medication was gradually decreased and then stopped within three months. At one and three month follow-up visits, the patient remained asymptomatic.

Discussion

The treatment of AO in the present case reports utilized amitriptyline, which was effective in eliminating the pain in both patients. The dosage used was very low in the first case, equal to 6.25 mg, while in the second case a higher dose was necessary, equal to 60 mg a day. In both cases complete pain relief was achieved, and it was possible to decrease and then discontinue the medication without relapse of symptoms. Our results are similar to the ones reported in many previous studies describing a favorable outcome for treatment of AO with amitriptyline^{2-4,10-16,18-19,21,39} with complete or partial relief from pain. The dosage used is similar, although there is no specific dosage indicated for the treatment. In fact the correct approach is to increase the dose of the medication until the pain subsides, with the limit imposed by the appearance of side effects. As previously mentioned, amitriptyline has been used in doses ranging from 20 mg to 75 mg^{2-3,10-11,15-16,21,39} which are higher than the dose



administered in the first case reported here but comparable to the dose used in the second case.

Discomfort from adverse reactions caused by the medication was easily managed by adjusting the dose. This happened paradoxically in the first case when a low dose of 25 mg produced severe drowsiness, while in the second case a higher dose of 60 mg did not create any discomfort for the patient. However, reducing the dosage in the first case eliminated the side effect without reducing the efficacy of the treatment.

Although it was not a necessity in the present case reports, when side effects cannot be tolerated by the patient and managed adjusting the dose of the medication, other tricyclic antidepressants such as imipramine^{2,10,39} and nortriptyline^{2,16} represent alternatives available.

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In fact, these drugs cause fewer adverse reactions. Imipramine and especially nortriptyline cause a lower incidence of drowsiness, orthostatic hypotension, and cardiac arrhythmia.⁴⁵ Unfortunately, their use in clinical practice is limited because a few reports are described in the literature^{2,10,16,39} but they are probably as effective as amitriptyline.

Another solution not required in the present cases was the supplemental use of phenothiazines with the tricyclic antidepressant to control the pain. As mentioned previously, there is the possibility of the appearance of tardive dyskinesia as a side effect that is an extrapyramidal movement disorder which is unfortunately a permanent condition.⁴⁶ Therefore, the use of these medications should be limited to the patients who do not respond to tricyclic antidepressants alone. The dosage should be gradually decreased and stopped after pain control has been achieved.

Conclusions

In the light of the results reported in the literature, amitriptyline seems to be the treatment of choice in the selection of a medication for the treatment of AO. Satisfactory relief from the pain is usually achieved by using this medication. The management and the outcome of the two cases reported in the present study appear to confirm this recommendation. The most important aspect of the AO patient is making the correct diagnosis, which in turn allows the clinician to take a conservative approach and avoid unnecessary dental treatment.

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