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# Clinical characteristics and diagnosis of atypical odontalgia

## Implications for dentists

Saravanan Ram, BDS, MDS; Antonia Teruel, DDS, PhD; Satish K.S. Kumar, BDS, MDS; Glenn Clark, DDS, MS

In recent years, investigators have recognized atypical odontalgia (AO) as a chronic trigeminal neuropathy affecting the maxillary or mandibular divisions of the trigeminal nerve.<sup>1,2</sup> Alternative terms for AO are “persistent orodental pain” and, if the patient has had teeth extracted, “phantom tooth pain.” Patients with AO often have continuous pain located in a tooth, the gingiva or an extraction site, and it often can involve other areas of the face.<sup>3-6</sup> Several reports indicate that the pain usually begins when the patient undergoes a dental or surgical procedure and persists long afterward.<sup>5,7,8</sup> Typically with this pain, no obvious tooth or periodontal pathologies are evident, and no radiographic signs of pathology are present. A local anesthetic block of the involved tooth usually produces modest-to-equivocal pain relief.<sup>6</sup>

To date, there are no universally accepted and well-established classification and diagnostic criteria for AO,<sup>5</sup> and hence this condition often is poorly understood and commonly misdiagnosed by dentists and physicians.<sup>2</sup> Patients with this condition often undergo multiple unnecessary dental or surgical procedures that

## ABSTRACT

**Background.** Atypical odontalgia (AO) is a poorly understood and commonly misdiagnosed condition for which patients often undergo multiple unsuccessful dental or surgical procedures. The authors conducted a study to determine the prevalence and describe the characteristics of patients with AO seen at the University of Southern California Orofacial Pain and Oral Medicine Center (USC OFP-OM Center), Los Angeles.

**Methods.** The authors conducted a retrospective record review from a database of more than 3,000 patient records from June 2003 to August 2007 to identify patients diagnosed with AO.

**Results.** The authors identified 64 patients (44 women and 20 men) between the ages of 26 and 93 years as having a diagnosis of AO. Of those 64 patients, 71 percent initially consulted a dentist regarding their pain, and 79 percent had undergone dental treatment that failed to resolve the pain. The pain of 64 percent of the patients had no known cause.

**Conclusions.** Dentists, who often are the first health care providers to see patients with AO, must be aware of this condition and must follow the appropriate steps to determine its diagnosis.

**Clinical Implications.** Dentists and physicians should understand the implications and importance of early diagnosis of patients with AO and of referral to pain specialists for treatment.

**Key Words.** Atypical odontalgia; chronic trigeminal neuropathy; phantom tooth pain.

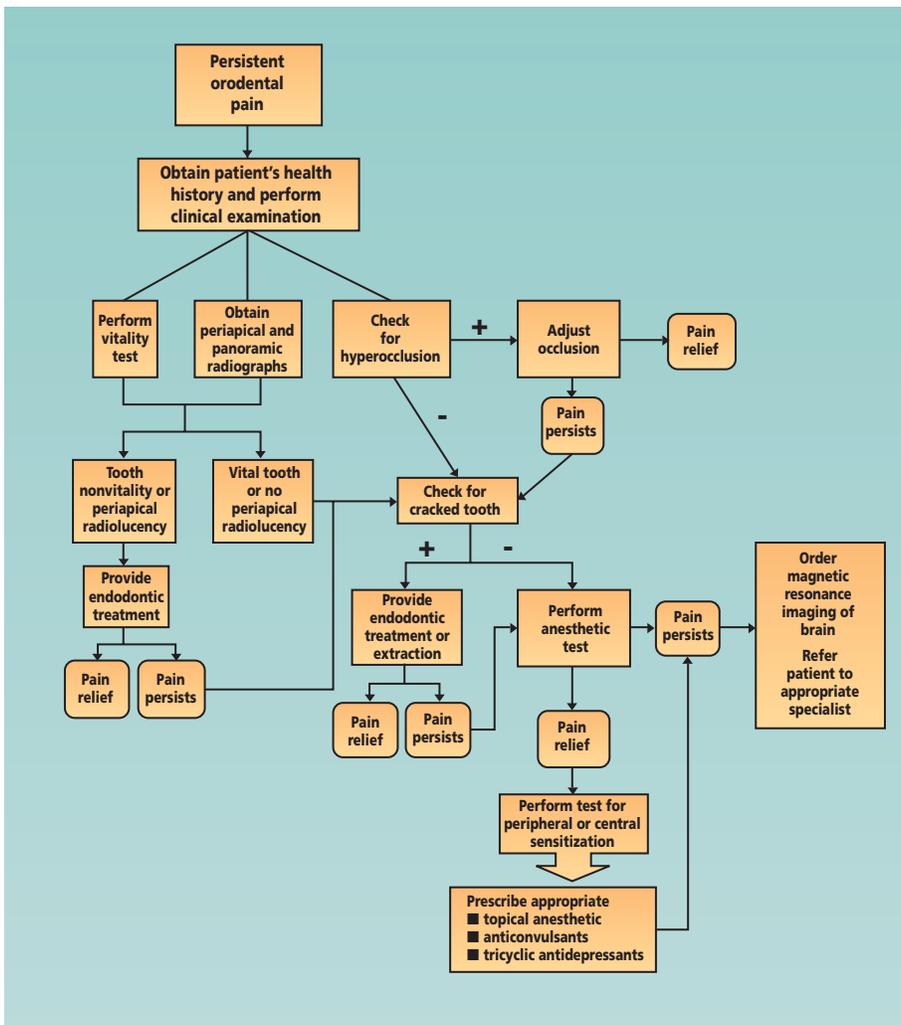
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**Figure 1.** The diagnostic work-up for atypical odontalgia of the Orofacial Pain and Oral Medicine Center, University of Southern California, Los Angeles.

are likely to be unsuccessful in suppressing the pain.<sup>9,10</sup> Few data are available regarding the incidence and prevalence of AO, and reports of studies in the pain literature have focused primarily on other neuropathic conditions such as trigeminal neuralgia, postherpetic neuralgia, painful diabetic neuropathy and phantom limb pain.<sup>2,11</sup>

We conducted a study to determine the prevalence and describe the characteristics of AO among the patient population seen at the University of Southern California Orofacial Pain and Oral Medicine Center (USC OFP-OM Center) at the USC School of Dentistry in Los Angeles between June 2003 and August 2007.

**SUBJECTS, MATERIALS AND METHODS**

We conducted a retrospective record review of data drawn from the electronic medical record

database (SOAPware, version 4.95, SOAPware, Fayetteville, Ark.) at the USC OFP-OM Center of more than 3,000 patient records from June 2003 to August 2007. We obtained approval to conduct the study from the University of Southern California University Park Institutional Review Board (USC UPIRB #UP-07-00416). We identified all patients who were diagnosed with AO by using the chart-search function in the SOAPware program with the appropriate search terminology. Either faculty members or residents under faculty supervision made the diagnosis of AO for all patients. The diagnosing clinician performed a thorough history and head and neck examination for every patient, as well as the radiographic investigations necessary to rule out all potential dental and bony pathologies. Inclusion criteria for AO include having a persistent pain with varying character in the absence of positive clinical and radiographic findings that may or may not be responsive to diagnostic local anesthetic injections or blocks. Before

arriving at a diagnosis of AO, the clinician excluded the potential pain causes of dental caries, periapical lesions, periodontal pockets with bone loss, cracked teeth, hyperocclusion, nonvital teeth and other bony pathologies by following the USC OFP-OM Center diagnostic work-up procedure (Figure 1).

On establishing a diagnosis of AO, the diagnosing clinician performed anesthetic testing (Box 1) to distinguish between peripheral and central trigeminal neuropathic changes. Complete relief of the patient’s pain with the anesthetic indicated

**ABBREVIATION KEY.** AO: Atypical odontalgia. ENT: Ear, nose and throat. OFP-OM: Orofacial Pain and Oral Medicine. TMD: Temporomandibular disorder. USC: University of Southern California. VAS: Visual analog scale.

**BOX 1**

**University of Southern California Orofacial Pain and Oral Medicine Clinic Anesthetic Test.\***

- Use a cheek retractor and cotton rolls to isolate the painful area.
- Dab the painful area dry with a 2-centimeter x 2-cm piece of gauze.
- Record the patient's pain level on a visual analog scale (VAS) of 0 to 10.
- Apply benzocaine 20 percent gel topically to the painful area.
- Every three minutes, record the patient's pain on the VAS.
- If there is incomplete pain relief, perform a local anesthetic infiltration or nerve block injection at the painful site with 2 percent lidocaine gel.
- Again, record the pain level on the VAS after three minutes.
- Repeat test during follow-up visit.

\* Adapted with permission of Journal of the California Dental Association from Ram and colleagues.<sup>12</sup>

that the neuronal changes were localized to the primary or first-order neuron in the peripheral nervous system. Incomplete pain relief with anesthetic indicated that changes were localized to the level of the second- and third-order neurons in the central nervous system.<sup>2,12</sup> The diagnosing clinician repeated the anesthetic testing at the patient's follow-up (second) visit to avoid placebo responses and equivocal test results.

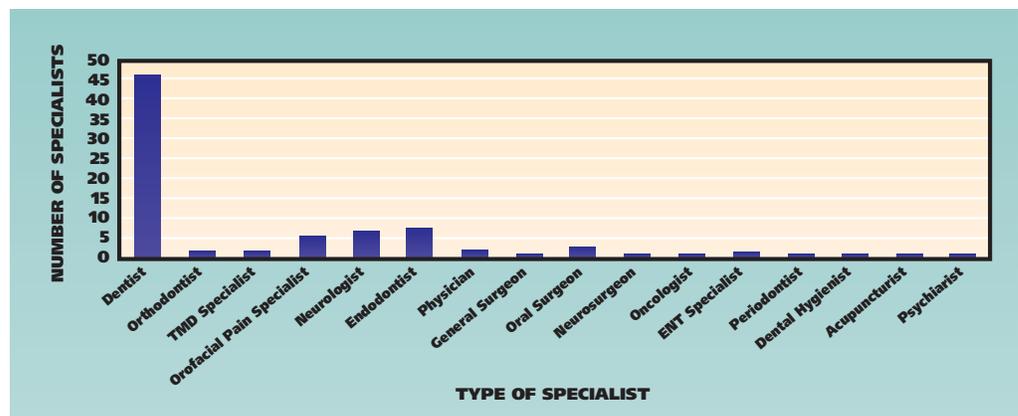
**RESULTS**

The prevalence of AO in the population we studied (N = 3,000) was 2.1 percent: diagnosing clinicians had given a total of 64 patients (44 women and 20 men) between the ages of 26 and 93 years (mean age, 55.4 years) a diagnosis of AO. The racial characteristics of our patient population were as follows: white, 30 patients; Hispanic, 20; Asian, five; African-American, four; American Indian, one; Pacific Islander, one; and others, three. Before undergoing the evaluation at USC OFP-OM Center, most patients had been diagnosed by various dentists, physicians and specialists as having dental disease-related pain. Routine dental treatment such as

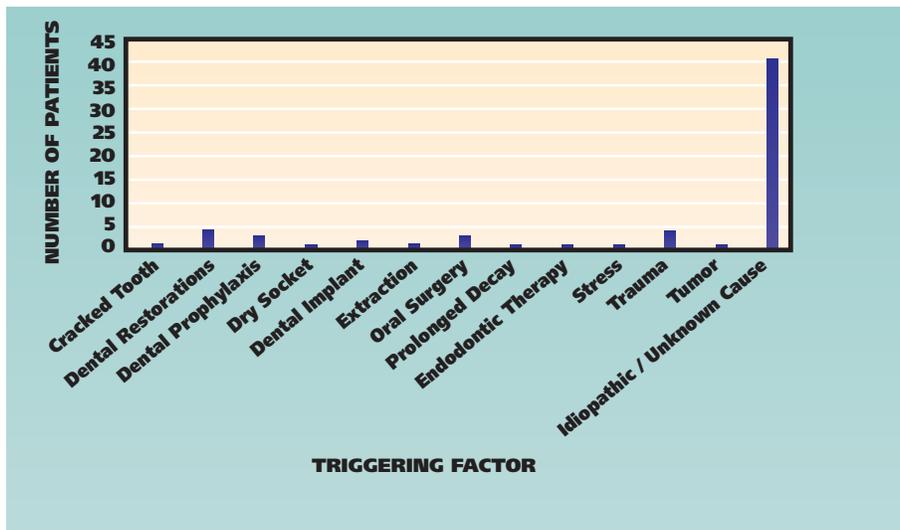
restorative therapy, endodontic therapy, extractions and implant therapy failed to resolve the pain and, in some cases, made the pain worse. The average number of dentists, physicians or specialists with whom each subject consulted regarding his or her pain before undergoing our evaluation was 1.7 (range, 1-5), and 71 percent (n = 46) of the patients saw dentists for their initial consultation and treatment (Figure 2). Figure 3 shows the reported causes or triggering factors for AO; 64 percent (n = 41) of the cases had no known (idiopathic) causes. Eighty percent (n = 51) of the patients had undergone some form of dental procedure that failed to resolve the pain. Figure 4 (page 227) presents the dental or medical interventions that clinicians had attempted to treat these patients' persistent tooth pain. Endodontic therapy with or without apicectomies or extractions of the suspected tooth was the dental procedure performed most commonly in these patients. Nearly 16 percent (n = 10) of the patients had no history of any dental or surgical procedures' having been performed for their pain. The average duration of pain before our evaluation was 33 months (range, 1-198 months). With regard to the pain location, almost 50 percent of the patients (n = 31) had AO localized to the maxillary posterior region, with the left side (n = 32; 50 percent of the total) being more commonly affected than the right side (n = 19; 29.7 percent) or both sides (n = 13; 20.3 percent).

**DISCUSSION**

The literature suggests that AO occurs in 3 to 6 percent of patients who undergo endodontic treatment,<sup>13,14</sup> it has a high preponderance among women and its onset is in the fourth decade of life



**Figure 2.** Types of specialists patients visited before receiving a diagnosis of atypical odontalgia at the Orofacial Pain and Oral Medicine Center, University of Southern California, Los Angeles. TMD: Temporomandibular disorder. ENT: Ear, nose and throat.



**Figure 3.** Patient-reported causes or triggering factors for atypical odontalgia.

with a peak in the fifth or sixth decades.<sup>13,15</sup> In our study population, which was composed of a heterogeneous mixture of patients with complex orofacial pain conditions, we noted a prevalence of 2.1 percent (n = 64). These patients were predominantly female, with a female-to-male ratio of 2:1, and the majority of them were in their fifth decade of life. Generally, molars and premolars are involved more frequently than are incisors and canines, with the maxilla being affected more often than the mandible.<sup>5,15-17</sup> These findings concur with ours about the patients we studied. It is unclear as to why women are affected more commonly than men or why the maxilla is affected more commonly than the mandible. In general, women tend to have a higher preponderance of chronic pain conditions. Sex differences in the function of endogenous pain modulatory systems and hormone levels may be important contributors to greater pain sensitivity and higher prevalence of chronic pain in women.<sup>18,19</sup> The results of studies in animals also have shown that female rats are more susceptible to developing neuropathic pain than are male rats,<sup>20</sup> and ovarian hormones may be an underlying predisposing factor.<sup>21</sup>

In our study, whites (46.8 percent; n = 30) and Hispanics (31.2 percent; n = 20) were affected more commonly with AO than were patients of other races. To our knowledge, data on racial differences in patients with AO have not been published in earlier studies. Los Angeles has a large population of Hispanics, which could explain the higher prevalence of AO we noted in Hispanics in this study. We acknowledge that our sample is a

convenience sample of patients visiting the OFP-OM Center, which is a tertiary center, and readers should view with caution the prevalence data we report here. However, we also note that few or no epidemiologic data regarding this unique population of patients are available.

The cause of AO is unclear, and studies reported in the literature indicate that a majority of cases usually are preceded by a traumatic event to the tooth (such as root canal treatment or extraction), and in the other cases, the precipitating factor is unknown.<sup>5,22</sup> Interestingly, in

our study, 64 percent (n = 41) of the patients reported no causes or triggering factors for their neuropathic pain. Eighty percent (n = 51) of our patients had had some form of dental treatment before being referred and receiving a diagnosis of neuropathic pain. Israel and colleagues<sup>9</sup> published a report regarding 120 patients with chronic facial pain who sought treatment at the Center for Oral, Facial and Head Pain at New York Presbyterian Hospital, New York City, for more than two years. They reported that on average, a patient with facial pain consulted with six specialists regarding pain before being evaluated in an orofacial pain clinic, which is three times higher than the number patients in our study reported (1.7). They also reported a high percentage of patients (40 percent) with atypical facial neuralgia. This high percentage obviously is due to the small sample size (N = 120) used in their study. Endodontics, extractions and apicectomies were the three surgical procedures performed most commonly in their study,<sup>9</sup> which finding is similar to that in our study. Unfortunately, dentists and physicians often mistake neuropathic pain for routine dental pain, and patients are made to undergo additional, unnecessary dental or surgical procedures in an often fruitless effort to ameliorate the pain.

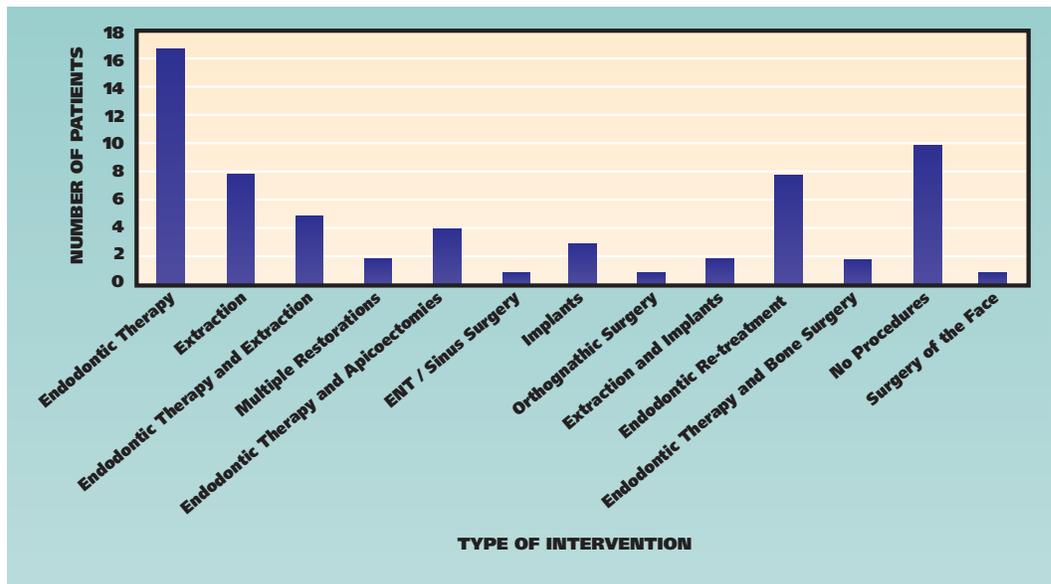
It is unknown as to why some dental patients develop neuropathies when most do not, even in the face of neurotraumatic events that can occur in everyday general dentistry.<sup>2</sup> It is likely that patients with AO may be predisposed to developing neuropathic pain owing to a combination of

genetic, environmental and psychosocial factors. Patients with AO experience neuronal changes at the level of the peripheral or central nervous system, and there are several possible mechanisms underlying these neuronal changes (Box 2).<sup>23-28</sup> AO is difficult to treat and often requires the administration of pain medications such as tricyclic antidepressants, anticonvulsants, serotonin and norepinephrine reuptake inhibitors, opioids, benzodiazepines and anesthetics that target some or most of the aforementioned neuropathic pain mechanisms.<sup>2,5</sup> Unlike the typical pain medications such as opioids, most of these medications have several other indications for use, including treatment of depression, epilepsy or insomnia. In general, these are centrally acting medications that research has shown to be effective in the treatment of AO.<sup>2,5</sup>

**CONCLUSIONS**

Dentists, who are likely to be the first health care providers whom patients with AO consult, must be aware of this condition and must follow the appropriate steps discussed in this article to establish an accurate diagnosis. If the dentist provides dental treatment but the patient's pain persists in the absence of clinically or radiographically evident pathology, then the clinician always must consider AO in the differential diagnosis. Dentists should avoid performing multiple endodontic treatments or extractions, as such procedures often result in failure to control the patient's pain. Dentists should be able to identify patients with AO early in the condition's development and refer them to a dental specialist who has expertise in treating such pain conditions, an orofacial pain specialist or a neurologist for further treatment. ■

**Disclosure.** None of the authors reported any disclosures.



**Figure 4.** Types of dental or medical interventions performed to treat the persistent pain before the patient's visit to the Orofacial Pain and Oral Medicine Center, University of Southern California, Los Angeles. ENT: Ear, nose and throat.

**BOX 2**

**Possible mechanisms for structural and functional neuronal changes in neuropathic pain conditions.\*†**

- Peripheral axonal injury or deafferentation
- Ectopic activity
- Receptor polymorphisms
- Sodium channel upregulation
- Altered gene expression at the trigeminal ganglion
- Sprouting of A-beta fibers
- Activation of glial cells
- Sensitization of wide dynamic range neurons
- Central sensitization
- Activation of N-methyl-D-aspartate neurons
- Suppression of the descending pain inhibitory system

\* Some or all of these mechanisms may occur in patients with atypical odontalgia.

† Sources: Merrill,<sup>23</sup> Benoliel and Eliav,<sup>24</sup> Marchand and colleagues,<sup>25</sup> Scholz and Woolf,<sup>26</sup> Baad-Hansen<sup>27</sup> and Woda and Pionchon.<sup>28</sup>

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