Atypical Odontalgia: Current Knowledge and Implications for Diagnosis and Management

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INTRODUCTION

Quite often patients with a persistent pain in the head and neck region are referred by family physicians to general dentists. The prevalence of chronic and persistent pain in the head and neck region is higher than in other parts of the body,1 therefore, many dentists encounter these conditions during their practice. However, case complexity may lead to improper diagnosis resulting in inadequate or indeed unnecessary dental treatment. To prevent this undesirable situation, a better understanding of the aetiology, diagnosis and management of these chronic pain conditions is desirable for a general dentist. In some cases a proper diagnosis and referral to a specialist may benefit both the patient and the dentist, as well as build up a better relationship between them. One of these complex pain conditions that is not always dealt with properly by dentists is atypical odontalgia (AO).

AO FEATURES

The term AO has generated confusions and controversy but is now defined by the International Headache Society as a subgroup of persistent idiopathic facial pain (PIFP), which is described as "persistent facial pain that does not have the characteristics of the cranial neuralgias and is not attributed to another disorder."2 This condition was first introduced to the medical community by the English anatomist and surgeon, John Hunter (1728-1793). The term AO has been applied to a continuous pain in the teeth or in the vicinity of the tooth socket after extraction or root canal therapy (RCT) in the absence of any identifiable dental cause.2 It can be very distressing for the AO patient, and often may spread and be poorly localized, with words such as diffuse, burning, stabbing or throbbing often used by the patient to describe the pain. The Orofacial Pain Special Interest Group of the International Association for the Study of Pain has revised the old terminology for AO,3 specifying it as "chronic continuous dentoalveolar pain" or CCDAP. This pain condition may constitute a sub-set of neuropathic pain, which has been defined as "pain arising as a direct consequence of any lesion or disease affecting the somatosensory system,"4 since AO is now generally thought to result from injury to sensory fibres supplying the extirpated pulp or extracted tooth. AO has been well characterized recently by Baad-Hansen, List et al., Benoliel et al. and Greene and Murray.3,5-7

Characteristically, AO pain persists during most of the day, it is non-paroxysmal5,7-9 and it can affect both sexes and all adult ages although it has a preponderance for women in their mid-40s. It has been suggested also that genetic predisposition and environmental influences can contribute to the severity of the pain.7-9 There are reports of AO occurring in patents having undergone endodontic treatment, which usually involves extirpation of pulp tissue and injury of the nerves supplying the pulp, or following routine dental treatment, including local anaesthetic administration and dental implant surgery.6,10-12 AO is often mistaken first as a relatively "simple" toothache but after a lack of success in alleviating the pain with regular dental treatment (eg, analgesic drugs or RCT on the suspected tooth), the patient may then receive a series of dental treatments (e.g. extraction, RCT) that do not help relieve the condition and indeed may often exacerbate it.5,7-9 The occurrence of persistent pain for up to 6 months after RCT has been reported in 3% to 12% of patients.10-15 Factors associated with persistent pain are long duration of preoperative pain, marked symptomatology from the tooth, history of chronic pain or painful treatment.6,12,14,15 Extrapolation of the data from the USA to an estimate for the Canadian population indicates that approximately 96,000 of new cases of persistent pain occur following relatively common dental treatment each year, with 61,000 of these cases attributed to AO.14,15

MECHANISMS OF AO

Usually, sensory nerve damage heals uneventfully, but in some patients, possibly with a genetic predisposition, damage to sensory nerves or an actual deafferentation (e.g. loss of the afferent nerve supply or sensory input from the tooth related to a RCT or tooth extraction) can lead to considerable morphological, neurochemical, and physiological changes in the peripheral nervous system (trigeminal ganglion or nerve branches) and/or central nervous system (CNS: brainstem, thalamus, somatosensory cortex). These particular neuroplastic changes have been well documented in research studies in animals.16-19 One of the important mechanisms involved in orofacial neuropathic pain conditions is central sensitization of trigeminal nociceptive neurons in the brainstem. Central sensitization can be produced by nerve damage, such as that associated with pulpectomy or transection of dental nerve fibres, and is reflected as an increase in the neuronal mechanoreceptive field (RF) size, a decrease in mechanical activation threshold and an increase in responses to noxious RF stimuli. Central sensiitzation thus reflects a hyperexcitability of pain processes in the CNS and has been implicated as an important mechanism in acute as

well as chronic pain conditions following injury or inflammation of peripheral tissues.16-19 Several studies have demonstrated the involvement not only of neurons but also of non-neural cells (eg, glia, and cells of immune system) in the development and maintenance of orofacial neuropathic pain states.17,20,21 It is likely that AO involves these changes in the brain, as well as possible changes in peripheral (i.e.,orofacial) tissues. Thus, diagnosis and management of AO needs to take this into account, i.e., that there may be CNS hyperexcitability changes that eventually become maintained independent of sensory inputs from peripheral tissues and that cannot be treated by approaches involving "peripheral" procedures targetting dental or other oral tissues. Indeed, as noted, such approaches may make the condition worse.

DIAGNOSIS OF AO

The diagnosis of AO is often difficult, and is based on exclusion of conditions in teeth or adjacent structures with known pathophysiology.8 The examination includes a routine physical assessment of the head and neck, the masticatory muscles and temporomandibular joints, dental and periodontal tissues and cranio-facial nerves. Diagnostic local anaesthetic blocks may be useful, especially when a local (e.g dental) peripheral pathological process is suspected. Examinations of radiographs and results of cone beam CT are necessary for conclusive assessment, and sensory testing can be used to assess altered sensitivity to mechanical, thermal, chemical and electrical stimuli of dental and periodontal tissues in AO patients since physiological testing7 shows that patients with AO have sensory alterations attributable to peripheral and central sensitization changes. However, no single pathognomonic sensory pattern is associated with AO and so every patient should be assessed on an individual basis. After elimination of all orofacial pain conditions with known aetiology and pathophysiology, a diagnosis of AO may be reached.

MANAGEMENT OF AO

Like other neuropathic pain conditions, AO is often difficult to manage effectively. Treatment of neuropathic pain is complex and challenging, and cannot be restricted to the implicated peripheral tissue. The basis for current management approaches to AO primarily comes from expert opinion and case reports. Some patients cannot accept the fact that the pain they are feeling has been generated in the CNS and not in the tooth or other orofacial tissue where they are feeling the pain, and they look for alternatives which can lead to excessive unnecessary dental treatments, including RCTs, extractions and implant placements, without the desired pain relief.22,23 Thus, it is highly important for general dentists to establish an effective collaboration with specialists who have the appropriate expertise in diagnosing and managing such neuropathic pain conditions. Here, we provide an algorithm that may be useful for AO management; it is based on currently accepted approaches24 (Fig. 1).

CONCLUSIONS

AO is an example of a not-uncommon orofacial neuropathic pain condition which requires thorough examination and proper diagnosis. Late or inadequate diagnosis can lead to unnecessary treatments, exacerbation of symptoms and loss of trust by the patient in the dentist. A consultation with an endodontic specialist or orofacial-pain specialist is advisable, before performing a RCT, dental extraction or some other procedure causing irreversible damage to oral tissues for pain relief in cases when no other symptoms are present and all other possible causes have been ruled out. **OH**

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