report

Atypical odontalgia – a diagnostic dilemma

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ABSTRACT

Atypical odontalgia (AO) is a chronic orofacial pain condition of unclear pathophysiology, often presenting as toothache or pain at an extraction site. Idiopathic, psychogenic, vascular, and neuropathic causes have been proposed. In view of demonstrable somatosensory changes, and responses to management proposed for other forms of neuropathic pain, the best current evidence supports a neuropathic hypothesis. It is proposed that certain individuals with as-yet-undefined genetic vulnerability can develop AO when exposed to certain risk factors, including invasive dental treatment. The diagnosis and treatment of AO can be challenging, but can be aided by a multidisciplinary approach. Two cases of differing complexity are presented in this paper.

INTRODUCTION

Dental and orofacial pain management are core components of dental practice. Most painful problems are managed according to Cartesian principles, pain being considered to arise from activated nociceptive afferents, and impulses transmitted centrally by 'hard-wired' pain pathways (Melzack and Wall, 1996). Reduction of nociceptive output is expected to control the pain. Diagnosis of orofacial pain is not always easy, and convergence of sensory neurons on higher centres can make location and interpretation challenging. Many practitioners will have encountered patients with apparent dental pain without discernible pathology—whether local or referred—and for whom the pain can persist in spite of professional attention (including re-treatment of any perceived cause). Such patients may present again after months or even years with a similar pain problem at the same site, or with persistent pain now spread more widely. This situation can be perplexing, and difficult for even the most experienced practitioner to diagnose and manage.

Atypical odontalgia (AO) is one such enigmatic chronic orofacial pain condition. It is described by the International Society for the Study of Pain as 'severe throbbing pain in the tooth without major pathology' (Merskey and Bogduk, 1994). The Headache Classification Committee of the International Headache Society (IHS, 2004) suggested that AO is a subgroup of persistent idiopathic facial pain (PIFP, formerly known as atypical facial pain), and stated: 'the term AO has been applied to continuous pain in the teeth or in a tooth socket after extraction in the absence of any identifiable cause'. It is a condition for which diagnostic criteria are unsettled, and for which classification is not agreed. The aetiology is controversial, the prevalence unclear, and the management recommendations varied. The clinical features were reviewed by Melis et al (2003), who noted that the maxillary molar and premolar areas were frequently involved. All adult ages can be affected, although there is a preponderance in females in their 40s and 50s. The pain varies in character but is usually described as aching or burning; it can be constant or intermittent, and moderate to severe in intensity. Pain can focus on one tooth or several teeth, on toothless areas, or be even more widespread. The condition usually (but not necessarily) follows deafferentation, such as occurs after pulp removal or exodontia; it can occur weeks or months after such an event. Very occasionally, signs such as facial swelling or reddening suggest sympathetic involvement (Merrill, 2006).

In this paper, we report on pain cases diagnosed as AO in a specialist Oral Medicine clinic.

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CASE ONE

In early 2008 Mr AB, a fully dentate, non-smoking 49-year-old motor mechanic in good health, developed lower right quadrant toothache. The pain was of sudden onset, intermittent, and not affected by eating, or temperature. His horizontally-impacted 48 was carious, and was removed surgically with good healing of the site. After some weeks, his lower right 'toothache' returned. His dental practitioner found that teeth 47 and 46 gave normal cold responses and were not tender to percussion. There was a small carious lesion on tooth 45, which was dressed, but pain persisted. Over the next 9 months, Mr AB visited 4 different general dentists and eventually had teeth 47, 46 and 45 extracted, again without effect on his pain. The final practitioner referred him to the Oral Medicine service at Christchurch Hospital, where he was seen in mid-2009.

He gave no history of pain concerns elsewhere, or of psychological problems. His pain was described as an intermittent dull ache with occasional stabs, rated by the patient as Visual Analogue Scale 4 (VAS – see below), and seemingly related to a specific tooth, but moving to other teeth after treatment. Sleep was not disturbed and the pain initially increased in duration as the day progressed. As weeks passed, the intensity of pain had reduced, however.

On examination, the oral mucosa appeared normal. The mucosa over the extraction sites gave normal response to light touch but was hyperalgesic to pressure. There was no dysaesthesia. Imaging of teeth and jaws was normal. Local anaesthetic blockage completely stopped the pain, as did local application of a eutectic mix of lignocaine and prilocaine (EMLA) on other occasions. The diagnosis of AO was made, and the condition was managed by EMLA applications as needed. The pain faded over the ensuing months, and Mr AB is currently quite free of his 'toothache'.

CASE TWO

Ms VK, a 48-year-old Domestic Purposes Beneficiary, suddenly developed pain in the upper left quadrant. The pulp of tooth 26 was removed by her general dental practitioner and the tooth was root-filled, but her pain persisted. Another

practitioner found deep caries in 37, which was extracted along with tooth 28, but again without effect on the pain. She was then referred by her current dentist to the Oral Medicine service at Christchurch Hospital, where she was first seen in October 2006.

Her general health screening was unremarkable, except for a history of four months of work-related stress. She was smoking 8-10 cigarettes each day. The pain (VAS 5-6) was described as imprecisely located but probably lower left quadrant, dull with episodes of sharp or hot sensation, and present most of the day but not interfering with sleep. It was not affected by temperature, sweetness, or pressure to the remaining teeth. On examination, the mucosa in the upper arch responded normally to light touch and pressure, and standing teeth 24, 25 and 27 gave normal thermal and percussion responses, as did the remaining lower premolar and molar teeth. There was pressure and light touch sensitivity of the buccal mucosa adjacent to the 37 site. No radiographic abnormalities of teeth or jaws were found. Local anaesthesia of the left inferior dental nerve almost completely blocked the pain, and so both topical EMLA and topical capsaicin 0.025% (Vickers et al, 1998) to the 37 site were tried. Pain control was poor and the tricyclic antidepressant (TCA) amitriptyline was prescribed in escalating doses. At 75 mg in divided doses each day, there was good pain control with minimal side-effects. After recall 2 months later, the TCA dose was gradually reduced; when reviewed 4 months later, the patient was pain-free without medication. She was invited to contact the clinic subsequently if needed.

Eighteen months later, she returned to the hospital with a pain pattern and location similar to that described above. She reported that her pain had recurred in various left-sided teeth, many of which had been extracted by various dentists. Tooth 26 had been root-filled but continued to give pain, and she had been referred for management by an endodontist, who was confident that the root-filling was well done. The endodontist did note an area of tenderness to palpation distal to tooth 26. The unusual history alerted the endodontist, who then referred her to the hospital specialist service. However, before her hospital appointment, tooth 26 was removed by yet another practitioner. She had now lost all of her teeth distal to 23 and 34. At the hospital, she was seen on a multi-disciplinary orofacial pain clinic, where sensory testing demonstrated pressure and light touch sensitivity of the painful area, which also included an area of pressure-evoked dysaesthesia. The pain symptoms were only partially reduced by local anaesthetic blockade, and it was felt she had AO with both peripheral and central components. Magnetic resonance imaging for cranial lesions was negative, and her psychosocial screening was unremarkable. After various medication trials, the daily administration of the anticonvulsant gabapentin (2500 mg), the TCA dothiepin (50 mg), and the alpha-2 adrenoceptor agonist clonidine (50 mcg) was found to give excellent sustained pain control. Through the multi-disciplinary service, she has undergone detailed psychosocial assessment with emphasis on pain management strategies, and she manages her pain by distraction and relaxation, supported by medication as needed.

DISCUSSION

There are references in the literature (Marbach et al, 1982) to descriptions by John Hunter (1728-1793) of an orofacial pain condition resembling AO. Wilson (1932) described cases of ‘atypical facial neuralgia’, including individuals with dental pain of unclear origin. McElin and Horton (1947) also described probable AO cases. The first use of the term AO was arguably that by Rees and Harris (1978), who considered the condition to be due to psychological and vascular factors.

Aetiology

The criteria and aetiology for AO are not absolutely defined. This is reflected by the host of alternative terms proposed, including idiopathic odontalgia, persistent orodental pain, phantom tooth pain, idiopathic periodontalgia, trigeminal deafferentation disorder, idiopathic toothache, chronic central trigeminal neuropathy, and neurovascular odontalgia (Greene and Murray, 2011). The term AO does not compellingly reflect present understanding of the condition, with ‘atypical’ and ‘idiopathic’ reflecting uncertainty about the underlying mechanisms. In the same way, the terms ‘psychiatric or psychological disorder’ could be a default diagnosis when there is no current physical explanation for a condition. Nonetheless, the IASP classification of AO (Merskey and Bogduk, 1994), based on earlier articles, implies that AO is the result of emotional problems. While the multi-dimensional nature of pain means that psychological issues cannot be ignored (Baad-Hansen et al 2008), and studies such as that of List et al (2007) show AO pain, somatisation, and depression to be linked, it is not yet clear whether these conditions are because of the pain or or in spite of it. Benoliel at al (1997) considered that some cases of AO could be primarily of vascular origin and had metamorphosed into (or co-existed with) neuropathic-type pains. Somatosensory changes—such as hyperaesthesia, allodynia, and pain exacerbation from temperature, palpation and percussion at the AO pain site—are reported (List et al, 2006; List et al, 2008). Atypical odontalgia can respond to treatment strategies which are useful with other neuropathic pains, supporting the hypothesis that AO is most likely a neuropathic pain. Psychogenic, vascular, and neuropathic pathophysiological mechanisms for AO were critiqued by Baad-Hansen (2008) using research-based evidence, with conclusive support for the neuropathic pain hypothesis.

It is not known why patients develop AO. It seems that some individuals are predisposed to it because of a combination of genetic, environmental and psychosocial factors (Diatchenko et al 2005). Spontaneous onset of AO has been reported (Vickers et al 1998), but the majority of cases follow injury, which can range from a procedure as trivial as polishing, right through to major trauma. Practitioners dealing with persistent dental and orofacial pain should be aware of the mechanisms of orofacial pain (Lavigne et al, 2005), including changes in neural processing which can occur in susceptible individuals, to activate both peripheral and central mechanisms and perpetuate the painful problem (Table 1). Nociceptive pain activates neural pathways in response to potentially tissue-damaging stimuli, while neuropathic pain is initiated by (or associated with) a lesion or dysfunction in the peripheral and/or central nervous system. Neuropathic pain is a ‘malefic force’ serving no protective function, and it is characterised by pain which occurs in the absence of identifiable nociception and is out of proportion to the degree of stimulation (Conti et al, 2003). Chronic neuropathic pain from any source is influenced by the specifics of neural pathology (Table 1), by psychosocial factors, and by genetic predisposition. Often, patients with AO and other chronic orofacial pains have seen a variety of practitioners, and they can be burdened by thoughts that they

Table 1. Mechanisms possibly affecting neural processing (after Gilron et al, 2006).

Peripheral	Central
Nociceptor sensitisation	Spinal V hypersensitivity and reorganisation
Ectopic and spontaneous discharge	Cortical reorganisation
Ephaptic conduction	Changes in inhibitory pathways
Sodium channel changes	
Glial cell activation	
Collateral sprouting of peripheral afferent neurones	
Sympathetic neurone sprouting	
Altered gene expression	

suffer from some 'dreadful disease', that 'no-one else has this problem', that 'it's all in the head', and that 'the pain will never stop.'

Prevalence

Neuropathic pain is reported to affect 1.0-1.5% of the population (Lewis et al, 2007), but there is no information on the population prevalence of neuropathic orofacial pain. Ram et al (2009) reviewed 3000 patient records from a university orofacial pain clinic and found 64 patients (2.1%) diagnosed as AO. Luo et al (2007) reported an AO diagnosis in 1.1% of 95 Hong Kong elderly people with chronic orofacial pain. Marbach et al (1982), and Campbell et al (1990), reported that 3-6% of patients develop AO after endodontic treatment. Polycarpou et al (2005) found 12 of 175 (6.8%) teeth had persistent pain up to 59 months after endodontic therapy; a literature review by Nixdorf et al (2010) reported that all-cause persistent post-endodontic tooth pain occurred in at least 5% of cases. The absolute diagnoses from those 2 reports are not clear, but they suggest that, in some circumstances, invasive therapy with deafferentation (as occurs in pulp removal and exodontia) can cause persistent pain. Oshima et al (2009) reported that 5.9% of 271 patients with persistent post-endodontic pain were diagnosed with neuropathic oral pain. Rodriguez-Lozano et al (2010) reviewed the literature on neuropathic pain following implant placement, and found one case. In contrast, Berge (2002) reviewed 1458 patients who had had impacted third molars surgically removed up to 81 months previously; 23 (2.2%) had long-term pain symptoms, but none met the criteria for AO.

Diagnostic Criteria and Differential Diagnosis

The several published diagnostic criteria for AO differ in details, but the simple scheme proposed by Pertes and colleagues (1995) is widely accepted. Presented here in modified form, it includes the following: (1) pain in tooth or tooth site which may spread to the upper or lower jaw, or wider area; (2) aching, burning, throbbing or shooting (the character can vary); (3) continuous or almost continuous pain, with variable intensity; (4) pain present longer than four months; (5) no sign of local or referred cause, and imaging normal; (6) pain may involve dysaesthesia, hyperalgesia or allodynia of the overlying area; (7) pain has a variable response to local anaesthesia; (8) pain is unresponsive to analgesics, surgery, dental procedures; and (9) the usually (but not necessarily) follows a dental procedure or trauma.

Atypical odontalgia is largely a diagnosis by exclusion, and the differential diagnosis is most important. The history and clinical examination are crucial. Dental disease and pain from maxillary sinuses, temporomandibular joints (TMJ) or the masticatory muscles are part of the differential diagnosis. It can be difficult to differentiate pulpal pain from AO, but five differentiating characteristics of AO have been listed by Okeson (1996): (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; (4) 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; and (5) response to local anesthesia is equivocal.

Trigeminal neuralgia (TN) can usually be ruled out by symptomatology, especially because the refractory period that follows pain paroxysms in TN is absent in AO. Some primary headaches can also present with AO-like symptoms. Other listed causes (after Melis et al, 2003) include cracked tooth syndrome, pretrigeminal neuralgia, ear and eye problems, migrainous neuralgia, cranial neuralgias, cervical referral, pre-eruptive herpes zoster, post-herpetic neuralgia, geniculate neuralgia, temporomandibular disorders, and cytotoxic chemotherapy-induced odontalgia (Zadik et al, 2010). Somatosensory changes at the AO pain site can be initially assessed by using light touch, sharp/blunt, percussion, palpation and cold stimuli. Cold allodynia with prolonged pain duration is a common feature of neuropathic pain states, and was reported for AO by Zagury et al (2011).

Supplementary investigations include imaging for intra-cranial pathology, neurophysiological testing, and neurological review. A Visual Analogue Scale (VAS) can be used to assess pain magnitude, which is marked as a number on a straight line (usually 10 cm long and divided into 10), with 0 representing no pain and 10 the worst pain imaginable (Price et al, 1983). The Symptom Checklist 90-revised (SCL 90R; Dworkin et al, 1992) is another tool available to assess the impact of pain and help to establish the role of psychological factors. Other tools which can be used include the McGill Pain Questionnaire and the Chronic Graded Pain Scale. (Dworkin, 2001).

Local anaesthesic blockade may help distinguish between peripheral and central trigeminal neuropathic changes. The anatomy of the trigeminal nerve pathways is complex. Sensory information (except proprioception, which goes to the mesencephalic nucleus) travels by primary afferent fibres to the trigeminal sensory complex in the brain stem. Here, sensation can be confounded by convergence (notably in the subnucleus caudalis of the V spinal tract nucleus) of afferent axons from the facial, glossopharyngeal, vagus, and upper cervical nerves. Second-order neurons then convey information via the spinothalamic tract to the thalamus, where information is projected via third-order neurons to the various parts of the somatosensory cortex for further interpretation and action.

Complete relief following local anaesthetic blockade suggests first-order sensitisation; incomplete relief suggests second- or third-order level changes, and no relief suggests either central sensitisation or a faulty diagnosis (Merrill, 2006; List et al, 2006). First-order sensitisation may respond to local therapy alone.

Spontaneous pain from sensitised c-nociceptors may present as burning or sometimes pricking pains, and it may include allodynia and/or dysaesthesias. Hyperalgesia to point stimulation and to pressure involves both c-fibres and A-delta fibres. A-beta fibres are important in allodynia, paraesthesia and dysaesthesia, which can be pressure- or brush-evoked. These sensory testing techniques require specific training and calibration to be valid and reliable, but they may demonstrate the underlying mechanism of the pain, and (theoretically at least) enable practitioners to direct treatment to mechanism- rather than symptom-orientated care (Lavigne et al, 2005).

Treatment/management

Invasive procedures are to be avoided during the treatment of AO because there is a real risk of aggravating the pain. Patients (and practitioners) may have difficulty in accepting the diagnosis of AO, perhaps feeling there is deficiency in the practitioner's diagnostic method and capability. There are no cures, and pain management is the goal of treatment, using methods in accord with evidence-based forms of neuropathic pain management. This is best carried out in a step-wise manner, commencing with patient education and information.

If first-order sensitisation has been demonstrated by LA blockade, peripheral techniques can be trialled. There are reports of repeated local anaesthetic injection being useful for pain control (Marbach et al, 2000). Topical medication (such as EMLA and/or capsaicin cream applied over some weeks) may also be of value (Vickers et al, 1998; Heir et al, 2008). If peripheral response is unsatisfactory, treatment can progress to TCA (such as amitriptyline or nortriptyline), selective serotonin reuptake inhibitors (SSRI; such as fluoxetine), or serotonin-noradrenaline reuptake inhibitors (SNRI; such as venlafaxine; Melis and Secci, 2007). Although these are mood-altering medications, their effectiveness is attributed to low-grade analgesia. Anticonvulsants such as gabapentin and pregabalin are suggested as third-line medication for neuropathic pain; off-label use of sodium valproate, carbamazepine, clonidine, or lamotrogine can also be used (Clark, 2006). If necessary, the addition of Tramadol or oxycodone is advocated by some authorities (Finnerup et al, 2005). Many of these medications require careful management and monitoring. Patients with evidence of sympathetically-maintained pain may well need referral for consideration of stellate ganglion block

Adjunctive therapies have been advocated in other forms of neuropathic pain, including transcutaneous electrical nerve stimulation (TENS), acupuncture, cognitive behaviour therapy, and self-hypnosis (Gilron et al, 2006). There are few adequately structured trials on these agents, and there is a paucity of information about their use in AO.

Patient beliefs and cognition affect not only their understanding and symptom interpretation but also their pain modulation, and psychological risk factors need identifying. A symptom/mechanism-based approach can depersonalise patient pain management, yet the physical component may be overlaid by psychosocial issues. Patients with AO may have other chronic pain problems, and unusual pain behaviours, depression, and somatisation can further complicate management. Pain is a highly personal multimodal experience, and when AO is not dealt with by simple measures, specialist referral is prudent. It is unlikely that any single clinician will have the skill and experience to deal with the multiple pathophysiological processes underlying AO and other neuropathic orofacial pains; multidisciplinary management offering comprehensive care (including coping skills and problem-solving techniques) is appropriate for cases which do not respond to simpler forms of treatment.

Future Developments

With advances in understanding of the mechanisms of neuropathic chronic pain, there are prospects of more focused diagnosis and treatment. Quantitative sensory testing (QST) assesses not only the fibre types involved, but also the degree of nerve damage (Eliav et al, 2004; List et al, 2006). When coupled with QST, clinical neurophysiological recordings—such as blink reflex and evoked sensory nerve potentials—are reported to give a favourable diagnostic yield in cases of chronic trigeminal sensory damage (Jääskeläinen, 2004). However, these technique-sensitive methods are presently best carried out in specialised units. Medications with actions specific to the location of changes in the peripheral and central nervous systems are increasingly available, and these may permit some pharmacological targeting of pain mechanisms. There are no structural or functional brain imaging techniques yet reported for AO, but, in the related condition PIFP, changes in the dopaminergic system have been shown by positron emission tomography (Hagelburg et al, 2003). To date, little is known about genes predisposing to chronic neuropathic pain, although Nissenbaum et al (2010) reported an association between the gene CACNG2 polymorphism and chronic neuropathic pain following breast surgery. The genetic basis for variations in pain perception, and the likelihood of developing a chronic pain condition, is also reported. For example, Diatchenko et al (2005) have shown that the likelihood of developing a myogenous temporomandibular disorder (TMD) is linked to the gene which encodes catecholamine-o-methyl transferase (COMT) activity. In addition, there is emerging evidence of an association between overall autonomic dysfunction and chronic pain disorders (described for TMD by Maixner et al, 2011). The relationship between these findings and other phenotypic risk factors and genetic polymorphisms is currently unclear. It could be that AO affects individuals with as-yet-undefined genetic vulnerability who develop neuropathic pain when exposed to certain risk factors (such as environmental and/or psychosocial stressors). In the future, genetic testing may identify these individuals, perhaps leading ultimately to preventive management strategies. As with all forms of chronic pain, the impact on quality of life can be considerable. The way dental and maxillofacial surgeons in a certain European country ignore the principles of multimodal and interdisciplinary pain management, in spite of various guidelines, has been criticised by Wirz et al (2005). There is no information on the NZ situation, but access to integrated multimodal teams dealing with all facets of the pain experience is prudent in the management of chronic pain, including AO.

Atypical odontalgia may not be common, but it behoves practitioners to be alert to the condition as a non-dental cause of orofacial pain.

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