



Differential diagnosis of non-odontogenic pain in the oral cavity

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Introduction

A survey of 42,370 adults in the United States showed that 9,072 individuals (21%) had experienced orofacial pain more than once in the last 6 months.¹ While 12.2% of the respondents had pain of odontogenic origin, almost 7% had pain involving the TMJ and other orofacial structures. Another study found that nearly one third of patients referred to a tertiary pain center for chronic orofacial pain had undergone prior surgical procedures to address their symptoms.² The procedures consisted of endodontics (30%), extractions (27%), apicoectomies (12%), TMJ surgery (6%), neurolysis (5%), ortho-gnathic surgery (3%), and debridement of bone cavities (2%). Many of the patients actually felt their pain was worse following the surgical interventions. The majority of the patients were diagnosed by the pain center as experiencing either muscle-related pain (50%) or atypical facial pain (40%). Both of these studies suggest there are a substantial number of patients who may present to the dentist with orofacial pain complaints of non-odontogenic origin and with conditions that are unresponsive to surgical procedures.

Establishing the correct diagnosis is essential before rendering treatment. Well-intentioned therapy for the wrong diagnosis can be ineffective and may exacerbate the existing condition. When evaluating a pain complaint, it is essential for the dentist to be knowledgeable of odontogenic sources of pain as well as other structures or conditions with the potential to refer pain to the orofacial region. Non-odontogenic orofacial pain is frequently of musculoskeletal, neurovascular, neuropathic or vascular origin.

Musculoskeletal Pain

It is important to consider the potential of referred muscle pain when a patient's symptoms and the findings of an appropriate examination are not consistent with an odontogenic source of pain. Masticatory muscles are probably the most common cause of chronic orofacial pain and often produce referred pain to the temporomandibular joint, ear, and oral cavity. Myofascial pain is characterized by trigger points – focal, tender, muscle sites capable of eliciting referred pain. Trigger points in the masseter and temporalis muscles can refer pain to teeth and adjacent structures. While the temporalis muscle refers pain to maxillary teeth, the masseter muscle can refer pain to both maxillary and/or mandibular teeth.³

If muscle palpation reveals the presence of a trigger point that reproduces the patient's chief complaint, the injection of a local anesthetic, such as 2% lidocaine without epinephrine, into the trigger point may relieve the pain.

Neurovascular Pain

Neurovascular headaches may produce symptoms that mimic odontogenic pain. Migraine, cluster, and paroxysmal hemicrania are types of trigeminovascular headaches that might prompt a patient to seek a dental evaluation.⁴ Obtaining a proper history is paramount in establishing the correct headache diagnosis. Migraine headaches typically last 4 to 72 hours, are unilateral, have a pulsating character and produce moderate to severe pain. Patients with migraines report nausea and/or photophobia-phonophobia and may relate that physical activity worsens the pain. Some patients experience an aura – a constellation of visual, motor, sensory or cognitive disturbances that precede the headache.

Cluster headaches are less common than migraines and usually occur in males between the ages of 20 and 40. Patients with cluster headaches describe intense pain lasting for 15 to 90 minutes involving the supraorbital, temporal and malar regions. Attacks can occur daily with 1 to 6 episodes per day. Cluster headaches are associated with autonomic symptoms such as conjunctival injection, lacrimation, nasal congestion, tearing or eyelid edema. These headaches can awaken patients from sleep and may be triggered by drinking alcohol. Paroxysmal hemicrania headaches are similar to cluster in location, signs and symptoms. Unlike cluster, paroxysmal hemicrania headaches are found twice as frequently in women, are of shorter duration (2-25 minutes), and may occur with greater frequency (up to 40 times per day). The resolution of headache symptoms with a trial of indomethacin is considered pathognomonic for paroxysmal hemicrania.

Neuropathic Pain

Neuropathic (nerve generated) pain can be divided into episodic and continuous forms. Trigeminal neuralgia is an example of episodic neuropathic pain. It most often begins in the 5th to 7th decades, has a unilateral presentation and usually involves the maxillary or mandibular division of the trigeminal nerve. The pain is frequently described as severe, electric or shock-like and lasts only a few seconds. Trigeminal neu-

ralgia may have intra- or extraoral trigger zones or areas where non-noxious stimuli can precipitate the onset of the pain. Although the cause of trigeminal neuralgia is often idiopathic, when it occurs in patients under the age of 50, appropriate medical evaluations should be performed to rule out intracranial neoplasm or multiple sclerosis.

Post-viral neuralgia is an example of continuous neuropathic pain. Such pain is usually characterized as a continuous, burning sensation of variable intensity and is often accompanied with allodynia – pain in response to light touch. Patients with a history of varicella-zoster virus infection, or “chickenpox,” may experience cutaneous reactivation of the virus known as herpes zoster or shingles. Viral reactivation results in the painful eruption of vesicles; however, the pain may remain long after the skin lesions have healed.

Neuropathic pain can also arise from trauma to the nerve or nearby tissues. The injury can range from mild to severe and may occur secondary to normal dental treatment, such as root canal therapy or the extraction of teeth. Traumatically induced peripheral neural changes include alterations in nerve membrane channels, nerve sprouting or neuroma formation. Peripheral injury can also produce a variety of cellular changes in the central nervous system, a process referred to as central sensitization, leading to clinical complaints of decreased pain thresholds and/or spontaneous pain.⁵ Either peripheral and/or central neural changes can result in lingering pain long after the initial tissue injury has healed. Traumatically induced neuropathies may be mistaken for protracted post-operative pain; thus, whenever pain persists beyond normal healing times, the possibility of a neuropathic pain should be considered.

Vascular Pain

Giant cell arteritis is a vascular pain and is termed temporal arteritis when the superficial temporal artery is affected. Giant cell arteritis occurs more often in women and has an average age of onset of 70 years. Palpation of the superficial temporal artery reveals the area to be extremely sensitive and the artery may appear to be erythematous and swollen. The etiology of giant cell arteritis is unknown. The disorder is characterized by chronic inflammation of the walls of mid-sized arteries leading to narrowing of vessels and ischemic complications.⁶ The facial pain symptoms of giant cell arteritis are usually unilateral, can be mild to severe, and have a throbbing to burning quality. In addition, the patients may complain of masticatory muscle weakness or pain with chewing, termed “jaw claudication.” Since giant cell arteritis may involve the ophthalmic artery and lead to loss of vision, suspected patients should be referred to their physician.

Miscellaneous

Atypical or idiopathic facial pain is a diagnosis of exclusion. The term is applied only after all other sources of pain or pain referral have been ruled out. Atypical facial pain has no iden-

tifiable etiology, does not follow the typical sensory nerve distributions and may spread to other areas of the head and neck. Many previously reported cases of atypical facial pain were most likely unrecognized forms of neuropathic pain. Atypical facial pain is more common in females than males and commonly occurs in the maxillary region. The pain is characterized by a continuous, diffuse, deep, constant ache that does not awaken the patient from sleep.⁷ When atypical facial pain involves the teeth, it is called atypical odontalgia. Patients with atypical odontalgia may erroneously seek endodontic therapy or extraction of the perceived painful teeth.

Conclusion

Orofacial pain may have many etiologies: odontogenic, musculoskeletal, neurovascular, neuropathic and vascular. Because of the diversity of orofacial pain conditions, it is incumbent on the dental provider to obtain a thorough history, perform a comprehensive examination and conduct appropriate diagnostic tests, i.e. diagnostic local anesthesia. If the evaluation does not identify an odontogenic source of pain, the dental provider should avoid performing invasive dental procedures that might obscure the true diagnosis, exacerbate existing symptoms and complicate further treatment.

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