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NEUROPATHIC PAIN CONDITIONS AFFECTING TEETH- A REVIEW FOR GENERAL DENTIST

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ABSTRACT

The majority of toothaches originate in either the dental pulp tissues or the supporting periodontal structures. These pains of dental origin (odontogenic) are reasonably easy to diagnose and can be managed with regular dental procedures which includes endodontic treatment or extractions. Nonodontogenic neuropathic pains in the maxillofacial complex are challenging, as the differential diagnosis can include paroxysmal conditions. Frequently, dentists may face the complex challenge to diagnose and treat pain of a neuropathic origin in the orofacial region. This article reviews management of various neuropathic pain conditions that affect orofacial structures, including such conditions as atypical odontalgia (AO), trigeminal neuralgia (TN), and glossopharyngeal neuralgia (GPN). The above conditions should be considered during an endodontic evaluation when patients complain of dental pain with no obvious pathology. To determine the source of the pain, the dentist needs to obtain a detailed medical history that includes information about the condition's duration, severity, pattern, and relieving factors. A thorough intraoral and extraoral clinical examination is mandatory to rule out odontogenic causes. Based on the pain history and the clinical examination, radiographic and other investigative procedures may be necessary to diagnose the condition. A good knowledge of nonodontogenic causes of pain may prevent unnecessary irreversible dental treatment. The diagnosis and management of nonodontogenic pain usually requires a multidisciplinary team approach involving a dentist, a neurologist, and an otolaryngologist.

Key words: **Neuropathic pain, Neuralgia, Odontogenic pain.**

INTRODUCTION

Neuropathic pain is the result of damage to or dysfunction of the peripheral or central nervous system, rather than stimulation of pain receptors. Chronic neuropathic pain conditions are more common in the head and neck region rather than in other parts of the body.[1] These pain conditions may affect dental tissue (including teeth), which means that dentists should know and understand these conditions before rendering any treatment. Unlike transmission of impulse in nerves (which is a response to disease or trauma), neuropathic pain is caused by the pathology (Disease), which actually affects the nerves. Orofacial neuropathic pain occurs in the absence of obvious pathology. Usually Orofacial neuropathic pain is chronic and difficult to localize, and its quality of pain is diverse. Unlike physiological pain, which warns of noxious stimuli, orofacial neuropathic pain can be paroxysmal or continuous in nature and serves no protective function.[2,3,4,5]

Dental patients commonly complain of a "toothache"; usually these kinds of pains are mostly odontogenic, originating in the pulpal tissues or periodontal structures. The pains of odontogenic origin are relatively easy to diagnose and predictable to treat. In contrast, nonodontogenic pains are difficult to identify and are challenging a dentist's diagnostic ability. A wrong diagnosis could lead to irreversible treatment, such as endodontic therapy and extractions. This article is an attempt to review various neuropathic pain conditions that affect orofacial structures (including teeth) and are of clinical significance to dental and endodontic practice.

I. Atypical odontalgia

Atypical odontalgia(AO) is a perplexing clinical condition that can challenge clinicians. The condition was first described by Hunter more than 200 years ago but was not reported in the literature until 1947.[2] Although AO is an accepted clinical condition, these cases are often overlooked in the differential diagnosis and are rarely reported.[3,4] The International Association for the Study of Pain (IASP) has defined AO as "a severe throbbing in the tooth without major pathology".[5] Researchers and investigators have used the terms neurovascular odontalgia, oral neuropathic pain, atypical facial pain, and facial arthromyalgia and idiopathic odontalgia to describe AO.[4,5,6,7]

Clinical appearance

The classical manifestation of AO includes throbbing, persistent pain in teeth, or an alveolar process occurring over a long period of time without any pathological, clinical, or radiological findings.[1] AO usually follows dental or surgical procedures such as pulp extirpation, apicoectomy, tooth extractions, or removing of the contents of the maxillary antrum.[8] It is usually idiopathic, making its diagnosis a challenge. Although the pain is chronic in nature, the patients sleep is undisturbed, and there may even be a brief symptom-free period upon waking.[7] Although the incidence of AO is higher in the maxillary molar and premolars, the condition can affect patients of all ages (except children), with a preponderance among women in their mid-forties.[9,10]

Patients with AO rarely find relief from analgesics, including narcotics.[7,11] Results from a 2004 study that utilized a diagnostic local anesthetic infiltration and block were mostly inconclusive, since the local anesthetic infiltration and block of the involved tooth produced equivocal pain relief. The results of this study indicated that a diagnostic local anesthetic infiltration and block cannot indicate the presence or absence of AO.[1]

Two conditions that have been associated with AO are osteoporosis that appears during menopause and neuralgia inducing cavitational osteonecrosis (NICO), which has been reported in post-menopausal women.[10] The theory for facial neuralgias postulates that a low grade osteomyelitis of the jaws produces neural degeneration and subsequently produces inappropriate pain signals. These conditions are very rare and should be considered only when other causes of nondental toothache have been ruled out.

The patient's psychological status has been reported as a cause of AO, but there is no scientific evidence to support this claim.[12]

Diagnosis and pathogenesis

As AO is a chronic form of dental pain that typically does not display obvious signs of tooth or periodontal pathology, the condition is usually diagnosed by exclusion.[1] The diagnostic criteria as proposed by Graff-Radford and Solberg for AO includes pain in a tooth or a tooth site, continuous or almost continuous pain, persistent pain for more than four months, no local or referred pain, and an equivocal somatic nerve block.[1] Pertes et al., (1995), revised the criteria to include a lack of response by pain to treatment.[11]

The exact mechanism through which pain is generated by AO is ambiguous. In 1978, Marbach hypothesized that AO was similar in etiology to phantom limb pain.[13,14] A 1993 study reported that organization and activity of central and peripheral nerves can change after injury, resulting in neuropathic pain.[14] Other mechanisms involved in the pathogenesis of AO pain include sensitization of pain fibers, cross-activation of afferents, and loss of inhibitory mechanisms.[l,10,11,12,14,15] Different neuropathic mechanisms may be involved, including nociceptor sensitization, phenotypic changes, and ectopic activity from the nociceptors. Central sensitization possibly maintained by ongoing activity from initially damaged peripheral tissues, sympathetic abnormal activity, alteration of segmental inhibitory control, and hyper or hypoactivity of descending controls can also contribute to the pathophysiology of AO.[16]

Nociceptors are sensory neurons, found in any area of the body, that can sense pain either externally or internally. Although nociceptor sensitization has been used to explain the pathophysiology of AO (along with other theories like central sensitization and sympathetic abnormal activity), the exact mechanism is still unclear. Woda and Pionchon hypothesized a concept, that the idiopathic pain depends on one or several neuropathic mechanisms, the development of which is triggered or favored risk factors; these risk factors are local inflammation, infections, or mechanical irritation as well as minor nerve trauma. The authors proposed that these different mechanisms may be at work at the same time.[10] In 1993, Marbach noted the similar etiology between AO pain and phantom limb pain.[14]

Treatment

Treatment for AO is similar to treatment for other neuropathic conditions. The drug of choice is a tricyclic antidepressant such as amitriptyline, alone or in combination with a phenothiazine. The result is usually good, with patients obtaining complete relief from pain.[12] Treatment is started with a low dose (20-75 mg) of amitriptyline that needs to be adjusted depending on pain control and adverse reactions. The dose is adjusted to that particular patient until an acceptable pain level is achieved; however, the side effects associated with amitriptyline (including behavior changes, anxiety, panic attacks, and drowsiness) can cause the clinician to switch to a different medication within the same category.[12] Although other tricyclics such as imipramine, nortriptyline,

and dothiepen have been suggested for treating AO, low doses of amitriptyline are used more frequently.[12]

Other medications (such as gabapentin, clonazepam, baclofen, doxepin, a- and p-blockers, and monoamine oxidase [MAO] inhibitors) have produced successful results, in patients who could not tolerate tricyclic antidepressants.[1,12] Topical capsaicin was used successfully treat neuropathic pain; it is a simple yet effective treatment for patients with AO.[17] It is important to defer any invasive procedures until a definitive diagnosis of AO has been reached.

II. Trigeminal neuralgia

Trigeminal neuralgia (TN) (also known as tic douloureux) is a neuropathic disorder of the trigeminal nerve that causes episodes of severe pain in the eyes, lips, nose, scalp, forehead, and jaws. The IASP defines classical idiopathic TN as a sudden (usually unilateral), severe, brief, stabbing, recurrent pain that is distributed to one or more branches of the fifth cranial nerve.[18]

Clinical appearance

A patient whose medical history includes sometimes incapacitating pain that recurs and remits frequently could be considered to have TN; however, due to the nature of its initial presentation, the condition is some times wrongly associated with dental pathology. The pain follows the V1, V2, or V3 branches of the trigeminal nerve and the pain can last from a few seconds to minutes; the patient usually does not have pain during intervals between attacks. As the time passes, the pain episodes increase in severity and frequency. The paroxysms of pain can occur in rapid succession while the patient is awake, but they rarely occur when the patient sleeps.[19,20] TN is characterized by spontaneous remissions that may last months or even years.

Epidemiologic information indicates that TN affects 4-5 people per 100,000 and has a predilection for women over the age of 40.[21] TN attacks can be attributed to the stimulation of trigger areas around the nose and mouth ipsilateral to the pain.[16] TN adversely affects a patient's lifestyle, as attacks can be provoked by simple stimuli such as talking, chewing, light touch to the face (during shaving or washing). In severe cases, extreme weather changes may trigger an attack. Dentists should consider TN when dental treatment fails to provide the intended long-term pain relief.[22] Because early diagnosis is difficult, many patients may go untreated for long periods and seek treatment from many sources before a definitive diagnosis of TN is made; once a definitive diagnosis is reached, the patient should be informed as to both medical and early neurosurgical options.[23,24]

Pathogenesis

Although many theories have been proposed to explain the pathophysiology of pain in TN, the exact mechanism of pain is still unclear. It has been postualated that demyelination resulting from tumor or vascular compression may lead to abnormal transmission and processing of impulses along the trigeminal nerve.[23] Focal areas of axonal demyelination resulting from nerve compression can generate spontaneous action potentials that travel in either direction along the nerve. Spontaneous activity, after discharges, and abnormal coupling between primary afferents may be important mechanisms in TN.[23,24,25]

Most idiopathic TN cases are now understood to be caused by vascular compression of the trigeminal nerve. This vascular compression leads to demyelination of the nerve's sensory fibers. Histopathological examination of the trigeminal nerve in patients with TN reveals focal loss of myelin, close apposition of the demyelinated axons, and lack of intervening astrocytic processes.[18,22,26] The vascular compression theory has been supported in studies that utilized MRI to document the presence of benign or malignant lesions, plaques of multiple sclerosis, and proximity of vessels to the trigeminal nerve.[27] Similarly, intraoral compression of the mental nerve by an ill fitting denture can lead to TN like symptoms. Gazzeri et al described a case of atypical TN associated with tongue piercing in which the condition resolved after the jewelery was removed.[28] It has been said that chronic irritation of or trauma to one of the trigeminal nerve branches can cause ectopic action potentials and failure of segmental inhibition, leading to symptoms of TN.[29]

Diagnosis and treatment

Because no single test can diagnose TN, diagnosis is confirmed by ruling out other conditions. Although the diagnosis remains based exclusively on history and symptomatology, modern diagnostic techniques (particularly high resolution MRI) have provided insights into the pathophysiology of these cases that can affect therapeutic strategies.[30] Relief of symptoms following administration of anticonvulsant medications (such as carbamazepine and gabapentin) confirms a diagnosis of TN. All patients with TN should undergo a brain MRI and repeated neurological evaluations to rule out the presence of underlying disease.

Drug therapy is the main treatment option. Various anti-epileptic drugs are used, including carbamazepine, oxcarbazepine, lamotrigine, phenytoin, and gabapentin.[23] Carbamazepine is most often the drug of choice; however, it can produce adverse effects, including sedation, fatigue, dizziness, blurred vision, nausea, vomiting, and allergic skin reactions.[31] Hematologic and hepatic profiles must be monitored routinely during treatment. Oxcarbazepine is a safer alternative to carbamazepine, since it does not require complete blood cell count and liver function tests.[22,32] Oxcarbazepine has fewer drug to drug interactions and does not autoinduce its metabolism; hence, dose titration and adjustment are relatively simple,

making it possible to achieve steady-state levels of the drug.[32] Other effective medications include baclofen, a y-aminobutyric acid receptor agonist, and tizanidine, an a adrenergic agonist.[22] For patients with TN that does not yield to pharmacological treatment or for those who cannot tolerate the adverse effects of drug therapy, surgical intervention is recommended. Surgical options include peripheral surgery, percutaneous ablative procedures, stereotactic radiosurgery, or microvascular decompression.[22]

III. Glossopharyngeal neuralgia

According to a 2005 study, only 0.2-1.3% of cases of facial pain are diagnosed as Glossopharyngeal neuralgia (GPN).[22] Since otolaryngologists and head and neck surgeons are consciously aware of the presence of the glossopharyngeal, facial, vagus, accessory, and hypoglossal nerves, they are more likely to recognize and treat this condition.[33] Dentists may consider this diagnosis, especially when the patient complains of pain when chewing. GPN was first described by Weisenberg in 1910 when discussing a patient with a cerebellopontine angle tumor.[34] In 1920, Sicard and Robineau described three patients who had pain in the area of distribution of the glossopharyngeal nerve without any known cause.[35]

Clinical appearance

GPN is a syndrome characterized by paroxysms of unilateral and severe lancinating pain occurring in the distribution of the nerve; this pain may be elicited by stimulating trigger points in the area of cutaneous distribution of the nerve.[36] The pain may be spontaneous or precipitated by a variety of stimulating actions, such as yawning, coughing, swallowing, talking, and (on rare occasions) chewing. GPN occurs only in adults, with a predilection for females and for patients over the age of 50.[37] GPN can have a sudden, abrupt onset, characterized by paroxysms of unilateral pain along the path of the glossopharyngeal nerve that affect the left side more frequently than the right.[38] This paroxysm of pain has been described as needle-like, sharp, and severe, lasting seconds to minutes in the distribution of the nerve, and sometimes involving the angle of the mandible and the retromolar region.[33,37]

GPN has been divided based on the distribution of pain into two clinical types: the tympanic type (affects the ear) and the oropharyngeal type (affects the oropharyngeal area).[33] It is difficult for patients with GPN to identify the triggering zones as they may be present in deep structures of the mouth, pharynx, and ear.

Patients with GPN may experience pain remission for a period ranging from months to years; the remissions are irregular in nature.[38] The acute phase is generally brief, lasting from a few seconds up to one minute and sometimes followed by syncopal episodes.[37] During paroxysms resulting from the intense GPN pain, patients may experience pallor, followed by hypotension associated with bradycardia, which can lead to a loss of consciousness and associated tonic-clonic limb jerking movements.[33,38,39]

Pathogenesis

GPN usually is idiopathic, and a thorough clinical head and neck examination usually does not reveal any abnormality other than the identification of trigger points.[33] In addition, radiological examinations (including CT and MRI scans) may be within normal limits.

GPN could be caused by vascular compression of the glossopharyngeal nerve at the nerve root entry zone (a condition by name vascular compression neuropathy), which is treated very effectively by microvascular decompression procedure of the relevant vessels. Although the majority of GPN cases are idiopathic (that is, they occur in isolation), they may be secondary to cerebellopontine angle tumors, intracranial vascular compression, carcinoma of the laryngeal and nasopharyngeal tumors (spreading locally), parapharyngeal abscess, cranial base tumors, calcified stylohyoid ligament, direct carotid puncture, trauma, multiple sclerosis, Paget's disease, and dental extractions.[34,36]

Diagnosis and treatment

After obtaining a detailed patient history, the dentist must establish a diagnosis and exclude other causes of pain, including neoplasia. In the absence of any obvious signs during a clinical examination, it is important to exclude intracranial lesions. A brain MRI or CT scan may help in diagnosing intracranial lesions that may have caused secondary GPN.

The first line of treatment includes antiseizure medications such as carbamazepine, gabapentin, or phenytoin. Analgesic action from an anti epileptic drug was first reported in 1942, and this action formed the rationale for the use of these drugs in neuropathic pain conditions. The rationale involves a monitored use of the drugs and clear evidence based guidelines for the pharmacologic management of neuropathic pain that take into account impact on health related quality of life, convenience, clinical efficacy, adverse effects and costs. Antidepressants like amitriptyline also have been tried, with varying degrees of success.[40] When the cause of neuralgia is identified, the dentist should initiate appropriate medical or surgical treatment. Surgical options, including nerve resection, tractotomy, or micro-vascular decompression, should be considered when patients either do not respond or stop responding to drug therapy.[36,41]

CONCLUSION

When neuropathic pain in the orofacial region affects teeth, treatment should start with a detailed patient history and a thorough clinical examination. The severity of pain and peculiar symptoms can help to diagnose neuropathic

pain of nonodontogenic origin. The management of orofacial pain is a challenge to dentists; severe cases of pain should be treated by a multidisciplinary team drawn from several specialties (including dentistry, neurology, otolaryngology and psychiatry). Early diagnosis of nonodontogenic neuropathic pain can improve the chances of successful management and prevent unnecessary irreversible dental treatment.

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