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Zeroing in on the cause of your patient's facial pain

The overlapping characteristics of facial pain can make it difficult to pinpoint the cause. This article, with a handy at-a-glance table, can help.

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PRACTICE RECOMMENDATIONS

› *Advise patients who have a temporomandibular disorder that in addition to taking their medication as prescribed, they should limit activities that require moving their jaw, modify their diet, and minimize stress; they may require physical therapy and therapeutic exercises.* (C)

› *Consider prescribing a tricyclic antidepressant for patients with persistent idiopathic facial pain.* (C)

Strength of recommendation (SOR)

(A) Good-quality patient-oriented evidence

(B) Inconsistent or limited-quality patient-oriented evidence

(C) Consensus, usual practice, opinion, disease-oriented evidence, case series

Facial pain is a common complaint: Up to 22% of adults in the United States experience orofacial pain during any 6-month period.[1] Yet this type of pain can be difficult to diagnose due to the many structures of the face and mouth, pain referral patterns, and insufficient diagnostic tools.

Specifically, extraoral facial pain can be the result of temporomandibular disorders, neuropathic disorders, vascular disorders, or atypical causes, whereas facial pain stemming from inside the mouth can have a dental or nondental cause (**FIGURE**). Overlapping characteristics can make it difficult to distinguish these disorders. To help you to better diagnose and manage facial pain, we describe the most common causes and underlying pathological processes.

Extraoral facial pain

Extraoral pain refers to the pain that occurs on the face outside of the oral cavity. The **TABLE**[2-15] summarizes the site, timing and severity, aggravating factors, history and exam findings, and management of several common causes of extraoral facial pain.

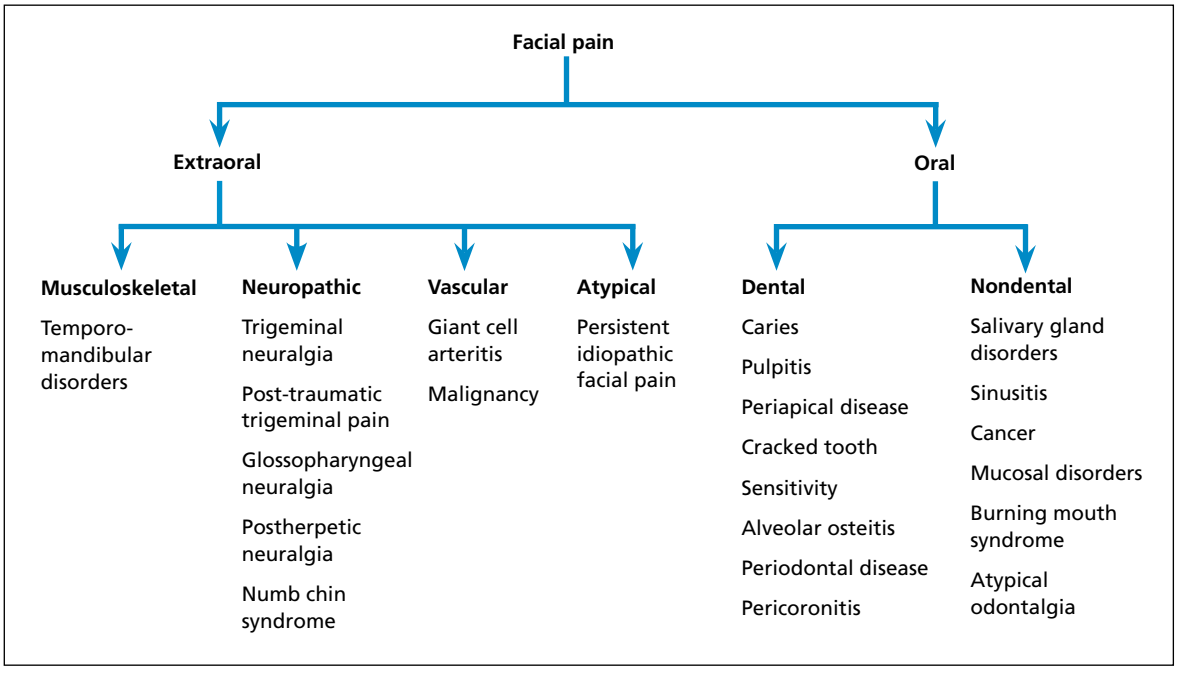
Musculoskeletal pain

Temporomandibular disorders (TMD) are a broad group of problems that affect the temporomandibular joint (TMJ), muscles of mastication, and/or associated bony and soft tissue structures.[6] They may occur secondary to malocclusion, traumatic injuries, oral parafunctional habits (eg, bruxism), hormonal influences, or psychogenic factors.[6] TMD is more prevalent in women, with a peak occurrence between ages 20 and 40 years.[6,8]

TMD can be articular (intracapsular) or nonarticular (extracapsular). Nonarticular disorders (>50% of TMD) usually affect the muscles of mastication and include chronic conditions such as fibromyalgia, muscle strain, and myopathies.[8]

FIGURE

Causes of facial pain



Muscle-related pain and dysfunction are believed to arise from parafunctional habits such as bruxism or clenching. Articular disorders include synovitis/capsulitis, joint effusion, trauma/fracture, internal derangement (disturbance in the normal anatomic relationship between the disc and condyle), arthritis, and neoplasm.[16]

What you'll see. Orofacial pain (usually dull and located in the preauricular region), joint noise, and restricted jaw function are key signs and symptoms of TMD. Exacerbation of pain with mandibular functions (eg, chewing, yawning, or swallowing) is a pathognomonic sign. Joint sounds such as clicking or crepitus are common. In most cases, crepitus correlates with osteoarthritis.[6] Nonspecific TMD symptoms include headache, earache, insomnia, tinnitus, and neck and shoulder pain.[6]

The gold standard of diagnosis of TMD consists of taking a detailed history, evaluating the patient's head and neck, and conducting a general physical examination and behavioral/psychological assessment.[17] Imaging of the TMJ and associated structures is essential.[17]

Treatment. Nonsteroidal anti-inflammatory drugs, opioids, muscle relaxants, antidepressants, anticonvulsants, anxiolytics, and corticosteroids are options for treating TMD.[6,8] Isometric jaw exercises, maxillomandibular appliances, and physical therapy are valuable adjuncts for pain relief. Advise patients to establish a self-care routine to reduce TMJ pain that might include changing their head posture or sleeping position, and limiting activities that require using their jaw, such as clenching, bruxism, and excessive gum chewing. Some patients may need to adopt a non-chewing diet that consists of liquid or pureed food. Massage and moist heat can help relax muscles of mastication and improve range of motion.

Approximately 5% of patients with TMD undergo surgery, typically simple arthrocentesis, arthroscopy, arthrotomy, or modified condylotomy.[6] Total joint replacement is indicated only for patients with severely damaged joints with end-stage disease when all other conservative treatments have failed. Joint replacement primarily restores form and function; pain relief is a secondary benefit.[8]

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TABLE

Extraoral facial pain: Differential diagnosis[2-15]

Site	Timing; severity	Aggravating factors	History and physical exam	Management
Temporomandibular disorders				
TMJ, muscles of mastication, ear. May radiate to the neck	Abrupt but often constant; moderate to severe	Chewing that is prolonged, opening mouth	Clicking or locking of TMJ, headaches, bruxism. Attrition of teeth, tenderness along the TMJ, clicking, reduced opening of mouth	NSAIDs, muscle relaxants, surgery
Trigeminal neuralgia				
Along the distribution of the second and third division of the trigeminal nerve	Sudden onset, lasts seconds to minutes, up to 30 attacks daily; moderate to severe	Eating, light touch, cold, some attacks could be spontaneous	Depression, fear of pain returning. Attack can be triggered with light touch; sensory changes very rare	Anticonvulsants
Post-traumatic trigeminal pain				
Trigeminal area	3-6 months after traumatic event; moderate to severe	Touch, cold or heat	History of trauma or dental procedure. Sensory changes, including allodynia	TCAs, pregabalin, gabapentin
Glossopharyngeal neuralgia				
Deep in the ear, throat, and posterior tongue	Sudden attacks, lasts seconds to minutes, often multiple attacks daily; moderate to severe	Chewing, talking, drinking, swallowing	Syncope (rare). Provoked by light touch	Anticonvulsants, TCAs, neuropathic medications, local anesthetics, surgery
Postherpetic neuralgia				
Site of zoster rash	Constant or intermittent; moderate to severe	Light touch	History of zoster. Skin changes. Hyperesthesia, hypoesthesia, allodynia	Anticonvulsants, TCAs
Numb chin syndrome				
Over the chin in the region supplied by the mental nerve	Abrupt or gradual	Odontogenic causes (eg, dental abscess, dental trauma, osteomyelitis) Systemic causes (sarcoidosis, HIV, malignancy)	Hypoesthesia, paresthesia, thermalgesic anesthesia or pain over the chin. If related to dental causes: percussion-induced pain, loosening of teeth. If malignancy is present, constitutional symptoms may be seen	Varies based on etiology
Persistent idiopathic facial pain				
Not well localized	Constant; moderate to severe	Stress, fatigue	Other chronic pain, significant life events. Exam is usually normal	TCAs, CBT
Giant cell arteritis				
Temporal region	Sudden onset, continuous; moderate to severe	Mastication	Vision changes, often associated with polymyalgia rheumatica. Temporal area tenderness	Prednisone

CBT, cognitive behavioral therapy; HIV, human immunodeficiency virus; NSAIDs, nonsteroidal anti-inflammatory drugs; TCAs, tricyclic antidepressants; TMJ, temporomandibular joint.

Neuropathic pain

Trigeminal neuralgia (TN) is sudden, usually unilateral, severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve.[9] It most commonly presents in the lower

2 branches of the trigeminal nerve and usually is caused by compression of the trigeminal nerve root by vascular or nonvascular causes.[4] The pain is severe and can profoundly impact a patient's quality of life.

TN attacks typically last from a few seconds to up to 2 minutes. As many as 30 attacks can occur daily, with refractory periods between attacks. After the initial attack, individuals are left with a residual dull or burning pain. TN can be triggered by face washing, teeth brushing, speaking, eating, shaving, or cold wind.[4]

Diagnosis can be tricky because more than half of patients with TN experience less severe pain after the main sharp attack; this presentation is called TN type II.[7] A detailed patient history and careful evaluation can help identify patients with TN type II. TN can be misdiagnosed as TMD, especially if it presents unilaterally.[15]

Treatment. Anticonvulsants are the primary medications used to treat TN.

Post-traumatic trigeminal pain is usually the result of an injury or dental procedure, such as facial trauma, tooth extraction, root canal, or dental implants.[12,18,19] Nerve injury is assumed to be the cause. This type of pain can start within 3 to 6 months of a trauma. It is located in the trigeminal area and patients describe it as burning, tingling and, at times, sharp.[15] Patients who have sustained injury to the lingual or inferior alveolar nerves have reported feeling "pins and needles."[12]

Common triggers include temperature changes or simple touch. Not all injuries result in pain; some patients may have only sensory impairment[15] or sensory deficits such as allodynia or hypoesthesia.

Treatment. The first line of treatment for post-traumatic trigeminal pain is tricyclic antidepressants (TCAs) followed by pregabalin or gabapentin.[14]

Glossopharyngeal neuralgia (GN) is similar in presentation to TN but is much rarer.[15] GN pain occurs deep in the throat, ear, or posterior tongue.[15] When the pain occurs in the inner ear, GN can be misdiagnosed as TMD. In most cases, no cause of GN can be determined.

Patients describe GN pain as shooting, sharp, and electrical shock-like, lasting from seconds to minutes, with recurrent attacks throughout the day. Like TN, GN can present as episodes of attacks that last weeks to months. Triggers include chewing, drinking, swallowing, and talking, as well as light touch.[13,15] Some patients with GN experience syncope due to the anatomical proximity of the vagus nerve.[14]

Treatment. Anticonvulsants are the first-line treatment for GN. Local anesthetics or surgery can be considered for patients who don't improve after medical therapy.[15]

Postherpetic neuralgia (PHN) can cause facial pain when the characteristic vesicular rash of the varicella zoster virus (shingles) occurs on the face. PHN usually affects the first division of trigeminal nerve, but the second and third divisions can be affected as well.[13]

What you'll see. The acute phase of PHN begins a few days before the initial rash has resolved and can last up to a month after. A new pain may begin one to 6 months after the initial rash has healed.[20] This pain, which patients often describe as sharp, stabbing, or burning, can be constant or intermittent. Dysesthesia, hypoesthesia, and allodynia may also occur within the affected dermatome.

PHN is usually diagnosed based on the patient's history and clinical presentation. However, direct fluorescent antibody stain, viral culture, or polymerase chain reaction performed on vesicular fluid from a herpetic lesion during the initial rash are the laboratory tests of choice if confirmation is needed.

Treatment. PHN is managed with anticonvulsants and TCAs.

Numb chin syndrome (NCS) is characterized by hypoesthesia, paresthesia, thermalgesic anesthesia, or pain over the chin in the region supplied by the mental nerve, a terminal branch of the mandibular division of the trigeminal nerve.[5,21,22]

NCS can be caused by odontogenic conditions, such as dental abscess, dental anesthesia, dental trauma, or osteomyelitis; systemic conditions such as amyloidosis, sickle cell disease, sarcoidosis, multiple sclerosis, human immunodeficiency virus, or diabetes; or malignancies such as lymphoma, leukemia, breast cancer, lung can-

Exacerbation of orofacial pain with mandibular functions such as chewing, yawning, or swallowing is a pathognomonic sign of temporomandibular disorder.

cer, prostate cancer, or head and neck cancers.[21] In one study of patients with NCS, cancer was the cause of the condition in 89% of patients.[22]

What you'll see. NCS is characterized by numbness of the skin in the lower lip, chin and mucous membrane inside the lip that extends to the midline.[5] Depending upon the etiology, patients may present with percussion-induced pain, loosening of teeth, sequestra, and mobility of fractured segments. Patients with metastatic malignancy may develop constitutional symptoms.

Making the diagnosis. Panoramic radiography is a useful starting point. If possible, a computerized tomography scan of the head and neck should also be done. Nuclear bone scintigraphy (bone scanning) may help identify bone disease such as osteomyelitis. A biopsy may be needed if a mass lesion is present.

Treatment. In NCS that is the result of a dental etiology, the prognosis usually is good. For example, NCS that is the result of an abscess usually resolves after the abscess is drained. However, if NCS is caused by metastasis, the prognosis is grim; the average length of survival after diagnosis is approximately 5 months if NCS is caused by mandibular metastasis and 12 months if leptomeningeal metastasis is present. Treatment does little to affect the outcome in these cases.[21,22]

Atypical pain

Persistent idiopathic facial pain (PIFP), previously known as atypical facial pain, is a persistent facial pain that does not have the classical characteristics of cranial neuralgias and for which there is no obvious cause.[2,10,23] PIFP is not triggered by any of the factors that typically precipitate neuralgias.[2] The onset may be spontaneous or associated with dental intervention or facial injury, but it usually does not have a demonstrable local cause.[24,25]

Neuropathic mechanisms that might be at work in PIFP include 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, or hyperactivity or hypoactivity of descending controls.[2]

PIFP is most frequently reported in women in their 40s and 50s.[25] The history of a patient with PIFP often include mood disorders, chronic pain, or poor coping skills.[14] Patients complain of a steady, unilateral, poorly localized pain that is deep, constant, aching, pulling, or crushing. It is usually present all day, every day. The constancy of the pain is its distinguishing feature. In the beginning, this pain may be in a limited area on one side of the face, usually the nasolabial folds or the angle of the mandible. Later, it may affect both sides of the face and extend to the neck and upper limbs.[23,24] Most patients with PIFP report other symptoms, including headache, neck and backache, dermatitis, pruritus, irritable bowel, and dysfunctional uterine bleeding.[26]

Making the diagnosis. A targeted history and accurate clinical examination are essential.[2,10] Although there are no formal diagnostic criteria, a patient can be assumed to have PIFP if:[2,10]

- There is pain in the face for most of the day or all day, every day.
- Initially, the pain may be confined to a portion of the face, but it is poorly localized and deep.
- The pain is not associated with other physical signs or loss of sensation.
- Imaging does not reveal an obvious anatomic or structural cause.

Treatment. Treatment of PIFP can be difficult and unsatisfactory.[23] Counseling to educate patients about the chronic and nonmalignant nature of the illness is the mainstay of treatment, followed by pharmacotherapy.[23] TCAs have shown a moderate effect in several trials. Gabapentin, topiramate, carbamazepine, and pregabalin also have shown limited to modest benefit in some patients. Surgical therapies appear to be of little or no use.[23] Experimental treatments such as pulsed radiofrequency, low-energy level diode laser have shown success in small studies.[10,23]

Vascular pain

Giant cell arteritis (GCA) is a systemic, chronic vasculitis involving the large and medium-sized vessels, mainly the extracranial branches of the carotid artery.[6,11] It predominantly affects people older than age 50 and is

Diagnosis of trigeminal neuralgia can be tricky; more than half of patients experience less severe pain after the main sharp attack.

more common among women and those of Scandinavian ethnicity.[27]

The cause of GCA is unclear. Genetic predisposition linked to humoral and cell-mediated immunity is believed to play a role.[28] Familial aggregation and predominance of the HLA-DR4 allele has been reported in patients with GCA.[6]

What you'll see. The most common signs and symptoms of GCA are temporal headache (seen in two-thirds of patients), jaw claudication and tenderness, and swelling of the temporal artery.[6,11] The headache of GCA usually is unilateral, severe, boring or lancinating, and localized to the temporal or occipital regions of the scalp.[6] Other orofacial manifestations include trismus, throat pain that develops while chewing, changes in tongue sensation and tongue claudication, tooth pain, dysphagia, dysarthria, submandibular mass, lip and chin numbness, macroglossia, glossitis, lip and tongue necrosis, and facial swelling.[11]

Visual symptoms include diplopia, ptosis, and possibly blindness if treatment is not instituted at first suspicion. Ocular symptoms result from anterior ischemic optic neuropathy, posterior ischemic optic neuropathy, or central retinal or cilioretinal artery occlusion.[6,28] Patients have also reported low-grade fever, asthenia, anorexia, weight loss, and generalized aches.[11,28]

Making the diagnosis. Arterial biopsy is the gold standard for diagnosis of GCA. It is usually performed on the temporal artery and is positive in 80% to 95% of people with the condition.[28] Other useful lab tests include erythrocyte sedimentation rate (ESR; elevated), white blood cell count (mildly elevated), and C-reactive protein (elevated).

Treatment. Prednisone is used to treat GCA, in initial doses ranging from 30 to 80 mg. A maintenance dose may be required for up to 2 years, with close follow-up and periodic ESR measurements.[28]

Malignancy is a rare cause of facial pain. The pain may be due to metastasis of extracranial bony or soft tissue as it compresses cervical and cranial nerves.[3] Lung cancer can cause referred pain in the periauricular region by compressing the vagus nerve, and this pain can be misdiagnosed as dental pain, atypical facial pain, TMD, or TN.[3,29] The facial pain of lung cancer is unilateral and on the same side as the lung neoplasm, and commonly is referred to the jaw, ear, or temporal region. While many patients have continuous pain, some report intermittent pain or pain that lasts for hours.[3] Facial pain caused by a malignancy is differentiated from other sources of facial pain by the presence of associated symptoms such as weight loss, cough, and hemoptysis.

Treatment. Treatment can include radiation or chemotherapy.[29]

A distinguishing feature of persistent idiopathic facial pain is that the pain is present all day, every day.

The mouth is often the source of lower facial pain

Pain in the oral cavity is the most common cause of pain in the lower face.[15] Intraoral pain usually is caused by disease in the following structures:

1. Dentition (eg, caries, dentin sensitivity, pulpal disease)
2. Periodontium (eg, gingivitis, acute or chronic periodontal disease, sensitivity related to gum recession, alveolar bone pathology)
3. Other soft and hard tissues, such as the palate, floor of mouth, buccal mucosa, non-tooth supporting bone, and tongue (eg, mucosal diseases, neoplasms, pain related to parafunction or trauma).

Rarely, intraoral pain may be referred. For example, myofascial pain might cause diffuse tooth pain.[30]

See **TABLE W1**[31-35] at the end of this article at jfponline.com for a summary of the etiology, signs/symptoms, diagnosis, and management of these and other dental causes of oral facial pain.

Nondental causes of oral facial pain can be associated with oral mucosal disorders, malignant disease and its therapy, salivary gland disorders, maxillary sinusitis, burning mouth syndrome, or atypical odontalgia. See **TABLE W2**[36-41] at jfponline.com for a more detailed description of these conditions. **JFP**

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The most common signs and symptoms of giant cell arteritis are temporal headache, jaw claudication and tenderness, and swelling of the temporal artery.

TABLE W1

Oral pain of dental origin[31-35]

Disorder	Site	Etiology	Signs/symptoms	Making the diagnosis	Management
Caries	Enamel, dentin, and cementum of the teeth	Bacterial invasion of tooth structure	Thermal sensitivity and/or pain when exposed to sweet or acidic foods	Risk assessment, clinical examination and interpretation of radiographs	Incipient caries: monitoring, remineralization, and control of risk factors. Deep caries: excavation and restoration
Pulpitis	Pulp of the teeth	Caries extend into pulp. Trauma, exposed dentin or cementum, and iatrogenic insults	Reversible pulpitis: discomfort that goes away within seconds of removal of stimulus Irreversible: sharp, spontaneous, lingering pain	Thorough history. Clinical exam and pulp test Interpretation of radiographs	Reversible: excavation and restoration Irreversible: root canal treatment
Periapical diseases	Apical periodontal tissues of the teeth	Infection from diseased pulp spreads into apical tissues	Acute periapical disease: sharp, throbbing, spontaneous pain, pain on biting, and/or swelling Chronic periapical disease: mild discomfort, sinus tract	Clinical exam and tests such as percussion, palpation Interpretation of radiographs	Root canal treatment or extraction with or without antibiotics. Abscess with fluctuant swelling: incision and drainage
Cracked tooth	Dentin and/or pulp of the teeth	Trauma, extensive restorations Incomplete fracture of dentin may or may not extend into the pulp	Sharp momentary pain on exposure to cold. Pain associated with release of biting pressure	Clinical history/exam. Periodontal probing, bite test. Trans-illumination with magnification Cone beam CT	Stabilization with a band, overlay, or crown Root canal treatment or extraction
Generalized sensitivity	Exposed dentin and/or cementum of the teeth	Gingival recession and/or abrasion of enamel. Most commonly due to incorrect brushing of teeth. Also due to recent scaling or tooth wear	Extreme sensitivity to cold fluids and air. Sharp localized pain that disappears immediately after removal of stimulus	Thorough history of symptoms and clinical examination shows exposed cementum and/or dentin	Improved oral hygiene practices, reduction of dietary acid, desensitizing agents and restorations
Alveolar osteitis	Extraction socket, mainly after mandibular tooth extraction	Lysis of a fully formed clot before it is replaced with granulation tissue	Moderate to severe deep, continuous, aching, radiating pain	Thorough patient history. Clinical exam shows the tissue around the socket is tender and white necrotic bone is exposed	Irrigation using saline or chlorhexidine. Placement of an obtundent dressing. Analgesics
Periodontal disease	Supporting tissues of the teeth: gingiva, periodontal ligament, and alveolar bone	Bacteria-induced inflammation of soft tissues and alveolar bone surrounding the teeth. Traumatic injury to the soft tissue	May be mild, persistent, or episodic dull pain	Clinical history of pain and associated symptoms. Clinical exam reveals gingival/periodontal pockets and/or abscess associated with vital teeth. Interpretation of radiographs	Scaling, root planing, and curettage. Periodontal abscess: drainage by incision or through pocket orifice
Pericoronitis	Gingival and mucosal tissues surrounding the crown of an erupting tooth	Inflammation and infection of the tissues around an erupting tooth, especially molars	Pain may be continuous or intermittent, ranging from mild to severe. Pain may radiate to ear, throat, and floor of mouth. Difficulty in opening/closing the jaw	History of trismus and discomfort during mouth opening/closing. Clinical exam shows erythematous and edematous tissue along with indentation of the opposing tooth	Removal of overlying operculum, irrigation with 2% chlorhexidine, debridement. Analgesics

CT, computed tomography.

TABLE W2

Oral pain of nondental origin[36-41]

Condition	Site	Etiology	Signs/symptoms	Making the diagnosis	Management
Oral mucosal disorders (oral candidiasis, herpes virus, recurrent aphthous stomatitis, lichen planus, pemphigus)	Oral mucosal epithelium	Infection, reactive process, systemic disorders or dysplasia	Vesicles, erosions, ulcerations, erythema, pseudomembrane formation, hyperkeratosis and hyperalgesia. Herpes zoster of the face may be associated with toothache	Risk assessment, clinical exam, and testing	Treatment of underlying mucosal condition
Pain associated with malignant disease and its therapy	Multiple sites	Oral mucositis secondary to chemotherapy and radiation therapy Longstanding ulcerations, secondary infection, infiltration into adjacent peripheral nerves	Injuries to peripheral nerves: pulpitis-like pain Oral mucositis: erythema, ulceration, pseudomembrane formation and shedding Angioleiomyoma and methemoglobinemia cause toothache	Thorough clinical exam of hard and soft oral tissues and appropriate judgment	Early diagnosis, referral, and management of underlying malignant disease
Salivary gland disorders	Typically localized to the gland itself	Bacteria, localized and systemic viruses, autoimmune diseases, secondary to sialoliths and strictures and congenital disorders	Swollen painful salivary glands, induration and erythema of overlying skin. Increase in pain with chewing and dry mouth	Pain can be localized by palpation. Radiograph of the gland and ducts Screening tests for dry mouth	Acute phase: supportive treatment with analgesics, hydration, antibiotics, and parasympathomimetics Sialoliths: surgical removal of salivary stones
Maxillary sinusitis	Posterior maxilla and maxillary teeth	Intranasal and sinus abnormalities, inflammation or anatomic variations Infection of maxillary sinus is perceived as dental pain in maxillary posterior teeth and vice versa	Facial pain and fullness in maxillary posterior region. Continuous dull or diffuse lingering pain. Teeth are sensitive to percussion, mastication, and/or temperature. Cough, headache, ear pain, nasal congestion, and discharge	Intraoral exam of the teeth and extraoral exam of the sinus. Fiber optic illumination of the sinus may reveal changes in the affected region	Symptomatic: decongestants, antihistamines, mucolytic agents, alpha-adrenergic agents, corticosteroids and analgesics. If sinusitis persists >7 days and features purulent discharge: antibiotics
Burning mouth syndrome	Multiple sites; mostly affects anterior two-thirds of the tongue, followed by dorsum and lateral borders of the tongue, lips, palate, and cheeks	Primary/essential BMS: no local or systemic causes, involves peripheral or central neuropathological pathways Secondary BMS: local, systemic, and psychological factors	Burning pain, dysgeusia, dysesthesia, loss of taste, and paresthesia	Thorough, comprehensive history of presenting symptoms. Identify underlying factors, such as mucosal disease, stress, nutritional deficiencies, diabetes, and medication adverse effects	Management of underlying condition Systemic medications (eg, benzodiazepines, anticonvulsants, TCAs, SSRIs), topical medications (eg, capsaicin, clonazepam), CBT
Atypical odontalgia	Teeth or in a tooth socket after extraction	Not clearly defined. Possibly neuropathic, vascular, or psychogenic mechanisms. Pain usually, but not necessarily, follows a dental procedure or trauma	Pain can be aching, burning, throbbing, or shooting. Moderate to severe in intensity	Diagnosis of exclusion	Pain management Topical analgesics: lidocaine/prilocaine or capsaicin cream TCAs, SSRIs, anticonvulsants, tramadol, oxycodone. Adjunct therapies: acupuncture, self-hypnosis, CBT

BMS, burning mouth syndrome; CBT, cognitive behavioral therapy; SSRIs, selective serotonin reuptake inhibitors; TCAs, tricyclic antidepressants.