Diagnosis and treatment of orofacial pain

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Abstract
Orofacial pain is a complex health care problem that may compromise the quality of life of the patient and frustrate the clinician. A variety of disorders may account for the development of pain in the orofacial structures. Temporomandibular disorders are among the most common causes of orofacial pain. Frequently orofacial pain is due to neuropathic, vascular or neurovascular mechanisms. In addition, numerous local pathologies or systemic diseases can result in the development of orofacial pain, while pain in the face may also be referred from a distant structure. Idiopathic and psychogenic types of orofacial pain are also recognized. Establishing a proper diagnosis is the most challenging part of managing orofacial pain and is an essential prerequisite to an effective treatment. A detailed medical history, a thorough evaluation of the patient’s complaint and an extensive clinical examination will provide valuable diagnostic information contributing to an accurate diagnosis. This review presents the main diagnostic characteristics and the therapeutic approaches of the most common types of orofacial pain.

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Introduction

Pain in the orofacial region is a common complaint affecting the lives of millions of people around the world. Orofacial pain may be due to a number of different causes (Table 1). Moreover, the site of painful sensation does not always coincide with the source of pain as referred pain is very common in the orofacial structures. Therefore, complexities and challenges in diagnosis are frequently encountered. Accurate diagnosis is the key to successful treatment and can be achieved only through a thorough history and examination. The evaluation of the orofacial pain patient should begin with a medical history, including history of trauma to the head and neck, significant illnesses, current medications, and complete review of systems, with special attention to systemic disorders which can cause facial pain. A detailed description of the pain complaint in terms of pain duration, location, intensity, quality, frequency and progression of pain since onset provides valuable diagnostic information. Associated symptoms accompanying the facial pain, as well as aggravating and alleviating factors, are also important diagnostically.

A brief psychological screening should be part of the history for all chronic pain patients. Depression and anxiety have a high prevalence among chronic pain patients. Psychological distress may be a factor contributing to the development or maintenance of pain, a consequence of pain, or a concurrent problem with independent sources. The clinical examination of the orofacial pain patient includes assessment of cranial nerves’ function, cervical spine evaluation (posture and range of motion), palpation of masticatory and neck muscles, temporomandibular joint examination (tenderness to palpation, range of motion, joint sounds), and complete intra-oral and dental evaluation. Depending on the findings of the history and clinical evaluation, appropriate laboratory tests and diagnostic imaging procedures may be required. The present article provides a synopsis of the diagnosis and treatment of orofacial pain.

A. Musculoskeletal pain

Temporomandibular disorders

Temporomandibular disorders (TMD) is a collective term that encompasses a number of pathologic conditions, involving the temporomandibular joints (TMJ) and the masticatory muscles, and manifesting with pain and/or dysfunction of the orofacial apparatus. The most common signs and symptoms of TMD include facial pain that is aggravated by jaw function, tenderness upon joint and muscle palpation, limited mandibular range of motion, deviation or deflection of the mandible on mouth opening, and TMJ sounds. TMD patients may also complain of tinnitus, earaches, headaches, and dizziness. Pain in the masticatory muscles, the TMJ and the associated structures is the most frequent presenting symptom, as well as the main symptom that motivates patients to seek treatment. Temporomandibular disorders constitute the most common cause of chronic pain in the orofacial region. Approximately 12% of the general population is affected by TMD, and 5% of the population has symptoms severe enough to warrant treatment. Temporomandibular disorders are more prevalent among women of childbearing-years. Women-to-men ratio is estimated to be 8:1 among individuals seeking treatment. Putative etiological factors include trauma involving local tissues, repetitive chronic microtrauma (e.g. clenching or bruxism), unaccustomed jaw use (e.g. opening the mouth too wide), and increased level of emotional stress. Temporomandibular disorders are classified into three main categories: a) masticatory muscle disorders, b) articular disc derangements, and c) temporomandibular joint disorders. The most common types of TMD are the following:

a) Masticatory muscle disorders

Myofascial pain

Myofascial pain may be due to emotional stress, sleep disturbances, muscle overuse, nutritional deficiencies, or fatigue. It is characterized by dull, aching pain and presence of trigger points in the affected muscle. Trigger points are localized firm hypersensitive areas that upon palpation produce a characteristic pattern of referred pain. Myofascial pain is constant and is exacerbated by muscle use. The patient may also complain of tinnitus, vertigo, toothache and tension type headache. Provocation of the trigger points replicates the symptoms of the patient confirming the diagnosis.

Treatment aims at elimination of precipitating factors, and inactivation of trigger points by vapo-coolant spray or injection of local anesthetic, followed by stretch. Relaxation therapy, daily stretching of the affected muscles, and medications such as analgesics, muscle relaxants, and antidepressants in low doses, can also be helpful.

Localized muscle soreness

Localised muscle soreness is a non-inflammatory muscular disorder presenting with little or no muscle pain at rest, but intensified pain during mandibular movement. Masticatory muscles are sensitive to palpation and mouth opening is restricted secondary to pain. Local tissue injury or microtrauma due to abusive or unaccustomed muscular activity constitute common causes of localized muscle soreness. Patient education on painless use of the mandible, soft diet, moist heat applications, NSAIDs or muscle relaxants, stabilization appliance and relaxation therapy can be part of the treatment.

b) Articular disc derangements
Disc displacement with reduction
Disc displacement with reduction is usually characterized by displacement of the articular disc anteriorly and medially, with improvement of its position during opening. Reproducible joint clicking occurs during opening and closing mandibular movements and the mandible deviates upon opening. The patient may complain of episodic and momentary catching of the jaw movement during mouth opening. Pain may or may not be present. Alterations in the disc-condyle structural relation may result from elongation of the discal ligaments, secondary to trauma or repetitive chronic microtrauma. Asymptomatic clicking is a common condition and does not require treatment.

Disc displacement without reduction
Disc displacement without reduction refers to an altered disc-condyle structural relation that is not improved during mouth opening. Frequently, there is a history of clicking and sudden onset of hypomobility. The patient presents with a limited (less than 30 mm) mouth opening and a restricted lateral excursion to the contralateral side. The mandible deflects to the affected side on opening and clicking noises are absent. Pain is typically present in the acute condition, while chronic disc dislocation is often non painful. With the progression of the condition, there is a gradual increase in the mandibular range of motion. The history and examination will point to the diagnosis; however, soft tissue imaging is essential for a definitive diagnosis. In acute disc dislocation there should be an effort to reduce the disc dislocation by manual manipulation, followed by insertion of an anterior repositioning appliance. Management of chronic disc dislocation may include a stabilization appliance, physical therapy and NSAIDs if pain is present. Patients who fail conservative treatment and complain of significant pain and dysfunction are candidates for arthrocentesis or arthroscopy.

c) Temporomandibular joint disorders
Synovitis and Capsulitis
Synovitis and capsulitis are characterized by inflammation of the synovial lining of the TMJ and the capsular ligament respectively. They are grouped together since they cannot be distinguished on the basis of historical or clinical findings. Synovitis and capsulitis are characterized by constant deep pain in the TMJ, which is aggravated by jaw function, and restricted mouth opening secondary to pain. Acute malocclusion of posterior teeth on the affected side may be present. Synovitis and capsulitis can be induced by trauma to the jaw or repetitive chronic microtrauma. In the case of acute trauma, ice should be applied to the affected joint 4-6 times daily for the first 24-36 hours. Then, moist heat applications can be applied for 10-15 minutes 3-4 times per day. NSAIDs should be taken on a regular basis for 10-14 days to reduce pain and inflammation. The patient should be instructed to restrict jaw movement to a pain free range of motion. A stabilization appliance can be of benefit, especially if parafunctional habits are present.

Osteoarthritis, osteoarthrosis
Osteoarthritis is a non-inflammatory arthritic condition characterized by deterioration of the articular surfaces. It presents with pain that is exacerbated by mandibular movement, tenderness upon palpation of the joint, crepitus and limited range of mandibular motion. Radiographic evidence of structural bony change is present. Conservative treatment including NSAIDs, moist heat applications, painless use of mandible, passive jaw exercises within painless limits and a stabilization appliance, is effective for most patients. For refractory cases, one or two single injections of corticosteroids in the joint, or surgery may be recommended.

Tension type headache
Episodic tension type headache (TTH) is characterized by bilateral pain that may involve the occipital, parietal, temporal or frontal areas. Since this type of headache has a high prevalence, it is suggested that individuals who experience less than fourteen pain episodes per year are regarded as headache-free. The pain has a dull, tightening or pressing quality, lasts for few hours to 3 days, and may be associated with tenderness in pericranial muscles. The pain intensity ranges from mild to moderate, and increases with greater frequency of pain episodes. The headache may be precipitated by stress and is usually associated with anorexia, fatigue and poor sleep. Treatment consists of elimination of contributing factors, stress management, and pharmacotherapy. Episodic tension type headache responds to most simple analgesics.

Chronic TTH headache has similar characteristics with episodic TTH, but occurs at a higher frequency and may be of greater severity. The pain occurs daily or almost daily, frequently constitutes the result of analgesic overuse, and is refractory to numerous treatments. Treatment of analgesic overuse, stress management and prophylactic pharmacotherapy with tricyclic antidepressants can be helpful.

B. Neuropathic pain
1. Episodic neuropathic pain
Trigeminal neuralgia
Trigeminal neuralgia (TN) has an incidence of 4 to 5 per 100,000 population. The disorder has higher prevalence among females (F:M sex ratio of 1.74:1), and affects primarily older individuals; the average age of onset is between the fifth and seventh decade. The majority of cases are unilateral; only approximately 4% of cases are bilateral. TN most commonly involves the maxillary or mandibular division of
Trigeminal neuralgia is characterized by episodic, severe, stabbing pain in the distribution of one or more of the trigeminal nerve divisions. Pain attacks last only seconds to 2 minutes and may recur repeatedly in clusters. The pain is characterized by sudden onset and cessation and the patient is completely asymptomatic between attacks. Pain paroxysms may be provoked by innocuous sensory stimulation of trigger zones in the receptive field of the affected branch. Common daily activities such as talking, eating, drinking, swallowing, shaving, brushing the teeth, or washing the face, can trigger the pain, compromising significantly the patient’s quality of life. The trigger zone is always ipsilateral to the pain; however it may not coincide with the area of pain. Common extraoral trigger zones occur above the supraorbital foramen, the inner canthus of the eye, lateral to ala, and over the mental foramen. Typically, immediately following a jab of pain, there is a refractory period during which further pain attacks cannot be evoked. Spontaneous remissions lasting months or years occur in some patients; however, TN is usually progressive and the pain attacks become more frequent and severe. The sharp, paroxysmal pain of TN is often localized in the dentist or the surrounding structures, and is misdiagnosed as dental pain. Frequently, TN patients undergo numerous dental procedures until the diagnosis of TN is made. These procedures may offer a temporary pain relief for a few weeks; however, the pain always recurs, often even worse than before. Failure of dental treatment to provide long-term pain relief should raise the suspicion of TN. An important feature that distinguishes TN from dental pain is that TN typically does not interrupt the patient’s sleep. Moreover, pain originating from dental pathology is usually progressive and its character changes with time.

Demyelination of trigeminal sensory fibers due to vascular compression of the trigeminal root-entry zone is implicated in the etiopathogenesis of TN. Demyelination may result in ectopic generation of nerve impulses presenting clinically as spontaneous pain, while ephaptic neural transmission may underlie the generation of pain by innocuous stimulation. Two to five percent of TN cases are caused by posterior fossa compressive lesions, or multiple sclerosis. Typically, patients with TN and multiple sclerosis are younger and more likely to have bilateral facial pain. Neurological examination and magnetic resonance imaging of the brain should be undertaken in all TN patients to rule out central nervous system lesions.

Carbamazepine is the drug of choice, while baclofen, oxcarbazepine, lamotrigin, phenytoin, and gabapentin are also effective. For patients who cannot tolerate adverse effects, or become refractory to pharmacological treatment, surgical intervention is recommended. Microvascular decompression of vessels compressing the nerve root is very effective and has low incidence of recurrence. However, it involves serious risks including hearing impairment, ataxia, brain stem infarction, cerebellar injury, and death. Percutaneous ablative techniques involve lesioning at the level of the gasserian ganglion by percutaneous radio-frequency thermocoagulation, injection of glycerol, or balloon compression. These procedures have good initial results, and carry less risk than microvascular decompression; however, they are associated with a higher incidence of pain recurrence. Potential complications include loss of touch sensation, dysesthesias, and anesthesia dolorosa.

**Glossopharyngeal neuralgia**

Glossopharyngeal neuralgia is similar to trigeminal neuralgia but it involves the distribution of the glossopharyngeal nerve. It is characterized by severe, sudden, unilateral, stabbing pain in the ear, base of the tongue, tonsilar fossa, or beneath the angle of the mandible. Pain typically lasts few seconds to 2 minutes, and can be triggered by swallowing, chewing, talking, coughing or yawning. Frequently, patients experience remissions of pain lasting months to years. Pharmacological treatment is the same with that used in TN. In patients who fail to respond rhizotomy of cranial nerve IX may be recommended.

**Nervus intermedius neuralgia**

Nervus intermedius neuralgia presents with similar characteristics to trigeminal neuralgia, but the pain is felt deeply in the auditory canal. Frequently, there is a trigger zone in the posterior wall of the auditory canal. Pharmacological treatment is similar to that for TN. Surgical treatment consists of section of the nervus intermedius or the chorda tympani nerve.

**2. Continuous neuropathic pain**

**Herpetic and postherpetic neuralgia**

Herpes zoster affects mainly older people. Approximately ten percent of cases involve the trigeminal ganglion, with the ophthalmic division being most commonly affected. The condition is due to reactivation of the varicella-zoster virus that has been latent in the trigeminal ganglion following a systemic varicella infection. Herpes zoster is characterized by vesicular eruption in the distribution of the affected branch, which is preceded and accompanied by pain. Oral acyclovir and systemic corticosteroids are the mainstreams of treatment.

Postherpetic neuralgia refers to pain that persists longer than 3 months following the outbreak of herpes zoster eruption. It affects 10-20% percent of herpes zoster patients, mainly elderly and immune compromised individuals. The
pain is described as severe and burning with sharp exacerbations. Associated symptoms include allodynia, hyperalgesia, and occasional sensory deficits. Postherpetic neuralgia responds poorly to treatment. Amitriptyline is the drug of choice, while topical capsaicin may be helpful in some patients.

Traumatic neuralgia

Traumatic neuralgia occurs following direct neural injury and deafferentation. The pain is described as constant, and burning; superimposed lancinating exacerbations may occur. Abnormal sensations, such as allodynia and hyperalgesia, and neural sensory or motor deficits often accompany the pain. Treatment consists of desensitization of the affected area with capsaicin, and pharmacological management with tricyclic antidepressants.

Eagle's syndrome

Eagle’s syndrome is caused by compression of glossopharyngeal nerve by an elongated styloid process or a calcified stylohyoid ligament. The syndrome presents with persistent sore throat, dysphagia, earache, and pain in the postmandibular area. Pain is usually triggered by rotation of the head to the contralateral side, swallowing, chewing and yawning. Pain may have a neuralgic component, mimicking glossopharyngeal neuralgia. Radiographic examination will reveal elongation of the styloid process or calcification of the stylohyoid ligament. The treatment of Eagle’s syndrome is primarily surgical.

C. Vascular pain

Giant cell arteritis

Giant cell arteritis (GCA) is a multifocal vasculitis that is characterized by giant cell infiltration of the wall of the large and medium-sized cranial arteries, especially the superficial temporal artery. Other commonly affected arteries include the maxillary, the ophthalmic and the posterior ciliary arteries. Patients are usually 50-85 years of age with a mean age of 70 years. Women are affected twice as often as men. The symptoms of GCA are related to the involved arteries. Frequently, a new-onset, throbbing, severe temporal headache is the major complaint. Pain is intensified when the patient lies down, and is typically accompanied by a variety of constitutional symptoms such as malaise, fatigue, low-grade fever, anorexia and weight loss. Chewing may induce masticatory muscle pain secondary to inflammation of the maxillary artery. Involvement of the lingual artery can result in pain and blanching of the tongue and rarely in tongue necrosis. Compromise of the arteries supplying the eyes can lead to transient or persistent visual disturbances including blindness, the most feared complication of GCA. Upon clinical examination, the superficial temporal artery is typically exquisitely sensitive to pressure and appears erythematous, swollen and tortuous. Temporal artery palpations may be decreased or absent, and in some cases the affected artery may become thrombosed after which it is palpated as a firm pulseless cord. Marked elevation of the erythrocyte sedimentation rate is present in almost all patients. Temporal artery biopsy remains the standard approach to the diagnosis of GCA.

The etiology of GCA is obscure; however, autoimmunity to the elastic lamina of the artery has been proposed. Involvement is characterized by chronic inflammation of the intima and tunica media with narrowing of the lumen from edema and proliferation of the intima. Corticosteroids are the drugs of choice for GCA. Permanent loss of vision occurs to 25-50% of untreated patients. Therefore, it is imperative that high dose corticosteroid therapy begins immediately upon clinical suspicion of GCA to prevent visual loss. GCA tends to run a self-limited course of several months to as long as five years. Relapses occur in up to 25% of cases; these are more likely to occur in the first 18 months of therapy or within 12 months after the cessation of corticosteroid treatment.

D. Neurovascular Pain

Migraine

Migraine is a recurrent headache that is two to three times more common in women than men. The pain is severe and pulsating, and in the majority of cases unilateral, involving the frontal, temporal and retro-orbital areas. Pain attacks last few hours to 3 days and may be accompanied by photophobia, phonophobia, nausea, and vomiting. Pain may be precipitated by various factors, such as stress, alcohol, tyramine-containing foods, menstruation and bright lights, and is typically aggravated by routine physical activity. The two main types of migraine are migraine without aura, which is the most common, and migraine with aura. Migraine with aura is characterized by an aura, namely focal neurological symptoms that precede the headache. These
symptoms most often include visual disturbances, such as flashing lights, and zigzag images, and less often unilateral numbness, unilateral paresthesia, weakness, aphasia and vertigo. The aura develops in minutes, lasts less than 1 hour and usually disappears before the onset of pain.

Management of migraine should begin with an effort to modify triggering factors. Acetaminophen or NSAIDs when taken at the onset of an attack may abort the pain. If the patient fails to respond, triptans or ergotamine should be tried. Frequent use of symptomatic medications can result in development of daily headache. Prophylactic management of migraine, with β-adrenergic agents, calcium channel blockers, or tricyclic antidepressants is recommended when the frequency of pain attacks is higher than twice per week.

Cluster Headache
Cluster headache affects primarily men (F:M sex ratio 1:6) in the third decade of their life. It is characterized by attacks of excruciating, throbbing, strictly unilateral pain in the orbital, supraorbital and/or temporal region. The pain attacks last 15-180 min and occur from once every other day to 8 times per day, usually at the same time each 24 h period, often in the middle of the night awakening the patient. The pain is associated with autonomic signs, such as conjunctival injection, ipsilateral lacrimation, nasal congestion, rhinorrhea, forehead and facial sweating. Alcohol and nitroglycerin can trigger the pain. The pain attacks occur in discrete time periods lasting weeks or months (cluster periods) separated by remission periods lasting months or years. Approximately 10% of patients have chronic cluster headache, without any remission. The pharmacological treatment of cluster headache includes abortive medications, such as oxygen, ergotamine, and intranasal lidocaine, and prophylactic medications, such as prednisone, methysergide, lithium, verapamil, and nifedipine. In refractory cases, trigeminal sensory rhizotomy, superficial petrosal neurectomy, or decompression of the nerve intermedium may alleviate the symptoms.

Chronic Paroxysmal Hemicrania
Chronic paroxysmal hemicrania has similar features with cluster headache but the pain attacks are shorter-lasting and more frequent. Moreover, the condition is more prevalent among females (F:M sex ratio of 2.36:1). Chronic paroxysmal hemicrania is characterized by unilateral attacks of severe pain that recur 1 to 40 times per day, and last 2 to 120 min, with a mean of approximately 15 min. The pain attacks may initially occur in clusters, but in most cases chronic symptoms subsequently develop. The pain affects most commonly the ocular, temporal, maxillary and frontal regions, and has a throbbing, or stabbing quality. Ipsilateral lacrimation and rhinorrhea, conjunctival injection, and nasal congestion constitute the most common coexisting symptoms. Attacks occur around the clock and interrupt the patient’s sleep. Head flexion or rotation and alcohol can precipitate the paroxysms. Response to indomethacin prophylactic treatment is dramatic and constitutes part of the diagnostic criteria.

E. Idiopathic Facial Pain
Idiopathic facial pain is a diagnosis of exclusion; the pain is not associated with any apparent objective signs, and all diagnostic tests are negative. The lack of a demonstrable organic cause and the high prevalence of anxiety and depression among patients with idiopathic facial pain have led to the belief that the condition is of psychogenic origin. However, the psychological profile of these patients is similar to that of other chronic pain patients. Clearly, their psychological distress may constitute a consequence and not a cause of their pain. It is essential that a thorough diagnostic assessment is carried out before the diagnosis of idiopathic facial pain is considered, in order to rule out other possible causes of the pain.

Atypical facial pain
Atypical facial pain is characterized by continuous, daily pain of variable intensity. Typically, the pain is deep and poorly localized, is described as dull and aching, and does not waken the patient from sleep. At onset the pain may be confined to a limited area on one side of the face, while later it may spread to involve a larger area. The pain is refractory to a variety of treatments. Frequently, patients present with a history of multiple consultations, multiple ineffective treatments, and surgical explorations and treatments that may have perplexed the condition. Women are affected more often than men. Management of the patient aims at reduction of pain and health care use, increase in activity, and return to work. Education, physical therapy, psychological counseling, medications, and alternative pain management strategies, such as acupuncture, may be useful.

Atypical odontalgia
Atypical odontalgia, often referred to as phantom tooth pain, is characterized by chronic, constant dento-alveolar pain in the absence of obvious pathology. The pain is described as dull, aching, or burning toothache of moderate intensity. An important characteristic of atypical odontalgia that differentiates it from pulpal dental pain is that the toothache remains unchanged for months or years. In addition, local provocation of the tooth or surrounding tissues, and temperature changes do not affect the pain. Tooth vitality tests and radiographic examination will also serve to exclude dental pathology.

The majority of patients are women in the fourth and fifth decades. Maxillary molars or premolars are most commonly affected. Frequently, the pain begins following a deafferentation procedure, such as dental pulp extirpation or tooth extraction, suggesting a neuropathic mechanism. Usually, patients undergo many unsuccessful, invasive dental procedures before diagnosis is made. The response
to analgesics or dental interventions is poor. Tricyclic antidepressants at low doses may alleviate the symptoms.

**Burning mouth syndrome**

Burning mouth syndrome is characterized by continuous or nearly continuous, chronic burning pain in one or more oral mucosal sites, including the tongue, palate, inner surfaces of the lips, and buccal mucosa. Burning mouth syndrome is more prevalent among post-menopausal women. It may constitute a primary disorder or arise secondary to another condition, such as denture stomatitis, candidiasis, xerostomia, diabetes mellitus, nutritional deficiencies, or anemia. Primary burning mouth syndrome is idiopathic; the clinical exam and the results of laboratory testing and diagnostic imaging fail to detect any evidence of pathology. The burning pain can be unilateral or bilateral or can start on one side and spread to the opposite side. It is usually milder upon awakening and progressively increases in the course of the day. The pain does not disrupt the patient’s sleep. Eating, drinking or chewing gum often attenuate the symptoms. Precipitating factors include stress, fatigue, cold, hot or spicy foods. Patients may complain of dry mouth, taste changes, thirst perception, and draining fluid. Spontaneous partial remission within six to seven years following onset has been reported in a subset of patients. Treatment of the secondary BMS depends on the underlying disease. Primary burning mouth syndrome usually responds to low-dose tricyclic antidepressants and benzodiazepines.

**F. Other diseases that may cause facial pain**

**Local pathology**

Pain in the orofacial region may be secondary to local pathology affecting any of the following structures: eyes, ears, nose, sinuses, pharynx, teeth, periodontium, mucogingival tissues, salivary glands, cranial bones, and TMJ. Dental pain is among the most common types of orofacial pain and can be referred to various head and neck areas. On the other hand, muscular, neuropathic and neurovascular pain may feel extruded. The accompanying malaise, fever, nasal obstruction and purulent nasal discharge facilitate the diagnosis, while radiographic examination is needed to confirm it. Salivary pain, intensified immediately prior to and during eating, is often due to blockage of a salivary duct by calculus, which causes chronic sialadenitis. Acute suppurative parotitis, such as that seen in newborns and debilitated patients after surgery, causes parotid swelling and pain of sudden onset, which may be accompanied by trismus. Salivary gland tumors may also cause pain; especially those tumors occurring in the deep lobe of the parotid gland, may cause TMJ symptomatology, simulating a number of primary joint disorders. Orofacial pain that has worsened rapidly in short period of time may indicate malignancy, which may affect almost any soft or hard tissue component of the oral and maxillofacial region, including the tissues of the temporomandibular joint.

**Distant pathology (referred pain)**

Pain may be referred to the orofacial region from distant structures. Cranial nerves V, VII, IX, X, as well as the upper cervical nerves converge in the trigeminal spinal tract nucleus, providing an anatomical substrate for pain referral in the orofacial region. Cervicospinal dysfunction constitutes a common source of referred orofacial pain. Accordingly, evaluation of the cervical region should consist an integral part of the examination of the patient with orofacial pain. Occasionally, angina is felt in the mandible and lower molars. Typical features include history of ischemic cardiac disease, precipitation of the pain by exercise, and alleviation of the symptoms by rest and nitroglycerin. Rarely, orofacial pain is a manifestation of intracranial pathology. Intracranial tumors may produce facial pain that is exacerbated by straining, coughing, exertion or postural changes, and is often accompanied by nausea, vomiting or blurring of vision. Subarachnoid hemorrhage is characterized by excruciating pain of sudden onset that is described characteristically as “worst headache of my life”. Facial pain can on rare occasions be the presenting symptom of lung cancer. The pain is unilateral, severe, and most commonly localized in the ear, the jaws and the temporal region. History of smoking, aggravation and expansion of the pain, digital clubbing, increased erythrocyte sedimentation rate, and weight loss can lead to the diagnosis.

**Systemic diseases**

Occasionally, orofacial pain is a manifestation of a systemic disease. Rheumatologic conditions, such as rheumatoid arthritis, systemic lupus erythematosus, and fibromyalgia are often associated with musculoskeletal facial pain. Fibromyalgia (FM) is characterized by widespread pain, and pain on palpation of eleven out of eighteen specific tender points. There is a significant overlap between masticatory...
myofascial pain and FM; approximately 75% of FM patients meet the diagnostic criteria for myofascial pain, while 15-20% of myofascial pain patients have FM. Patients with hypothyroidism or Lyme disease may also experience masticatory muscle pain or weakness, while neurological disorders, such as multiple sclerosis, often lead to the development of neuropathic facial pain.

G. Psychogenic Pain
Various mental disorders have been associated with chronic orofacial pain, including somatoform disorders, factitious disorders and malingering. A diagnosis of psychogenic pain requires not only exclusion of other organic disorders, but also fulfillment of specific diagnostic criteria. As noted by Graff-Radford “the labeling of a disease process as psychogenic without clear documented objective criteria is grossly unfair to the patient”. Given the complexity of diagnosis and management of psychiatric disorders, referral to a psychiatrist is warranted.

Table 1 - classification of orofacial pain

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