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Non Dental Origin Facial Pain : A Detailed Review

[1] Manish Bhargava
[2] Deepak Chaudhary
[3] Pankaj Dhawan

[1] Reader,
[2] Senior Lecturer,
Department of Oral Pathology
[3] Professor & Head
Department of Prosthodontics
Manav Rachna Dental College
Faridabad, Haryana. India

Address For Correspondence:
Dr. Pankaj Dhawan
Prof. and HOD
Department of Prosthodontics
Manav Rachna Dental College
Faridabad, Haryana India
Email: pankaj_dhawan@hotmail.com

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Abstract
Dentistry has enjoyed a remarkable period of technological and scientific growth over the past several decades. With the increase in life expectancy, the number of individuals seeking dental care also has escalated. One of the most common reasons for seeking care is because of pain and/or dysfunction, usually involving the teeth or periodontal tissues. However, musculoskeletal, vascular, and neuropathic causes of orofacial pain occur frequently. The need to understand pain and all of its ramifications is of utmost importance in diagnosis and case-specific, evidence-based management of conditions afflicting the masticatory system.

Key Words
pain, facial pain, non dental origin pain

Introduction:
The most recent definition of pain, produced by the task force on taxonomy of the International Association for the Study of Pain (IASP) is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage" [1]. Orofacial pain encompasses a myriad of signs and symptoms within and outside the oral cavity. To establish a differential diagnosis for orofacial pain we must first consider the history, examination and relevant investigations. Although both may co-exist, the more rare non-dental pain must be distinguished from dental pain to avoid unnecessary dental treatment and to organize appropriate referral for the patient.

Principles Of Pain Diagnosis [2]
The examination and assessment of patients with chronic orofacial pain is challenging for all clinicians. In most disorders, no specific biologic marker validated diagnostic criteria or gold standard exists. A systematic approach for collecting diagnostic information is needed to minimize the risk of missing critical information.

Identifying the true source of pain
History, physical examination, and behavioral assessment usually serve as the basis for diagnosis. Frequent re evaluation including assessment of the effects of treatment is an important part of this.
A description of the pain complaint should take into account details concerning the following:

1. Location of pain : The patient's ability to locate the pain with accuracy has diagnostic value, but the examiner should guard against assuming that the site of pain necessarily identifies the true source of pain – the structure from which the pain actually emanates.

2. Intensity of pain : The intensity of pain should be established by distinguishing between mild and severe pain. This can be based on how the patient appears to react to the suffering – mild pain being what the patient describes but to which he displays no visible physical reactions and severe pain being what causes muscular and autonomic effects that are objectively evident to the examiner.

3. Mode of onset : The mode of onset of individual pains is important diagnostically. The onset may be wholly spontaneous. It may be induced by certain activities such as yawning, chewing, drinking hot or cold liquids, or bending over. It may be triggered by major superficial stimulation such as touch or movement of the skin, lips, face, tongue or throat.

4. Manner of flow of pain : The manner of flow yields important information by determining whether the individual pains are steady or paroxysmal.

5. Quality of pain : The quality of pain should be classified according to how it makes the patient feel. This classification is usually termed bright or dull. When the pain has a stimulating or exciting effect on the patient it is classified as bright when the pain has a depressing affect that causes the patient to withdraw to some extent, it is classified as dull. Further evaluation of the quality of pain that constitutes the patients complaint should be made to classify it as pricking, itching, burning, aching or pulsating. Bright, tingling pain is classified as a pricking sensation.

6. Temporal behavior : Reflects the frequency of pain as well as the periods between episodes of pain. If the suffering comes and goes leaving pain free intervals of noticeable duration, it is classified as intermittent. If such pain

free intervals do not occur, it is classified as continuous.

7. Duration of individual pains : A pain is said to be momentary if its duration can be expressed in seconds. Longer lasting pains are classified in minutes, hours or a day. A pain that continuous from one day to the next is said to be protracted.

8. Localization behavior : If the patient is able to define the pain to an exact anatomic location, it is classified as localized pain. If such description is less well defined and some what vague and variable anatomically, it is termed diffuse pain. Rapidly changing pain is classified as radiating. A momentary cutting exacerbation is usually described as lanciating. More gradually changing pain is usually described as spreading and if it progressively involves adjacent anatomic areas, the pain is called as enlarging. If it changes from one location to another, the complaint is described as migrating. Referred pain and secondary hyperalgesia are clinical expressions of secondary pain.

9. Associated symptoms such as hyperesthesia, anesthesia, hypoesthesia, parasthesia, and swelling should be noted. Any concomitant change in special senses affecting vision, hearing, smell or taste should be noted.

Establishing The Pain Category

The second major step in making a diagnosis is to establish the proper category of the patient's complaint.

1. Chronic pain

Evidence of chronicity: clinical indications

1. Continuity of pain input.
2. Protracted duration
3. Progressive non physiological behavior of the pain.
4. Progressive inadequacy of local cause.
5. Progressive emotional and physical determination

2. Neurogenous pain

Evidence of neurogenous pain – clinical evidence

1. Burning type pains that are spontaneous, triggered or ongoing and unremitting.
2. Pains that occur disproportionately to the stimulus.
3. Pains that are accompanied by other neurological symptoms.

3. Primary or secondary pain

Primary pains are arrested by local anesthesia at the site of pain. Primary somatic pains arising from superficial skin or mucogingiva is arrested by a topical application of anesthetic agent. Secondary pains are not arrested by analgesic blocking at the site of pain.

4. Superficial somatic pain

The pain is bright, stimulating sensation that is precisely localizable by the subject.

5. Deep somatic pain

1. The pain is a duller, more depressing sensation that is less precisely localizable by the subject.
2. Response to provocation is less faithful, especially with regard to location and size.
3. The pain frequently and expectantly exhibits secondary manifestations such as referred pain, secondary hyperalgesia, autonomic symptoms and muscle effects.
4. The pain is arrested by analgesic blocking of the site of pain.

6. Musculoskeletal pain

Musculoskeletal structures are primarily engaged in biomechanical function and pain from such structures exhibit two characteristic clinical features.

1. They relate intimately to biomechanical function.
2. Response to provocation is a gradient reaction that is proportional to the stimulus.

7. Inflammatory pain

1. Concomitant signs of inflammation such as swelling, redness, heat and dysfunction are present.
2. The pain reflects the type, intensity, location and phase of inflammatory reaction.
3. The pain follows an inflammatory time frame – it requires time to develop, plateau and resolve.

Confirmation of the clinical diagnosis

Before undertaking definitive therapy, confirmation of the clinical diagnosis is advisable. There are three methods that can help confirm the diagnosis:

1. Diagnostic analgesic blocking.
2. Utilization of diagnostic agents.
3. Consultations

Non Dental causes of facial pain

As most primary care practitioners are familiar with examining the oral cavities, these will not be detailed here. Oral malignancy, in particular gingival or alveolar bone malignancy, may present with facial pain. Clinical features of oral malignancy may also include sensory changes or trismus. Metastatic disease spreading to the jaws must also be considered. Evidence of oral candidosis or xerostomia may be relevant when excluding a diagnosis of burning mouth

syndrome (BMS). Pain associated with the salivary glands may occur as a result of salivary stones blocking the ducts and giving rise to intermittent pain typically associated with eating. There may be tenderness of the glands. Bimanual palpation of the submandibular glands is essential. Infections and tumours of the salivary glands may also give rise to localized pain in the region of the glands and a purulent discharge may be seen from the openings of the ducts within the mouth, indicative of infection. Pain may be relieved by massaging the glands involved.[3]

Burning Mouth Syndrome

Burning mouth syndrome has been defined as burning pain in the tongue or oral mucus membranes, usually accompanied without accompanying clinical and laboratory findings. Oral pain represents the cardinal symptom of BMS. Type I BMS is characterized by a pain free waking, with burning sensation developing in the late morning, gradually increasing in severity during the day, and reaching its peak intensity by evening. This type is linked to systemic disorders such as nutritional deficiency, diabetes, etc. Type II consists of continuous symptoms throughout the day, which, once started, often make falling asleep at night difficult for many

individuals. This subgroup of patients often reports mood changes, alterations in eating habits, and a decreased desire to socialize, which seem to be due to an altered sleep pattern. Common clinical findings in these subjects include parotid gland hypo function related to the use of antidepressant drugs. Type III BMS is characterized by intermittent symptoms with pain free periods during the day. Frequently, these patients show anxiety and allergic reactions, particularly to food additives. Overall, this sub classification is not universally considered essential for BMS patient management. However, it suggests the value of investigating possible local and systemic factors which ultimately lead to the neuropathic disturbance. Almost 70% of the BMS patients, persistent taste disorders are also evident. 46 to 67% of BMS patients complain of dry mouth. In these individuals, the feeling of oral mucosal dryness generally reflects a subjective sensation rather than one objective symptom of salivary gland dysfunction. Symptomatically, BMS must be differentiated from other chronic pain conditions such as painful traumatic/ inflammatory/ immune-mediated stomatitis or orofacial pain disorders. The location of the main symptom (pain) in the oral mucosa excludes diseases such as atypical facial pain, atypical odontalgia, and idiopathic facial arthromyalgia, which affect bones, teeth, muscles, and articulation, respectively. Specific details about pain, such as its localization, overall duration, and daily evolution, lead to a suspicion of BMS symptoms, rather than persistent oral mucosal lesions. Topical capsaicin administration have experienced a partial or even complete remission of pain. However, the proposed pharmacological protocols have not consistently proved to be predictable and effective in all BMS subjects. Vitamin B complex replacement therapy (pyridoxine, riboflavin, thiamine, etc.) may yield a good response in very few cases of patients with nutritional deficiency.[4-8]

Atypical Facial Pain (AFP)

It falls within the category of "Facial Pain Not Fulfilling Other Criteria" in the classification system of the International Headache Society (Classification, 2004)[9]. As this implies, the diagnosis is generally one of exclusion, that is, it is made only after local orofacial disease, neurological disorders, and related systemic diseases have been ruled out. It is not associated with objective neurological, facial, or oral findings and often presents with a non anatomical and even a migratory pattern. Other terms, used synonymously or as variations of the theme including Phantom tooth pain, Atypical odontalgia, Atypical facial neuralgia ,Migratory odontalgia ,Wandering tooth syndrome ,Dental causalgia .It is characterized by an intense, deep and constant pain. The pain is burning or aching, and it is poorly localized. Although mostly found on one side of the face, a bilateral occurrence is not uncommon. Generally the pain distribution does not follow anatomic pathways of the peripheral nerves.The cause and pathophysiology of AFP remain enigmas. There are several theories about the pathophysiology of AFP. The psychosocial factors are important in atypical facial pain, perhaps by opening or closing various "gates" either peripherally or centrally.Patients are generally not compliant with prescribed treatment,paradoxically, in spite of its known resistance to management, patients usually have had many different treatment approaches leading to no relief or even worsening the symptoms.[10]

Atypical Odontalgia (AO)

Atypical odontalgia is a diagnosis of exclusion and is therefore frequently associated with another ill defined pain syndrome, atypical facial pain. Unfortunately these terms constitute a waste basket for all unexplainable pain in the face. Although similar to AFP, AO is better defined anatomically.Atypical odontalgia has been referred as tooth pain with no obvious organic cause. It is a poorly understood phenomenon associated with persistent pain in apparently normal teeth and surrounding alveolar bone. Several causal theories to explain atypical odontalgia have been proposed, but little evidence has been found to support these theories [11]. Those problems most commonly described as causal for atypical odontalgia are Psychological disorders, Deafferentiation pain, Vascular pain.Of these theories, psychological dysfunction, usually depression, is the most commonly mentioned one.Treatment of atypical odontalgia is similar to that of other neuropathic conditions. Tricyclic antidepressants (TCAs), alone or in association with phenothiazines, have been prescribed with good results. Although these are mood-altering medications, their effectiveness is attributed to their ability to produce a low-grade analgesia in low doses.[11,12]

Maxillary sinusitis

Maxillary sinusitis causes a constant pain with zygomatic and dental tenderness from the inflammation of the maxillary sinus. [10] Acute maxillary sinusitis is defined by the International Association for the Study of Pain (IASP) as constant burning pain with zygomatic and dental tenderness from the inflammation of the maxillary sinus. In chronic cases there may be no pain or just occasional mild diffuse discomfort. Diagnostic criteriafor maxillary sinusitis include:

- Purulence in the nasal cavity;
-
- Simultaneous onset of headache and sinusitis;

Pain over the antral area which may radiate to the upper teeth or forehead; and Headache disappearing after treatment of acute sinusitis. The character of the pain of maxillary sinusitis is dull, aching, boring and tender, of mild to moderate severity, is usually continuous and may be either unilateral or bilateral starting after an upper respiratory tract infection. The pain is triggered by bending forward, touching the area or biting on the upper teeth. Headache is located over the antralarea. In the presence of the key diagnostics symptoms, investigations are not required but confirmation of the diagnosis can be confirmed by maxillary sinus radiographi cexamination (although this is not generally advised as, apart from showing possible fluid levels in acute sinusitis, it is not of great benefit), computerized tomography(CT) or magnetic resonance imaging (MRI).[13]

Temporomandibular disorders

Temporomandibular disorders encompass pain affecting the masticatory muscles and/or temporomandibular joints (TMJs). They consist of muscular pain MSK (referred to by some as myofascialpain), TMJ disc interference disorders and TMJ degenerative joint disease; this latter rarely causes pain but results in limitation of opening. In the case of trauma, the pain is usually self-limiting but psychological aspects may contribute to chronicity of the pain, therefore it is important to manage it early. TMD(MSK) is more prevalent in females and the natural history is that of

intermittent pain with continuation for many years.Tension type headaches can be mistaken for TMD. There is increasing evidence that TMD is linked to many other chronic pain conditions, such as headaches, migraine, post-traumatic stress disorder and fibromyalgia. The relationship between TMD pain and clenching habit or bruxism is far from simple and daily variations in pain do not correlate with self-reports of clenching or grinding.[14,15,16]

Trigeminal Neuralgia

Both the International Association for the Study of Pain (IASP) and International Headache Society (IHS) has suggested their own diagnostic criteria for trigeminal neuralgia. These are remarkably similar and highlight the sudden, explosive nature of the pain.18 Trigeminal neuropathy, whether painful or non-painful, is associated with a structural lesion or systemic disease. It may be seen following direct trauma to the nerve (e.g. supra- and infra-orbital neuralgias following facial fractures. Evidence has been mounting that in a large proportion of cases, compression of the trigeminal nerve root at or near the dorsal root entry zone by a blood vessel is a major causative or contributing factor. Trigeminal neuralgia is an extremely painful disorder commonly involving maxillary division followed by mandibular division and rarely ophthalmic branch of trigeminal nerve. Characterized by recurrent lancinating shock like unilateral pain lasting seconds to minutes is provoked by non noxious stimulation (allodynia) of the skin at specific sites around the face called trigger zones and less frequently by the movement of the tongue. These trigger zones are usually within the same dermatome as the painful sensation. After each episode there is usually a refractive period during which stimulation of the trigger zone will not induce the pain. The pain is limited to one or more branches of the Trigeminal nerve with no motor deficit in the affected area. No specific tests exist for the diagnosis of trigeminal neuralgia. A thorough clinical examination should be done, including assessment of cranial nerve function. Definite facial sensory loss or other cranial nerve dysfunction, if it cannot be explained by a previously known injury to the nerve, should prompt cerebral imaging. Even in typical trigeminal neuralgia, imaging studies may well be of use. Following the first reports of successful use of MRI in detecting vascular compression of the nerve in trigeminal neuralgia. Carbamazepine is something of a gold standard against which other drugs have been compared in subsequent controlled trials. These drugs include tizanidine, baclofen, pimozide, tocainade, and oxcarbazepine. Other treatment modalities include Microvascular diseection, radiofrequency gagngliolysis, balloon compression, peripheral neurectomy, cryotherapy, alcohol block and other peripherally targeted procedures producing controlled peripheral neural trauma include radiofrequency lesions and injections using glycerol, phenol, high concentration tetracaine or a mixture of lignocaine and streptomycin.In all, the average reported pain relief is measured in months and many of these methods are associated with a high number of initial failures. From the existing literature, it is difficult to draw conclusions as to their true efficacy, but it is doubtful whether they ever provide long-lasting benefit. Authors advocating the use of these measures frequently quote failure rates or complications from other, more invasive treatments that do not accurately reflect the current literature.[17-22]

Post Herpatic neuraligia

Herpes Zoster of the maxillary and mandibular divisions is a cause of orofacial pain. In majority of cases, the pain of Herpes Zoster resolves within a month after the lesions heal. Pain that persists longer than a month is classified as post herpetic neuralgia although some authors do not make the diagnosis of post herpetic neuralgia until the pain has persisted for longer than 3 or even 6 months. Post herpetic neuralgia may occur at any age but the major risk factor is increasing age. The pain and numbness of post herpetic neuralgia results from a combination of both central and peripheral mechanisms. The varicella zoster virus injures the peripheral nerves by demyelination, wallerian degeneration and sclerosis, but changes in the CNS including atrophy of dorsal horn cells in the spinal cord have been associated with post herpetic neuralgia.[23] This combination of peripheral and central injury results in the spontaneous discharge of neurons and an exaggerated painful response to non painful stimuli. Patients with post herpetic neuralgia experience persistent pain, parasthesia, hyperesthesia and allodynia months to years after the zoster lesions have healed. The pain is often accompanied by a sensory deficit and there is correlation between the degree of sensory deficit and the severity of pain. Episodes of severe lancinating or burning pain occur at the site of former eruption usually accompanied by hyperalgesia of the healed scars.[2] There is no trigger zone. Many treatment options are available which include topical and systemic drug therapy and surgery. Topical therapy includes the use of topical anesthetic agents such as Lidocaine or analgesics particularly Capsaicin. Lidocaine used either topically or injected gives short term relief from severe pain. Capsaicin, an extract of hot chilli peppers that depletes the neuro transmitter Substance P when used topically has been shown to be helpful in reducing the pain of post herpetic neuralgia but with a side effect of burning sensation at the site of application. The use of tricyclic antidepressants such as Amitriptyline, Doxepine and Desipramine is well established method of reducing the chronic burning pain that is characteristic of post herpetic neuralgia. In elderly patients gabapentin reduced pain and also improved sleep and overall quality of life. Patients who undergo episodes of shooting pain may experience relief through the use of anticonvulsant drugs such as carbamazepine or phenytoin.When medical therapy is ineffective in managing intractable pain nerve blocks or surgery at the level of the peripheral nerve or dorsal root has been effective. The best therapy for post herpetic neuralgia is prevention. There is evidence that the use of antiviral drugs particularly Famicyclovir along with a short course of systemic corticosteroids during the acute phase of the disease may decrease the incidence and severity of post herpetic neuralgia.[23-25]

Glossopharyngeal Neuralgia

Glossopharyngeal neuralgia is a rare condition, occurring with a frequency of about 1% of that of trigeminal neuralgia. Its reported incidence is approximately 0.8 per 100,000 people. The most common causes of glossopharyngeal neuralgia are intracranial or extracranial tumors and vascular abnormalities that compress cranial nerve IX.This neuralgia occurs without sex predilection in middle aged or older persons. The location of the trigger zone and pain sensation follows the distribution of the glossopharyngeal nerve and manifests itself as a sharp, shooting pain in the ear, the pharynx, the nasopharynx, the tonsil or the posterior portion of the tongue. It is almost invariably unilateral and the paroxysmal rapidly subsiding type of pain characteristic of trigeminal neuralgia is also a feature here. Pain is triggered by chewing,

talking and swallowing, all of which stimulate the pharyngeal mucosa. The pain can be easily confused with geniculate neuralgia due to the common ear symptoms or with temporomandibular disorders due to pain following jaw movement. Glossopharyngeal neuralgia may occur with trigeminal neuralgia, and when this occurs, a search for a common central lesion is essential. Glossopharyngeal neuralgia also maybe associated with vagal symptoms, such as syncope and arrhythmia owing to the close anatomic proximity of the two nerves. Application of topical anesthetic to the pharyngeal mucosa eliminates glossopharyngeal neuralgia and can aid in distinguishing it from other neuralgias. Medical treatment of glossopharyngeal neuralgia with carbamazepine or gabapentin can be effective in suppressing painful paroxysms. Although spontaneous remissions are common, the relapses may become refractory to drug therapy. Surgical methods include nerve section, tractotomy or microvascular decompression . Intracranial root section has been the most often employed and is generally effective but additional section of the upper vagal rootlets is considered necessary in some cases .More recently, endoscopy has been employed as the sole imaging modality in glossopharyngeal nerve decompression. **26-27**

Geniculate neuralgia

Geniculate neuralgia is an uncommon paroxysmal neuralgia of cranial nerve VII. It most commonly results from herpetic (zoster) inflammation of the geniculate ganglion and nerves. Ramsay-Hunt syndrome is Herpes Zoster of the nervus intermedius, the sensory component of the facial nerve. This rare syndrome causes neuritic pain and superficial herpetic lesions in the external ear auditory canal and mastoid area. Intra orally the heterotopic pain and herpetic lesions affect the fauces, soft palate and anterior part of the tongue. The symptoms result from inflammatory neuronal degeneration. The location of pain matches the sensory distribution of cranial nerve VII, namely external auditory canal and a small area on the soft palate and posterior auricular region. Pain may be provoked by stimulation of trigger zones within the ipsilateral distribution of the nerve. The pain is not as sharp or intense as in trigeminal neuralgia, and there is often some degree of facial paralysis, indicating simultaneous involvement of motor root. Viral vesicles may be observed in the ear canal or on the tympanum. High dose steroid therapy and acyclovir 800mg five times per day for 10-14 days significantly reduces the duration of the pain. Carbamazepine (200-800mg/day) may also be prescribed. Patients who do not respond to these medications may undergo surgery to section the nerves intermedius. [28]

Vascular pain disorders

Pains originating from vascular structures may cause facial pain that can be misdiagnosed for other oral disorders such as tooth ache or temporomandibular joint disorder. Following are the major pain disorders of vascular etiology that cause prominent orofacial signs and symptoms.

Cranial arteritis

Cranial arteritis (temporal arteritis, gaint cell arteritis) is an inflammatory disorder involving the medium – sized branches of the carotid arteries. The temporal artery is the most commonly involved branch. The blood vessel abnormality may be localized to the head and face or may be part of the generalized disease, polymyalgia rheumatica. Both cranial arteritis and polymyalgia rheumatica are caused by immune abnormalities that affect cytokines and T lymphocytes, resulting in inflammatory infiltrates in the walls of arteries. This infiltrate is characterized by the formation of multinucleated gaint cells. The underlying trigger of the inflammatory response is unknown.Cranial arteritis most frequently affects adults above the age of 50 years. Patients have a throbbing headache accompanied by generalized symptoms including fever, malaise, and loss of appetite. Patients with polymyalgia rheumatica will have accompanying joint and muscle pain. Examination of the involved temporal artery reveals a thickened pulsating vessel. Since the mandibular and lingual arteries may be involved, a throbbing pain in the jaw or tongue may be an early sign or even a presenting sign. A serious complication in untreated patients is ischemia of the eye, which may lead to progressive loss of vision or sudden blindness. These visual manifestations may be prevented by early diagnosis and prompt therapy. 18Laboratory abnormalities include an elevated erythrocyte sedimentation rate (ESR) and anemia. Abnormal C-reactive protein may also be an important early finding. The most definitive diagnostic test is a biopsy specimen (from the involved temporal artery) that demonstrates the characteristic inflammatory infiltrate. Since the entire vessel is not involved, an adequate specimen must be taken to detect the changes. A negative biopsy result does not rule out temporal arteritis, and the diagnosis should continue to be considered in patients over 50 years or age who have chronic pounding head or orofacial pain and an elevated ESR. [29]

Sphenopalatine neuralgia (Cluster Headache)

Sphenopalatine neuralgia is a pain syndrome originally described as a symptom complex referable to the nasal ganglion. In recent years the term "periodic migrainous neuralgia" has been used to describe the clinical symptom The most widely accepted evidence currently indicates that this syndrome is caused by vasodilatation involving the internal maxillary artery, a branch of the external carotid particularly that portion supplying the sphenopalatine region. Is characterized by unilateral paroxysms of intense pain in the region of the eyes, the maxilla, the ear and mastoid, base of the nose, and beneath the zygoma. Sometimes the pain extends into the occipital areas as well. These paroxysms of pain have a rapid onset, persist for about 15 minutes to several hours, and then disappear a rapidly as they began. There is no trigger zone. The attacks develop regularly, usually at least once a day, over a prolonged period of time. Interestingly, in some patients the onset of the paroxysm occurs at exactly the same time of day and, for this reason, the disease has been referred to as "alarm clock" headache. After some weeks or months, the attacks disappear completely and this period of freedom may persist for months or even years. Sneezing, swelling of the nasal mucosa and severe nasal discharge often appear simultaneously with the painful attacks, as well as epiphora, or watering of the eyes, and blood shot eyes, Paraesthetic sensations of the skin over the lower half of the face also are reported. Individuals with cranial arteritis should be treated with systemic corticosteroids as soon as the diagnosis is made. The initial dose ranges between 40 to 60 mg of prednisone per day, and the steroid is tapered once the signs of the disease are controlled. The ESR may be used to help monitor disease status. Patients are maintained on systemic steroids for 1 to 2 years after symptoms resolve. Steroids may be supplemented by adjuvant therapy with

immunosuppressive drugs, such as cyclophophamide, to reduce the complications of long-term corticosteroid therapy. Immediate steroid therapy should be initiated if visual symptoms are present.An acute attack of cluster headache can be aborted by breathing 100% oxygen, and cluster headache patients may keep an oxygen canister at bedside to use at the first sign of an attack. Several drug protocols are recommended for preventing cluster headache during active periods. Lithium is effective therapy for those who can tolerate the side effects, and patients who are using long term lithium must be monitored for renal toxicity. Other drugs that are useful for preventing attacks include ergotamine, prophylactic prednisone, and calcium channel blockers. Methysergide is also effective therapy, best pulmonary or cardiac fibrosis are potential side effects, particularly during prolonged use. [30]

Chronic paroxysmal hemicrania

Chronic paroxysmal hemicrania is believed to be form of cluster headache that occurs predominantly in women between the ages of 30 to 40 years. The episodes of pain tend be shorter, but attacks of 5 to 20 minutes duration can occur up to 30 times daily. Initially episodes of chronic paroxysmal hemicrania occur with a periodicity similar to that of cluster headache; however, chronic paroxysmal hemicrania symptoms tend to become chronic over time. Chronic paroxysmal hemicrania responds dramatically to therapy with indomethacin, which stops the attacks within 1 to 2 days. Chronic paroxysmal hemicrania will recur if indomethacin is discontinued.[3]

Migraine

Migraine is the most common of the vascular headaches, which may occasionally also cause pain of the face and jaws. It may be triggered by foods such as nuts, chocolate, and red wine, stress, sleep deprivation, or hunger. Migraine is more common in women. The classic theory is that migraine is caused by vasoconstriction of intracranial vessels (which causes the neurologic symptoms), followed by vasodilatation (which results in pounding headache). Newer research techniques suggest a series of factors, including the triggering of neurons in the midbrain that activate the trigeminal nervous system in the medulla resulting in the release of neuropeptides such as substance P. These neurotransmitters activate receptors on the cerebral vessels walls, causing vasodilatation and vasoconstriction. There are several major types of migraine: classic, common, basilar, and facial migraine (also referred to as Carotodynia). Classic migraine starts with a prodromal aura that is usually visual but that may also be sensory or motor. The visual aura that commonly precedes classic migraine includes flashing lights or a localized area or depressed vision (scotoma). Sensitivity to light, hemi anesthesia, aphasia, or other neurologic symptoms may also be part of the aura, which commonly lasts from 20 to 30 minutes. The aura is followed by an increasingly severe unilateral throbbing headache that is frequently accompanied by nausea and vomiting. The patient characteristically lies down in a dark room and tries to fall asleep. Headaches characteristically last for hours up to 2 or 3 days. Common migraine is not preceded by an aura, but patients may experience irritability or other mood changes. The pain of common migraine resembles the pain of classic migraine and is usually unilateral, pounding, and associated with sensitivity to light and noise. Nausea and vomiting are also common.Basilar migraine is most common in young women. The symptoms are primarily neurologic and include aphasia, temporary blindness, vertigo, confusion, and ataxia. These symptoms may be accompanied by an occipital headache. Facial migraine (Carotodynia) causes a throbbing and / or sticking pain in the neck or jaw. The pain is associated with involvement of branches of the carotid artery rather than the cerebral vessels.14 The symptoms of facial migraine usually begin in individuals who are 30 to 50 years of age. Patients often seek dental consultation, but unlike the pain of a toothache, facial migraine pain is not continuous but lasts minutes to hours and recurrent tenderness of the carotid artery. Face and jaw pain may be the only manifestation or migraine, or it may be an occasional pain in patients who usually experience classic or common migraine. Patients with migraine should be carefully assessed to determine common food triggers. Attempts to minimize reactions to the stress of everyday living by using relaxation techniques may also be helpful to some patients. Drug therapy may be used either prophylactically to prevent attacks in patients who experience frequent headaches or acutely at the first sign of an attack. Drugs that are useful in aborting migraine include ergotamine and sumatriptan, which can be given orally, nasally, rectally or parentally. These drugs must be used cautiously since they may cause hypertension and other cardiovascular complications. Drugs that are used to prevent migraine include propranolol, verapimil, and TCAs. Methysergide or monoamine oxidase inhibitors such as phenelzine can be used to manage difficult cases that do not respond to safer drugs.[31]

Tension type headahe

It is the most common type of headache, formerly known as tension or muscle contraction headache. The pain is bilateral, deep and often band like from the occipital to frontal region and often worsens as the day progresses, lasts from hours to days and can be chronic or episodic. This condition occurs nearly equally among men and women and the age of onset is variable. Trigger points and tender spots are commonly observed in the pericranial and cervical muscles. Although migraine and tension type head aches generally have unique and distinguishing features, there can be considerable over lap of symptoms so that some headaches have mixed features. These are so called mixed headaches, or "benign recurring headaches of variable severity" usually develop in older individuals who previously had more typical migraine of tension type headaches. It is not uncommon for a tension type headache to develop the features of a migraine headache as the symptoms become more chronic and intense. [32]

Conclusion

Differentiating the many causes of facial pain can be difficult for busy practitioners, but a logical approach to history-taking is important and will aid more rapid diagnoses with effective management. Although primary care clinicians are not be expected to diagnose rare pain conditions, they should be able to assess the presenting pain complaint and refer to the appropriate centre. It is important that primary care practitioners provide sufficient detailed information of history, examination and investigation findings in their referral letters to ensure appropriate direction of the referral within the secondary/tertiary care institution. Underlying causes of orofacial pain are wide ranging and complex, but a greater understanding of a patient's facial pain symptoms, towards establishing a diagnosis or differential diagnosis, can be achieved by obtaining a good pain history and carrying out a good clinical

examination.

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