The Causes of Facial Pain are Numerous

Siniša Franjić
Faculty of Law, International University of Brcko District, Bosnia and Herzegovina

Abstract

One of the most difficult problems in modern medicine is facial pain. Sometimes an experienced doctor does not immediately recognize the symptoms and makes a misdiagnosis. The causes of facial pain are numerous. Therefore, the patient should be examined by physicians of several specializations.

Introduction

Facial pain occurring in the absence of trauma may be caused by a variety of disorders, many of which may be associated with referred pain, thereby making accurate localization of the source difficult [1]. For this reason, a careful examination of the face, orbits, eyes, oral and nasal cavities, auditory canals, and temporomandibular joints is an essential aspect of the evaluation of these patients.

Pain can result from many different disease processes [2]. The most common causes of facial pain are trauma, sinusitis, and dental disease. The history suggests the diagnosis, which is usually confirmed with the physical findings. With appropriate treatment and resolution of the disease, the pain also abates. Sometimes the cause of the pain is not apparent or the pain does not resolve with the other symptoms.

The trigeminal nerve (cranial nerve V) supplies sensation to the face. The first division (ophthalmic) supplies the forehead, eyebrows, and eyes. The second division (infraorbital) supplies the cheek, nose, and upper lip and gums. The third division (mandibular) supplies the ear, mouth, jaw, tongue, lower lip, and submandibular region. When pain is located in a very specific nerve distribution area, lesions involving that nerve must be considered. Tumors involving the nerve usually cause other symptoms, but pain may be the only complaint, and presence of a tumor at the base of the skull or in the face must be ruled out. When the work-up is negative, the diagnosis may be one of many types of neuralgia, which is a pain originating within the sensory nerve itself. Treatment is medical or, in some cases, surgical.

After immobilization, patients who are unconscious without respiratory effort require intubation to establish a functional airway, and this must be a first priority [3]. Laryngoscopically guided oral intubation is the technique of choice and must be undertaken without movement of the cervical spine; an assistant is essential in this regard and should remain at the patient’s head providing constant, in-line stabilization. Patients with inspiratory effort may be nasotracheally intubated provided that significant maxillofacial, perinasal, or basilar skull injuries are not present; when present or suspected, nasotracheal intubation is relatively contraindicated.

Facial pain remains a diagnostic and therapeutic challenge for both clinicians and patients [4]. In clinical practice, patients suffering from facial pain generally undergo multiple repeated consultations with different specialists and receive

---

Citation: Siniša Franjić. The Causes of Facial Pain are Numerous. Jour of Clin Cas Rep, Med Imag and Heal Sci 1(1)-2022.
various treatments, including surgery. Many patients, as well as their primary care physicians, mistakenly attribute their pain as being due to rhinosinusitis when this is not the case. It is important to exclude non-sinus-related causes of facial pain before considering sinus surgery to avoid inappropriate treatment. Unfortunately, a significant proportion of patients have persistent facial pain after endoscopic sinus surgery (ESS) due to erroneous considerations on aetiology of facial pain by physicians. It should be taken into account that neurological and sinus diseases may share overlapping symptoms, but they frequently co-exist as comorbidities. The aim of this review was to clarify the diagnostic criteria of facial pain in order to improve discrimination between sinogenic and non-sinogenic facial pain and provide some clinical and diagnostic criteria that may help clinicians in addressing differential diagnosis.

**History**

Facial pain is pain localised to the face, and the diagnosis of facial pains has puzzled clinicians for centuries [5]. Some of the confusion is related to the delimitation of the facial structure and how pain is classified. The face is here defined as the part of the head that is limited by the hairline, by the front attachment of the ear and by the lower jaw, both the rear edge and the lower horizontal part of the jaw. The face also includes the oral and nasal cavity, the sinuses, the orbital cavity and the temporomandibular joint. Pain in the facial region can be classified in multiple ways, for example according to underlying pathology (malignant vs. non-malignant), the temporal course (acute vs. chronic), underlying pathophysiology (neuropathic, inflammatory or idiopathic), localisation (superficial vs. deep), the specific structure involved (the sinus joint, skin etc), and underlying etiology (infection, tumour etc). In some instances, the diagnosis of facial pain focuses on the involved structure, for example temporomandibular joint disorder, in other cases it is the underlying pathology (sinusitis), and in others it is the specific character of the pain that will dictate the diagnosis (e.g. trigeminal neuralgia).

A history of carious dentition in association with a gnawing, intolerable pain in the jaw or infraorbital region is seen in patients with gingival or dental abscesses [1]. Pressurelike pain or aching in the area of the frontal sinuses, supraorbital ridge, or infraorbital area in association with fever, nasal congestion, postnasal discharge, or a recent upper respiratory tract infection suggests acute or chronic sinusitis. Redness, swelling, and pain around the eye are suggestive of periorbital cellulitis. The rapid onset of parotid or submandibular area swelling and pain, often occurring in association with meals, is characteristic of obstruction of the salivary duct as a result of stone. Trigeminal neuralgia produces excruciating, lancinating facial pain that occurs in unexpected paroxysms, is initiated by the tactile stimulation of a “trigger point” or simply by chewing or smiling. Temporomandibular joint dysfunction produces pain related to chewing or jaw movement and is most commonly seen in women between the ages of 20 and 40 years; patients may have a history of recent injury to the jaw, recent dental work, or long-standing malocclusion. Facial paralysis associated with facial pain may be noted in patients with malignant parotid tumors. Dislocation of the temporomandibular joint causes sudden local pain and spasm and inability to close the mouth. Acute dystonic reactions to the phenothiazines and antipsychotic medications may closely simulate a number of otherwise perplexing facial and ocular presentations and must be considered. Acute suppurative parotitis usually occurs in the elderly or chronically debilitated patient and causes the rapid onset of fever, chills, and parotid swelling and pain, often involving the entire lateral face.

**Injuries**

Facial injuries are among the most common emergencies seen in an acute care setting [6]. They range from simple soft tissue lacerations to complex facial fractures with associated significant craniofacial injuries and soft tissue loss. The management of these injuries generally follows standard surgical management priorities but is rendered more complex by the nature of the numerous areas of overlap in management areas, such as airway, neurologic, ophthalmologic, and dental. Also, the significant psychological nature of injuries affecting the face and the resultant aftermath of scarring can have devastating and long-lasting consequences. Despite the fact that these injuries are exceedingly common, they are cared for by a large group of different specialists and as such have a remarkably heterogeneous presentation and diverse treatment schema. Nonetheless, guiding principles in the care of these injuries will provide the basis for the best possible outcomes. The following questions will guide general management and provide a framework for understanding the principles in the acute care of patients with facial injuries and trauma.

Despite the extremely common presentation of such injuries, there remains little standardization on repairing and then caring for the wounds or lacerations. There is great variation in the repair of lacerations as well as the different materials used to repair them. This is again because of the numerous different specialties involved in the care of the injuries and their desires to provide the best possible outcome with regard to scarring. Pediatricians, emergency department personnel, and surgeons may not all agree on the best modalities for repair. Placement as well as type of...
dressing are also controversial.

The timing of facial skin laceration closure is the same as that of any open wound. The presence of contaminating factors in the management of wound would generally not allow closure after six hours and would favor delayed closure. However, clinical practice is slightly more variable with facial lacerations because of the uniquely sensitive nature of facial scarring. Although we generally ascribe to experimental data regarding timing of closure, in practice the six-hour rule is often overlooked with an attempt to be vigorous in cleaning the wound. The presence of exceptionally rich blood supply in the face is also deemed of benefit in extending the six-hour rule.

TN

Facial pain, for all its rarity, can be a significant cause of morbidity when present [7]. The two types of non-odontological causes of facial pain that appear to be the most likely to be mistaken one for the other are trigeminal neuralgia (TN) and what used to be called atypical facial pain, but that is now called persistent idiopathic facial pain (PIFP). Confusion between causes of facial pain persists despite the fact that the diagnosis of classical TN should be rather straightforward and not present diagnostic difficulties to the trained clinician. (The term classical TN is generally restricted to TN caused by neurovascular compression.) The caveat is that secondary causes of TN need to be considered, and the cause of classical TN needs to be established for reasons that will be discussed later. A common mistake that should not be made is to treat TN medically without establishing the cause. PIFP, on the other hand, is a diagnostic problem that confronts us head on. Clearly stated guidelines are in fact ambiguous. Descriptive terms include dull, poorly defined, non-localized.

Individuals in whom attacks of pain last minutes to hours, or are persistent or chronic, waxing and waning over the course of the day, or in whom pain extends beyond one division of the trigeminal nerve, may still be mistakenly diagnosed as having trigeminal neuralgia. Such individuals may point to one side of the face as the site of their pain or may indicate that pain is bilateral. Their pain may be further atypical in lacking the usual triggers of pain such as brushing teeth or touching a trigger area. Such pain that is atypical for TN is a different kind of facial pain than classical TN. However, even in cases that are not characteristic trigeminal neuralgia, chewing, and even speaking, for example, may be triggers. Chewing and speaking activate orofacial and neck muscles, and are accompanied by small movements at the cervical–cranial junction. Nociceptive sites in these muscles may be activated by chewing or speaking. Patients with atypical facial pain are unlikely to have trigeminal neuralgia, and more likely to have what is now called persistent idiopathic facial pain (PIFP).

The diagnosis of classical TN is made on the basis of a characteristic history of lightning-like sharp, electrical pain that is felt in one division of the trigeminal nerve, leaving a dull after pain that lasts for a variable, usually short, period of time. There is often a trigger, but there does not need to be one. The attacks are typically infrequent at first, but become more frequent with the passage of time, and may increase in frequency to occur hundreds of times a day. Remissions occur, but relapses become more frequent with aging. There is no dullness or loss of feeling reported. Some patients tell atypical stories in which pain crosses divisions of the trigeminal nerve, or paroxysms of pain last longer than lightning attacks of pain. The neurological examination is normal in classical TN. Motor and sensory examination of the face in particular is normal in classical TN, but is useful in identifying secondary trigeminal nerve dysfunction that could lead to a diagnosis of secondary TN or trigeminal neuropathy. The same is true of the blink and other trigeminal reflex tests, as the presence or absence of abnormal result does not affect the diagnosis of TN, but may indicate a need to examine for causes of secondary TN.

TMD

Painful temporomandibular disorder (TMD) is the most frequent form of chronic orofacial pain, affecting an estimated 11.5 million US adults with annual incidence of 3.5%. As with several other types of chronic, musculoskeletal pain, the symptoms are not sufficiently explained by clinical findings such as injury, inflammation, or other proximate cause [8]. Moreover, studies consistently report that TMD symptoms exhibit significant statistical overlap with other chronic pain conditions, suggesting the existence of common etiologic pathways. Most studies of overlap with orofacial pain have focused on selected pain conditions, classified according to clinical criteria (eg, headaches, cervical spine dysfunction, and fibromyalgia), location of self-reported pain (eg, back, chest, stomach, and head), or the number of comorbid pain conditions. Although there is a long tradition of depicting overlap between pain conditions qualitatively using Venn diagrams, we know of few studies that have quantified the degree of overlap between TMD and pain at multiple locations throughout the body.

Overlap of pain symptoms can occur when there are common etiologic factors contributing to each of the overlapping pain conditions. One example is diabetes that contributes, etiologically, to neuropathy in the feet and retinopathy in the eye, thereby creating overlap, statistically,
of diseases at opposite ends of the body. The etiologic factor most widely cited to account for overlap of pain conditions is central sensitization, defined as “amplification of neural signaling within the central nervous system (CNS) that elicits pain hypersensitivity.” The amplification means that otherwise innocuous sensations are perceived as painful (ie, allostynia) and that formerly mildly painful stimuli now evoke severe pain (ie, hyperalgesia). However, somatosensory afferent inputs into the CNS are segmentally organized, making it plausible that sensitization is not uniform throughout the neuraxis.

Regardless of pain location, overlap creates serious problems for patients, adding to the suffering and disability caused by a single pain condition, and potentially complicating diagnosis and treatment for one or all of the overlapping conditions. This has broader implications for patients with multiple chronic illnesses who have poorer health outcomes and generate significantly greater health care costs than patients with a single illness. Thus, the aim of this epidemiological study was to quantify the degree of overlap between facial pain and pain reported elsewhere in the body.

CRS

Unfortunately, little is known of the underlying mechanisms that produce pain associated with CRS (chronic rhinosinusitis), but several mechanisms that may all contribute to some degree to the manifestation of facial pain in CRS have been postulated [9]. It has been hypothesized that occlusion of the osteomeatal complex may lead to gas resorption of the sinuses with painful negative pressures, yet most subjects with CRS have an open osteomeatal complex. Patients’ observations that pain and pressure is postural may reflect painful dilatation of vessels; however, postural pain is also observed in subjects with simply tension type headache. Local inflammatory mediators can excite nerves locally within the sinonasal mucosa directly illiciting pain. For example, maxillary rhinosinusitis can cause dental pain through the stimulation of the trigeminal nerve. In addition, local tissue destruction and inflammatory mediators may influence the central mechanism of pain via immune-to-brain communication through afferent autonomic neuronal transmission, transport across the blood brain barrier through the circumventricular organs and/or direct passage across the blood brain barrier.

The impact of inflammatory cytokines on the central nervous system have been associated with both pain as well as other health-related factors associated with chronic inflammation and sickness behavior such as disruption of sleep and mood. Interleukin-1[Beta] (IL-1[Beta]) and tumor necrosis factor-[alpha] (TNF-[alpha]) are two key pro-inflammatory cytokines with a pivotal role in the immune-to-brain pathway of communication. They are both upregulated in subjects with CRS and are two potential pro-inflammatory cytokines that have been implicated in fatigue, sleep dysfunction, depression, and pain. Characterizing the differential cytokine profiles of CRS subtypes and identifying associated symptom profiles may be an important step in understanding why some subjects experience greater health-related burden of disease, which is an important predictor of electing surgical intervention over continued medical therapy.

Examination

Carious dentition, gingivitis, and gingival abscesses may be diagnosed by inspection of the oral cavity and face [1]. Percussion tenderness over the involved tooth, swelling and erythema of the involved side of the face, and fever may be noted in patients with deep abscesses. Percussion tenderness to palpation or pain over the frontal or maxillary sinuses with decreased transillumination of these structures suggests sinusitis. Redness, tenderness, and swelling around the eye may suggest periorbital cellulitis. Pain with eye movement or exophthalmos may suggest an orbital cellulitis or abscess. Malocclusion may be noted in patients with temporomandibular joint dysfunction; tenderness on palpation of the temporomandibular joint, often best demonstrated anteriorly in the external auditory canal with the mouth open, is noted as well. Patients with temporomandibular joint dislocation present with anxiety, local pain, and inability to close the mouth. Unusual ocular, lingual, pharyngeal, or neck symptoms should suggest possible acute dystonic reactions. A swollen, tender parotid gland may be seen in patients with acute parotitis, in parotid duct obstruction secondary to stone or stricture, and in patients with malignant parotid tumors; evidence of facial paralysis should be sought in these latter patients. Palpation of the parotid duct along the inner midwall of the cheek will occasionally reveal a nodular structure consistent with a salivary duct stone. In patients with herpes zoster, typical lesions may be noted in a characteristic dermatomal pattern along the first, second, or third division of the trigeminal nerve or in the external auditory canal. It is important to remember that patients with herpes zoster may have severe pain before the development of any cutaneous signs. This diagnosis should always be considered when vague or otherwise undefinable facial pain syndromes are described. Simple erythema may be the first cutaneous manifestation of herpetic illness. Patients with trigeminal neuralgia have an essentially normal examination.
Ventilation

In patients with inspiratory effort but without adequate ventilation, mechanical obstruction of the upper airway should be suspected and must be quickly reversed [3]. The pharynx and upper airway must be immediately examined and any foreign material removed either manually or by suction. Such material may include blood, other secretions, dental fragments, and foreign body or gastric contents, and a rigid suction device or forceps is most effective for its removal. Obstruction of the airway related to massive swelling, hematoma, or gross distortion of the anatomy should be noted as well, because a surgical procedure may then be required to establish an airway. In addition, airway obstruction related to posterior movement of the tongue is extremely common in lethargic or obtunded patients and is again easily reversible. In this setting, insertion of an oral or a nasopharyngeal airway, simple manual chin elevation, or the so-called jaw thrust, singly or in combination, may result in complete opening of the airway and may obviate the need for more aggressive means of upper airway management. Chin elevation and jaw thrust simply involve the manual upward or anterior displacement of the mandible in such a way that airway patency is enhanced. Not uncommonly, insertion of the oral airway or laryngeal mask airway may cause vomiting or gagging in semialert patients; when noted, the oral airway should be removed and chin elevation, the jaw thrust, or the placement of a nasopharyngeal airway undertaken. If unsuccessful, patients with inadequate oxygenation require rapid sequence oral, or nasotracheal, intubation immediately.

If an airway has not been obtained by one of these techniques, Ambu-bag–assisted ventilation using 100% oxygen should proceed while cricothyrotomy, by needle or incision, is undertaken rapidly. In children younger than 12 years, surgical cricothyrotomy is relatively contraindicated and needle cricothyrotomy (using a 14-gauge needle placed through the cricothyroid membrane), followed by positive pressure insufflation, is indicated. During the procedure, or should the procedure be unsuccessful, Ambu-bag–assisted ventilation with 100% oxygen and an oral or a nasal airway may provide adequate oxygenation.

In addition, rapidly correctable medical disorders that may cause central nervous system and respiratory depression must be immediately considered in all patients and may, in fact, have precipitated the injury by interfering with consciousness. In all patients with abnormalities of mental status, but particularly in those with ventilatory insufficiency requiring emergent intervention, blood should immediately be obtained for glucose and toxic screening, and the physician should then prophylactically treat hypoglycemia with 50 mL of 50% D/W, opiate overdose with naloxone (0.4–2.0 mg), and Wernicke encephalopathy with thiamine (100 mg). All medications should be administered sequentially and rapidly by intravenous injection and any improvement in mental status or respiratory function carefully noted. Should sufficient improvement occur, other more aggressive means of airway management might be unnecessary.

Conclusion

Facial pain can be painful and frightening. Facial pain can be caused by a cold, sinusitis, muscle tension in the jaw or neck, dental problems, nerve irritation or trauma. One of the most common causes is sinusitis, but another common cause is jaw dysfunction which often occurs after trauma and can lead to jaw injury or meniscus irritation. In the case of major trauma, fractures of the jawbone or fractures of the face may also occur.

References