Curr Pain Headache Rep (2010) 14:33–40
DOI 10.1007/s11916-009-0085-y

Chronic Orofacial Pain

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Published online: 10 January 2010

Abstract *Chronic orofacial pain* (COFP) is an umbrella term used to describe painful regional syndromes with a chronic, unremitting pattern. This is a convenience term, similar to chronic daily headaches, but is of clinically questionable significance: syndromes that make up COFP require individually tailored diagnostic approaches and treatment. Herein we describe the three main categories of COFP: musculoskeletal, neurovascular, and neuropathic. For many years, COFP and headache have been looked upon as discrete entities. However, we propose the concept that because COFP and headaches share underlying pathophysiological mechanisms, clinical characteristics, and neurovascular anatomy, they should be classified together.

Keywords Myofascial pain · Neurovascular · Burning mouth syndrome · Neuropathy

Introduction

Orofacial pain is prevalent in the general population—around 17–26%, of which 7–11% is chronic [1]. For clinical purposes we divide chronic orofacial pain (COFP) into three main symptomatic classes: musculoskeletal, neurovascular, and neuropathic [2••]. Table 1 summarizes the salient diagnostic features of the more common entities. This article reviews the clinical presentation of COFP entities, but not their pathophysiology and treatment [3].

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Musculoskeletal Pain

Musculoskeletal pains, consisting of disorders affecting the temporomandibular joint or the muscles of mastication, are classified under the umbrella term *temporomandibular disorders* (TMD). The term TMD is of limited value, as it classifies articular problems with muscle disorders that have different etiologies and treatment approaches, but researchers often continue to group these together.

TMD-related facial pain has been found to occur in 4–12% of the population, and severe symptoms are reported by 10% of subjects. Some signs or symptoms are extremely common; joint clicking, for example, is found in 20–30% of the population. However, only 3–11% are assessed as needing treatment, and TMD treatment is demanded by up to 7% of the general population [4]. Masticatory muscle and temporomandibular joint (TMJ) tenderness are present in about 15% and 5% of the population, respectively [5]. However, muscle tenderness may be "asymptomatic" in a third of such cases.

Masticatory Myofascial Pain

Clinical Features

Masticatory myofascial pain (MMP) is characterized primarily by pain and tenderness from the jaw-closing muscles, often with resultant masticatory dysfunction. At present, the diagnosis of MMP is based on the history and clinical examination of the patient [6, 7].

MMP is characterized by regional, unilateral pain, typically around the ear, the angle/body of the mandible, and the temporal region. Referral patterns include intraoral, auriculotemporal, supraorbital, and maxillary areas,

Table 1 Diagnostic features of chronic orofacial pain in comparison to headache

Parameter	Migraine	NVOP	TTH	MMP	TN
Demographics					
Onset age	20–30	40–50	20–30	20–40	50–60
Gender ratio (M:F)	1:3	1:3	5:4	1:2	1:2
Family history	60%	N/A	None	None	None
Prevalence/1000	100–150	R	180–430[a]	30–70	0.043
Pain duration	4 h to 3 d	1–12 h	30 min to 7 d	5 h to days	<120 s (PTN)
Unilaterality, %	80	90	<10	60	96
Sleep association	REM, III/IV	+	–	–	–
Awakens subjects	+	+	–[b]	Rare[b]	+
Time/frequency	Early morning	35%	N/A	N/A	20%
Frequency	1–4/min	20/min	6/min	24/min	High[c]
Predominant temporal pattern	Episodic	Chronic	Episodic	Chronic	Triggered
Changes sides	Yes	Yes	Bilateral	No	No
Intensity	++/+	++	+/++	+/++	++++
Paroxysmal	+	+	–	–	+
Throbbing	++	++	–	–	-(PTN)
Location	Frontal	Lower	Frontal/temporal	Mandible	II > III > I
Remission	Pregnancy	–/?	+/–	+	Weeks to years
Triggering	Multiple factors	?	Multiple factors	Jaw function	Mechanical
Muscle sensitivity	+	+/–	++/–	+++	–
Worse with physical activity	+	+/–	+/––	–	–
Autonomic signs	+	+/–	–	–	+/–
Systemic signs	+++	+	Chronic>episodic	–	–

Severity of pain, signs, and symptoms are graded as follows: –, absent; +, mild; ++, moderate; +++, severe; ++++, very severe

MMP masticatory myofascial pain; *NVOP* neurovascular orofacial pain; *PTN* pretrigeminal neuralgia; *REM* rapid eye movement; *TTH* tension-type headache; *TN* trigeminal neuralgia

[a] Relates to frequent episodic TTH

[b] Sleep disturbances common

[c] Usually related to triggering events, but of normally high frequency

depending on the muscles involved and the intensity of the pain [8]. Referral to the teeth may be prominent. Trigger points in the deep part of the masseter refer to the TMJ and ear, causing possible misdiagnosis with intra-articular or ear disorders.

Although MMP is typically a unilateral pain syndrome, it may also occur bilaterally, particularly when associated with generalized disorders such as fibromyalgia. Pain quality is dull, heavy, or aching. Pain severity may fluctuate during the day, but it is usually about 3 to 5 on a 10-cm visual analogue scale (VAS) and varies considerably across patients [9••]. MMP is mostly chronic and unremitting [9••, 10].

In addition to pain, there may be fullness of the ear, dizziness, and soreness of the neck. Dizziness has been associated with pain in the sternocleidomastoid muscle and ear stuffiness with spasm of the medial pterygoid. Examination usually reveals limited mouth opening (<40 mm between front teeth) and deviation of the mandible on opening. The presence of limited mouth opening in an MMP patient may also indicate TMJ pathology. Localized tender sites and trigger points in ipsilateral masticatory muscle, tendon, or fascia are distinguishing features of MMP patients [11]. A hypersensitive bundle or nodule of muscle fiber of harder than normal consistency is consistent with a trigger point. Trigger points may be active (induce clinical symptoms) or latent, in which case they only induce pain on stimulation. As has been pointed out, tender points are far more common in MMP patients than are trigger points [6, 9••].

The masseter muscle is most commonly involved (>60%), and the medial pterygoid and temporalis muscles are tender in about 40–50% of cases, commonly unilaterally. The sternocleidomastoid, trapezius, and suboccipital muscles are usually tender in 30–45% of patients, very often bilaterally.

Differential Diagnosis

Pain referral to teeth has often caused serious misdiagnosis and unwarranted dental treatment. The differentiation from painful TMJ disorders may also be complex due to overlapping symptomatology; regional pain, pain-referral patterns, and pain evoked by mandibular movement, which are common to both MMP and TMJ disorders. The clinician must be alert to the possible contribution of systemic comorbidities such as fibromyalgia, hypothyroidism, connective tissue disease, and AIDS. It should be noted that MMP is very much like tension-type headache (TTH), with two major limitations: most MMP pain is unilateral and largely located around the angle of the mandible and ear. Thus, although not common, bilateral MMP may be quite similar to TTH in both symptomatology and clinical findings (muscle tenderness), but the location of MMP in the mandibular and periauricular regions is characteristic. There is also much overlap in the pathophysiology of MMP and TTH.

Pain Associated with the Temporomandibular Joint

The most common conditions associated with pain of the TMJ are disc displacements; these may be painful when associated with joint inflammation [12]. More rarely, degenerative joint disease or inflammatory arthritic diseases may affect the TMJ. Often these TMJ entities are comorbid with muscle pain, usually MMP, and thus require a combined treatment approach. More rarely, the TMJ may be affected by infectious or metabolic arthritides [13].

Disc displacement signifies an abnormal anatomical relation between the condylar head of the mandible and the articular disc when the jaw is closed. In most cases, the disk is anteriorly displaced. In patients with anterior disc displacement with reduction (ADDwR), during mouth opening the condyle on the affected side meets the posterior band of the anteriorly displaced disc, encounters some resistance, then moves under the disc (reduction). During closing, the condyle will slip off the anteriorly positioned disc and return into the glenoid fossa. It is the sound that the condyle makes on mounting the disc during opening and then slipping off during closing that causes the characteristic reciprocal clicking of ADDwR, and these are easily felt by palpation anterior to the ears. Patients with ADDwR may be totally asymptomatic and only report joint clicking or popping sounds that may or may not concern them. ADDwR may, however, be symptomatic with pain that is associated with chewing, particularly hard foods [12]. The affected TMJ may be tender to palpation. Although pain is usually mild, it may become moderate to severe with VAS scores of 6 to 7. When the disc is displaced unilaterally, the mandible will deviate to the affected side during opening until the first click occurs—this signifies "reduction" or a return to normal relationship. Mouth opening then continues undeviated and is usually not limited; there may be some pain at maximal opening.

In patients with anterior disc displacement without reduction (ADDwoR), the condyle is unable to mechanically pass under the disc and there is no reduction. Attempts to open the mouth further induce pain located over the joint and are usually associated with no increased opening. The joint is usually very tender to palpation. Analysis of fluids obtained from the upper joint space during arthrocentesis reveals proinflammatory cytokines so that an active inflammatory process is present. Pain in ADDwoR may become quite severe with VAS of 7 to 9 and will severely affect the patient's functional capabilities. The patient will present with limited mouth opening (20–25 mm) and a sharp deviation to the affected side when asked to open the mouth. Onset is usually acute and may be preceded by a history of painful clicking (ADDwR). In some patients, there may be a slow transition from ADDwR to ADDwoR. The patient has a consistent reciprocal click but reports that at times there is restricted mouth opening of sudden onset with no click. Mandibular manipulation often relieves this situation, and they return to ADDwR; this condition is simply termed *ADDwR with intermittent locking*. If ADDwoR is untreated, there is a degree of physiological adaptation whereby the articular tissues are stretched and the patient may therefore present with minimally limited mouth opening (30–35 mm) that is accompanied by a lesser degree of deviation.

Degenerative Joint Disease

Degenerative joint disease (DJD) is a chronic noninflammatory disease that affects the articular cartilages of synovial joints, particularly load-bearing ones. Radiographic evidence of TMJ-DJD is very common (14–44%) in asymptomatic individuals, but only 8–16% will have clinically detectable disease. DJD affects women particularly after the age of 40–50 years. Pain is located in the TMJ and periauricularly and may spread to adjacent structures. Patients complain of difficulty in chewing, jaw stiffness, and tiredness. Jaw stiffness is usually more severe in the evening. The TMJ and muscles of mastication are very tender. Mouth opening is restricted and deviates to the affected side; crepitation over the affected joint is usually present. Imaging usually reveals subchondral bone sclerosis, flattening, lipping (osteophyte formation), and microcyst formation [14]. Although not inflammatory in origin, DJD rapidly induces an inflammatory response. DJD may occur following macrotrauma to the TMJ and may be aggressive in its clinical presentation.

Inflammatory Joint Disease

The TMJ may be involved in the clinical presentation of a number of systemic arthritides, including rheumatoid and psoriatic arthritis. In most cases, the patient is already diagnosed, and TMJ pain and dysfunction are signs of increasing disease severity. The management of such cases therefore involves the addition of selected TMJ-specific therapies to the existing treatment. The TMJ may be very painful, at times swollen, and will limit function. Imaging reveals joint destruction that is similar to that observed in DJD but often more rapid in its progression. Patients may complain of a lack of anterior tooth contact, a result of vertical loss of the condylar height. Laboratory findings may include a raised erythrocyte sedimentation rate, anemia, leukocytosis, and the presence of autoantibodies.

Neurovascular Orofacial Pain

Orofacial pain may occur as a result of referral in migraine or one of the trigeminal autonomic cephalalgias (TACs) [15, 16]. Some patients, however, present with primary lower facial pain accompanied by autonomic signs, nausea, and photo- or phonophobia [17–19]. These cases are often classifiable as lower cluster headache (CH) or an atypically located but otherwise classical migraine (lower half migraine).

Alternatively, patients may present with orofacial pain with neurovascular features that is neither diagnosable as lower CH nor lower half migraine, an entity we refer to as neurovascular orofacial pain (NVOP). The rationale for introducing NVOP is based on features that segregate it from other primary neurovascular headaches, such as its intraoral and perioral location (ie, second and third divisions of the trigeminal nerve). Additionally, NVOP is a term that allows us to keep an open mind as data accumulate and point toward a possible pathophysiology.

Clinical Features

Pain location is most commonly reported in the oral and perioral areas (60–70%) [9••, 17]. Pain may refer to the infraorbital or periauricular regions [9••, 17]. Moderate to strong, episodic, unilateral pain (80%) is characteristic of NVOP [17]. In 48% to 65% of cases the pain throbs, and in 35–48% it wakes the patient from sleep [9••, 17], particularly when the pain is more severe [9••]. Pain may last from minutes to hours (45–72% of cases) or continues for more than 24 h (28–55% of cases) [17]. Many NVOP patients present with daily chronic pain with a mean duration of 17 h but may be as short as 1.7 h [9••].

Pain can be accompanied by autonomic signs in 35% of cases [9••]. Specifically, tearing (10–17%), nasal congestion (7–9%), a feeling of swelling or fullness (7%) particularly in the cheek, and a complaint of excessive sweating (7%) were reported [9••, 17]. Other phenomena such as photo- or phonophobia and nausea (24–30%) are observed [9••, 17]. Patients often report dental hypersensitivity to cold food or drink.

The onset of NVOP is around 35–50 years of age, and it affects women more often than men [9••, 17]. Some patients have a positive history of migraine [20], and in 30% of cases the onset of NVOP was associated with trauma, often in cases with previous migraine [9••]. These cases exemplify the possible phenomenon of "relocation" of the original migraine to the orofacial region [9••, 20].

In an attempt to establish diagnostic criteria, we recently found that the combination of facial pain, throbbing quality, autonomic and/or systemic features (in particular nausea), and attack duration of more than 60 min gave a positive predictive value of 0.71 and a negative predictive value of 0.95 in the diagnosis of NVOP [9••].

Differential Diagnosis

Unilateral, throbbing pain that is accompanied by sensitivity to cold drinks is most often due to dental pathology. It is very common that patients with NVOP are misdiagnosed and undergo unwarranted dental treatment. As in other neurovascular pains, NVOP may change sides or jaws; very uncommon in acute dental pathology.

Classification Issues

The most prominent differentiating characteristic of NVOP is its location: oral and perioral [17] or midface [18]. TACs and migraine have been linked to the ophthalmic division of the trigeminal nerve, whilst NVOP occurs in the second and third divisions. The paucity of autonomic signs in NVOP (10%) relative to TACs (62–91%) and migraine (41–46%) may be a reflection of this basic difference. If NVOP is a migraine variant, one would expect to see longer pain attacks, a younger onset, and more photophobia, phonophobia, and nausea [9••, 17]. Similarities to CH are limited by the fact that there is an overwhelming female preponderance, and treatment response is different. NVOP responds to classical antimigraine drugs, establishing an association with migraine [17]. The dental sensitivity to cold experienced by NVOP patients may be a parallel to the allodynia reported by migraine patients. The throbbing quality, paucity of autonomic signs, long attack duration, and chronic pattern observed in NVOP are similar to that observed in hemicrania continua. However, clinically we have found these cases not to respond to indomethacin. NVOP is characterized by a chronic pain pattern, and many of these patients describe a history of episodic migraine,

suggesting a possible link to chronic migraine [17]. However, pain in NVOP is largely unilateral and throbbing. The possibility of a relocation phenomenon should therefore be considered in these cases [20].

Of particular interest is the fact that serotonin type 1D receptors ($5HT_{1D}R$) are differentially distributed in craniofacial neurones. Thus, the density of $5HT_{1D}R$ is significantly greater in tissues known to produce migraine-like pain than in structures in which triptans are ineffective [21]. Activation of $5\text{-}HT_{1B/1D}R$, by local injection of naratriptan into ventrolateral periaqueductal gray produces selective inhibition of trigeminovascular nociceptive afferent input but not facial afferents [22]. These data point to possible different pain-producing mechanisms at different locations in the craniofacial region and may be of therapeutic significance for NVOP.

Neuropathic Orofacial Pain

Neuropathic orofacial pain includes a number of clinical entities; the most common are trigeminal neuralgia, burning mouth syndrome, and traumatic neuropathies. More rarely, facial postherpetic neuralgia, central poststroke pain, glossopharyngeal neuralgia, and nervus intermedius neuralgia are encountered, but these are beyond the scope of this article.

Trigeminal Neuralgia

Trigeminal neuralgia (TN) is an excruciating, short-lasting, unilateral facial pain. The diagnostic criteria published by the International Headache Society (IHS) recognize two subsets of TN: a classical form (CTN) unrelated to pathology, and a symptomatic (or secondary) form that is related to a variety of clear pathologies, including tumors, cysts, viral infection, trauma, and systemic disease such as multiple sclerosis [23]. Most TN patients (> 85%) are diagnosed as CTN. Recent evidence suggests that most cases of CTN result from compression of the trigeminal nerve root by a vascular malformation. Unrecognized by any current classification are atypical TN cases that present with most but not all diagnostic criteria or unusual features. CTN is well known to headache specialists and, other than its unusual presentations, will not be further discussed.

Unusual Trigeminal Neuralgia Presentations

Atypical Presentations Up to 30% of TN patients report atypical features; some may present with longer TN pain attacks or with a constant background pain that makes them "atypical" [24]. Diagnostic criteria have been proposed for atypical TN, but these have not been adopted [24]. In a recent study, microvascular decompression provided absolute postoperative pain relief in CTN for 80% of cases and for 47% of atypical TN cases [25]. Long-term follow-up (>5 years) revealed excellent results in 75% of classical and 35% of atypical TN cases. Such a subclassification would thus be advantageous in clearly establishing long-term prognosis and treatment outcomes.

About 20% of TN cases present with ipsilateral autonomic signs, usually tearing and particularly when pain is severe; this may cause confusion with short-lasting TACs [26, 27].

Pretrigeminal Neuralgia An early form of TN termed *pretrigeminal neuralgia* (PTN) has been reported in 18% of TN patients and is characterized by a dull continuous pain (days to years) in one of the jaws [28]. As PTN progresses, it becomes more typical with characteristic flashes of pain. Thermal stimuli may cause triggering at a relatively higher rate, and a throbbing quality to PTN pain is sometimes present mimicking dental pathology. Indeed, these qualities combined with the success of regional anesthesia have led to misdiagnosis of PTN as pain of dental origin. PTN is, however, highly responsive to carbamazepine, and careful dental assessment should help differentiate it. Unfortunately, the lack of clear and consistent diagnostic criteria makes this a problematic entity to recognize; it is usually diagnosed when all other possibilities are exhausted or in retrospect once CTN develops.

Burning Mouth Syndrome

Burning mouth syndrome (BMS) is a poorly understood pain condition that is most probably neuropathic. Also known as *stomatodynia*, this condition is characterized by a burning mucosal pain with no significant physical signs and is common in postmenopausal women.

BMS may be subclassified into "primary" or idiopathic BMS for which a neuropathological cause is likely and cannot be attributed to any systemic or local cause and "secondary BMS" (SBMS) resulting from local or systemic pathological conditions [29]. BMS is unfortunately characterized by resistance to a wide range of treatments and is one of the most challenging management problems in the field of orofacial pain [30•].

Clinical Features

The tongue, usually the anterior two thirds, is the primary location of the burning complaint in the majority of cases. However, usually more than one site is involved; in addition to the tongue, the hard palate, lips, and gingivae are frequently involved.

Pain is most commonly described as burning or hot, and intensity varies from mild to severe [30•]. BMS is typically of spontaneous onset and lasts from months to several years [31]. The pain pattern may be irregular, but some patients may complain that pain increases toward the end of the day. Although a chronic unremitting pattern is usual, partial remission has been reported in about one half to two thirds of patients, 6 years to 7 years after onset. Spontaneous remission of BMS has been reported in 3% of patients about 5 years after onset.

More than two thirds of the patients complain of altered taste sensation (dysgeusia) accompanying the burning sensation, in many cases described as a spontaneous metallic taste. A complaint of dry mouth and true hyposalivation is common.

Athough some BMS patients may exhibit considerable emotional distress, as a group, BMS patients have shown no evidence for significant clinical depression, anxiety, and somatization but report fewer disruptions in normal activities relative to other chronic pain patients [32]. This suggests that although some BMS patients suffer significant distress, the vast majority cope better than most chronic pain patients [32].

Oral and perioral burning sensation accompanying local or systemic factors or diseases is classified as SBMS [29]. Local factors and diseases known to induce SBMS include oral candidiasis, lichen planus, and allergies. Systemic disorders known to induce SBMS include hormonal changes; deficiencies of vitamin B12, folic acid, or iron; diabetes mellitus; drugs; and autoimmune diseases [29]. Successful treatment of the primary disease will usually (but not invariably) alleviate the burning sensation in SBMS patients.

Traumatic Trigeminal Neuropathies

Traumatic trigeminal neuropathies is our preferred terminology but is essentially equivalent to the *anesthesia dolorosa* as defined by the IHS. However, because not all nerve injuries lead to anesthesia, the term *painful traumatic trigeminal neuropathy* when there is resultant pain is in our view more suitable. Some patients develop chronic pain following negligible nerve trauma such as root canal therapy or following considerable injury to nerve bundles such as in fractures of the facial skeleton [33, 34].

Following dental implant surgery and following orthognathic jaw surgery, 1–8% and 5–30% of patients, respectively, may remain with permanent sensory dysfunction, but the incidence of chronic pain is unclear [35–37]. Third molar extractions may lead to disturbed sensation in the lingual or inferior alveolar nerve for varying periods in 0.3–1% of cases [38], but follow-up failed to identify any neuropathic pain cases [39, 40]. Patient complaints of tongue dysesthesia after injury may remain in a small group of patients (0.5%). Persistent pain after successful root canal therapy was found in 3–13% of cases [34], whilst surgical root therapy resulted in chronic neuropathic pain in 5% of cases [41].

Clinical Features

Following identical injuries, onset of neuropathic pain and its characteristics vary from patient to patient. Such variability is probably due to a combination of environmental, psychosocial, and genetic factors. A further consideration is that, relative to spinal nerves, the trigeminal nerve may show subtle differences in the pathophysiological events that may lead to pain [42, 43].

Painful neuropathies may present with a clinical phenotype involving combinations of spontaneous and evoked pain and of positive (eg, dysesthesia) and negative (eg, anesthesia) symptomatology. Pain is of moderate to severe intensity and usually burning but may possess paroxysmal qualities. Pain is unilateral and may be precisely located to the dermatome of the affected nerve with demonstrable sensory dysfunction, particularly if a major nerve branch has been injured. The pain may be diffuse and spread across dermatomes, but in our experience rarely, if ever, crosses the midline. Patients may complain of a feeling of swelling, foreign body, hot or cold, local redness, or flushing.

Conclusions

The headache literature has, for many years, adopted the concept and definitions of chronic daily headache (CDH); it seems logical to apply these to chronic (>3 months) orofacial pain. Consequently COFP could be divided into daily, long (>4 h) or short-lasting, and episodic. To test this, we analyzed data collected for previously published material [9••, 44] and applied CDH definitions on a cohort of COFP patients. Significant correlations were only found between a chronic pattern and diagnosis ($\chi 2$, $P<0.0001$) [44]. This finding lends support to our view that COFP should be regarded as an umbrella term necessitating subclassification and accurate diagnosis. This situation is similar to that observed in CDH, in which each subentity requires different diagnostic definitions and treatment approaches. Precise and consistent definitions for COFP are essential to study the effects of the "chronicity" of COFP on quality of life and related issues [45].

We have continually strived to integrate the diagnosis, classification, and management of orofacial pain and headache [2••]. There is much overlap between COFP and headache. About 60% of patients seeking treatment for COFP report headaches [46]. Moreover, there is a signif-

icant relationship in the general population between the frequencies of chronic facial, head, and cervical pain [47]. There is also pathophysiological, diagnostic, and behavioral overlap between COFP and headache.

Recent publications in the headache literature also point to serious limitations of the latest edition of the IHS in classifying orofacial pain entities [9••]. Although the IHS classifies all headaches and many orofacial pain disorders, TMDs are omitted. The non-dentist is therefore often at a loss in recognizing and classifying common COFP conditions. Unfortunately, orofacial and head pain have been regarded as discrete entities; unjustified by the common underlying anatomical, neurophysiologic, and psychological factors. A unified classification system is seriously lacking and would bridge the gap between dentists and physicians.

Disclosure No potential conflicts of interest relevant to this article were reported.

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