Chapter 124

Headache Attributed to Orofacial/Temporomandibular Pathology

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Oromandibular structures may be involved in headache when pain is referred into such structures or, less commonly, when pain arises within the oromandibular structures and is referred to the head. This chapter attempts to explore the relationship between common oromandibular diseases and headache pain.

TEETH, JAWS, AND RELATED STRUCTURES

International Headache Society (IHS) code and diagnosis: 11.6 Headache attributed to disorders of teeth, jaws, and related structures
World Health Organization (WHO) code: G44.846

The most common cause of intraoral pain is dental disease. Inflammatory dental disease may be pulpal, periodontal, or a combination of both. With limited capacity for repair, inflamed or damaged pulpal tissue frequently becomes necrotic. The management of irreversible pulpitis is root canal therapy (amputation of the symptomatic pulp) or extraction. Symptoms generally resolve, but postoperative persistent pain or dysesthesia can ensue. Periodontal disorders involve the supporting structures of the teeth: the bone, periodontal ligament, and cementum.

Acute dental pain may spread unilaterally but (unlike headache) rarely crosses the midline. Convincing research on the epidemiology and qualitative characteristics of dental pain is lacking. However, acute dental pain is intense, throbbing, poorly localized, and generally provoked by stimulation of the offending tooth (58). Differentiation from comorbid headache pain does not usually pose a significant diagnostic dilemma. However, there are occasions where primary headache disorders such as migraine or cluster or paroxysmal hemicrania are located in the lower half of the face and mimic acute dental disease. Diagnostic problems are sometimes associated with a condition called "cracked tooth syndrome." A crack that extends through enamel and dentine into pulpal tissue may arise during mastication or external trauma. Pain is usually poorly localized and radiographs are not sufficiently sensitive to identify a fracture (26).

Periodontal disease is normally associated with chronic discomfort/pain. It is clearly localized to the affected teeth (unlike pulpal pain), a characteristic attributed to the proprioceptive and mechanoreceptive sensibility of the periodontium. Acute periodontal conditions can arise but they are generally limited in duration and rarely associated with, or misdiagnosed as, headache. Chronic periodontal disease may lead to pulpal involvement and this progression naturally alters symptom presentation. Although chronic periodontal disease is the most frequent cause of tooth loss in adults, the exact relationship between headache and chronic periodontal disease is unclear.

Pain associated with pulpal or periodontal disease is managed with conventional dental therapies and is not a cause of long-term disability. However, this view was challenged recently when approximately 12% of patients treated in a specialist endodontic unit reported persistent pain, despite clinical and radiographic absence of dental disease (46). This raises the question of possible change in the peripheral or central nervous system (CNS) following amputation of the pulpal tissue.

Persistent tooth site/jaw pain in the absence of dental disease, or "atypical facial pain," does not have the characteristics of the cranial neuralgias and is not associated with physical signs or a demonstrable organic cause (47,60) (Table 124-1).

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TABLE 124-1 Atypical Facial Pain

Pain present daily and present most of the day
Confined at onset to one side of face but may spread; deep and poorly localized
Not associated with sensory loss or other physical signs
Laboratory investigations including radiographs show no relevant abnormality

From ref. 47.

The diagnosis of atypical facial pain is controversial and not even recognized in the International Association for the Study of Pain (IASP) Chronic Pain Classification (64). Furthermore, data on its cause and epidemiology remains scarce and unconvincing. Suggestions that atypical facial pain is psychogenic in origin are unfounded. A hypothesis implying neurovascular mechanisms in the genesis of atypical facial pain is supported partially by the success of tricyclics and the $5HT_1$ agonist sumatriptan (24).

Atypical odontalgia (AO), which is considered a subcategory of atypical facial pain, is defined as a localized pain in a tooth or tooth site (62) and is linked to head pain (Table 124-2). Neurovascular (59) and differentiation mechanisms are some hypotheses on the pathogenesis of AO (20,36,37,59). Others have implicated the sympathetic nervous system due to the high frequency of associated trauma, the equivocal effect of somatic block, and the positive effects of sympathetic block (20,21).

TEMPOROMANDIBULAR DISORDERS

IHS code and diagnosis: 11.7 Headache or facial pain attributed to temporomandibular joint (TMJ) disorder
WHO code: G44.846
Temporomandibular disorders (TMDs) are a collection of clinical problems that involve the masticatory musculature or the TMJ and associated structures (46).
Other terms: Other terms have included Costen syndrome (7), craniomandibular disorders, oromandibular dysfunction (46), temporomandibular joint syndrome, and facial arthromyalgia (23).

TABLE 124-2 Suggested Diagnostic Criteria for Atypical Odontalgia

Major criteria
Dentoalveolar pain with no local cause
Continuous pain
Duration >4 months
Hyperesthesia
Somatosensory block equivocal
Minor criteria
Sympathetic block effective
Thermogram positive
History of trauma

EPIDEMIOLOGY

Epidemiologic data on facial pain in general are weak. Available evidence fails to differentiate headache from facial pain, which further confuses the relationship between TMD and headache. Also, studies lack conformity in diagnostic criteria, data collection, and interpretation. One Swedish study indicated that 12% of patients report pain on wide opening of the mouth, 7% reported limitation of mouth opening, 39% had noise on opening the mandible, and 2% complained of joint pain and stiffness (3). Facial pain and headache were reported by 24% of respondents (3). Generally, women report more headache, TMJ clicking, TMJ tenderness, and muscle tenderness than men (2,32,51).

GENETICS

No evidence exists to implicate genetic factors in the cause of functional disorders of the TMJs.

The TMJ is unique in its bilateral location with an upper and lower compartment separated by a fibrocartilaginous disc. This diarthrodial structure allows for both rotatory and translational movement of the mandible. This relatively simple anatomic scheme underlies masticatory movements that require complex neuromuscular control for adequate function. Although the TMJ is subject to the same pathologic disorders that may affect other synovial joints, it is unique in certain anatomic aspects. Both joints move as a functional unit and are lined by a fibrous connective tissue that is more resistant to degenerative change and has a greater capacity for repair. The masticatory system includes the articulation of the upper and lower dentition, which in itself may limit or support joint function and stability. TMDs are generally divided into those that are joint related (arthrogenous) and those that are muscular (myogenous). Clinically, the two frequently occur together but this arbitrary separation facilitates research and discussion.

PATHOPHYSIOLOGY

Sensory innervation of the TMJ is mediated through the mandibular division of the trigeminal nerve. Pain-sensitive elements within the TMJ include the joint capsule, the posterior attachment tissues, and the discal ligaments. The posterior attachment is highly innervated, richly vascularized, and frequently implicated in the pathophysiology of joint pain. In contrast, the intra-articular disc is largely devoid of neural or vascular tissue, but plays a vital role in maintaining condylar stability during mandibular movement. Trauma to the TMJ may result in acute capsulitis, but this inflammatory process tends to resolve quickly without complication. Chronic joint disorders are more frequently

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associated with painful derangement of the TMJ. Articular disc displacement frequently underlies the mechanism of joint derangement, but the cause is unclear.

The remarkable adaptive capacity of the TMJ is well documented (30,53). Failure of this mechanism may lead to tissue breakdown and disc displacement. This may be affected by age, stress, gender, systemic illness, and previous trauma. However, acute and chronic disc displacement is not always painful. Indeed, a magnetic resonance imaging (MRI) study indicated that 32% of anterior disc displacements are asymptomatic (31).

Although the cause of muscle pain is unclear, putative mechanisms underlying the transition from acute to chronic muscle pain include:

Chronic sensitization of nociceptors
Changes in innervation density
Reflex within the CNS
Neuroplasticity
Disturbance of the antinociceptive system
Psychosomatic interactions
Aggravating and perpetuating factors

Myofascial pain, which is considered a regional pain syndrome, is the most common form of TMD (40). However, the recognition of this entity as a specific disease of the masticatory system is debated. Myofascial pain is characterized by discrete tender areas (trigger points) that reproduce classical patterns of pain referral when palpated. Muscle tenderness may cause headache (19,29,34) by a mechanism that remains elusive (40). This is the case in many patients with tension-type headache whose pain frequency increases with increasing degrees of tenderness (29). However, such a relationship is not seen in migraine (28,44).

Tenderness and pain in masticatory muscles are the most frequently reported symptoms and signs of TMD. These symptoms may represent dysfunction of the trigeminal nervous system, rather than a musculoskeletal disorder. Indeed, a central origin of the myofascial trigger point syndrome is supported by referred pain, dysesthesias, hypesthesias, autonomic symptoms, and disturbance of motor function. These particular characteristics are commonly observed in the orofacial area.

Trauma, occlusal interferences, and emotional stressors are common etiologic factors that are implicated in TMD. However, theories linking emotional stress with muscle hyperactivity and facial pain have not been validated by electromyography (EMG) studies. (1,16,35,54). Also, tooth clenching or grinding (bruxism) increases in sleep disorders, which are common in chronic pain, including TMD (40). It is difficult, however, to attribute pain to bruxism because it is quite prevalent in the absence of pain (55,56) and it occurs during rapid eye movement (REM) and stage 2 sleep irrespective of occlusal status (10).

Finally, parafunctional habits have been implicated in TMD (35,57), but their relationship to headache is unknown.

Occlusal interferences remain a controversial aspect of the cause and treatment of TMD and related headache (19). A recent review indicates a minor association between occlusal factors and TMD (55,56). Also, mock equilibration of the occlusion has been shown to be equally effective to real treatment, suggesting a strong placebo effect (31). Finally, over 90% of the population has some type of malocclusion, which is believed to be a normal state (29).

CLINICAL FEATURES

Diagnostic criteria for headache or facial pain attributed to TMD (Revised International Classification of Headache Disorders [ICHD-II]) (25) are as follows:

A. Recurrent pain in one or more regions of the head and/or face fulfilling criteria C and D
B. Radiograph, MRI, and/or bone scintigraphy demonstrate TMJ disorder.
C. Evidence that pain can be attributed to the TMJ disorder, based on at least one of the following:
  1. Pain is precipitated by jaw movements and/or chewing of hard or tough food.
  2. Reduced range of or irregular jaw opening
  3. Noise from one or both TMJs during jaw movements
  4. Tenderness of the joint capsule(s) of one or both TMJs
D. Headache resolves within 3 months, and does not recur, after successful treatment of the TMJ disorder.

Pain is the most frequent presenting symptom of TMD and is usually localized in the muscles of mastication, the preauricular area, or the TMJ. The pain is typically aggravated by jaw function. Additional characteristics are limited or asymmetric jaw movements, joint noise upon movement, and jaw locking upon opening. Headache is a common associated complaint of TMD, and one study reported it to be the most common symptom (17). The association of TMD and headache is described by some to be directly related to the pathology of the TMD (17,54,61). An alternate hypothesis is that headache in the presence of TMD is an associated symptom, possibly triggered but not cause related.

PROGNOSIS

TMD is self-limiting, and patients improve irrespective of treatment received. Patients who are unresponsive to treatment should be carefully screened for a behavioral disorder (59).

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TABLE 124-3 Basic Principles of Management of Temporomandibular Disorders

Patient education and self-care
Cognitive–behavioral interventions
Pharmacologic management (e.g., analgesics, anti-inflammatory drugs, muscle relaxants, sedatives, antidepressants)
Physical therapies (e.g., posture training, stretching exercises, mobilization, physical modalities, appliance therapy, occlusal therapy)
Surgery

MANAGEMENT

Treatment outcome studies of headache related to TMD use various methods of assessing pain reduction, seldom with the same outcome variables. Also, it is often assumed that an improvement in headache following TMD treatment establishes a cause-and-effect relationship (1,16,35). Lastly, the impact of behavioral factors and CNS variables on pain outcomes is seldom addressed. Consequently, therapies that are advocated for headache with TMD remain empirically based.

General principles of management of headache with TMD include pain control or elimination, increased mandibular mobility where necessary, reduced joint loading, and resumption of normal functional activity of the mandible. These goals may be achieved through a structured, time-limited program, which addresses the physical disorder and potential perpetuating factors (Table 124-3).

Patient Education and Self Care

Explanation and reassurance for the patient is a prerequisite for satisfactory management. This approach is supported by the National Institute of Health Technology Assessment Conference Statement (1996). In susceptible patients, persistent jaw joint noise may be interpreted as a sign of disease. Understanding that joint noise may occur in otherwise healthy joints may be difficult for a patient to accept. Likewise, complaints of limited mouth opening and other signs of joint dysfunction must be interpreted and assessed in the context of patient age, gender, and general health. Expectations regarding treatment outcome must be realistic and provide reasonable goals for the patient. Simple modification of lifestyle and oral habits may be sufficient to alter symptom intensity. Ultimately, treatment outcome will be contingent on patient compliance with the program offered.

COGNITIVE–BEHAVIORAL INTERVENTION

Behavioral modification programs including relaxation training, hypnosis, or biofeedback are employed with some success. Muscle relaxation training techniques are varied, and the choice of technique will depend on the skill of the therapist and suitability of the patient. This approach has been shown to be generally effective in reducing or controlling muscle pain (13). Biofeedback training is a specific technique that depends on measurement of EMG levels from the masseter or temporalis muscles, with simultaneous auditory or visual feedback to the patient. By teaching patients to relax their jaw muscles, pain levels may decrease and range of mouth opening may increase. Ultimately, this approach will also be beneficial to patients with comorbid headache disorders.

PHARMACOLOGIC MANAGEMENT

Antiinflammatory drugs (both steroidal and nonsteroidal), muscle relaxants, and antidepressants are used to control symptoms of TMD. Level I evidence for the use of nonsteroidal antiinflammatory drugs (NSAIDs) is lacking, however. In fact, one study demonstrated that piroxicam was not superior to placebo in controlling pain of TMJ (50).

The scientific evidence for the use of sedative medications with muscle relaxant properties is equally limited. Jagger reported that diazepam was superior to placebo (27). In contrast, tricyclic antidepressants have been used extensively for chronic musculoskeletal pain, and numerous studies attest to their efficacy (14,58). In a placebo-controlled trial, 71% of patients on dothiepin were pain free after 9 weeks as compared to 46% who took placebo (14). Another study demonstrated that low-dose amitriptyline was effective in chronic facial pain. Twenty-eight patients, mostly with musculoskeletal pain, were treated in a placebo-controlled trial. There was a greater improvement with amitriptyline as compared with placebo, and the effect was independent of depression. Using the Hamilton depression score, two groups were created, one with depression and one without. Improvement was independent of depression (58).

PHYSICAL THERAPY

Physical therapy is not well researched for its role in headache with TMD. However, it is a popular approach that is considered safe, noninvasive, and reversible. Clark et al. reviewed the literature regarding the use of physical therapy for TMD and concluded that evidence for the effectiveness of this treatment modality is inconclusive (6). Treatment goals are generally based on physical rehabilitation of the joint by reducing joint inflammation, restoring joint mobility, and eliminating muscle pain through heat and stretching (6).

Occlusal appliances have been the mainstay of dental therapies for TMD since Costen first published his report on jaw joint pain in 1934 (7). Indeed, some studies have

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shown a reduction in TMD pain and an increase in function (9,59). Typically, occlusal appliances are made from rigid heat-cured acrylic that covers the occlusal surfaces of either the upper or lower dentition. The potential benefits of appliance use have been attributed to unloading of the joint surface, relaxation of masticatory muscles, and reduction or elimination of tooth clenching and grinding. These putative mechanisms of action are largely unsubstantiated in the literature.

Clinically, the most common type of occlusal device that is used is the stabilization appliance. This design allows the mandible to open and close on a normal path of movement, leaving an even $^4/_5$-mm layer of acrylic between the upper and lower teeth with the resultant suspension of the condyle in a position that is inferior and anterior to its normal location. The joint can articulate freely in this position without restriction. Most patients with TMDs can benefit almost immediately from this appliance, but its excessive use causes occlusal changes, requiring further dental treatment. Thus, patients are generally advised to use the appliances only when sleeping.

The repositioning device is another less commonly used appliance. Its function is to reposition the mandible in a protrusion direction, theoretically to facilitate recovery of the inflamed retrodiscal tissues. The awkward nature of the appliance design, coupled with the requirement to use it both during the day and at night, makes it an unpopular mode of treatment with patients. The permanent articular and occlusal changes that may accompany use of this device increase the need for extensive dental reconstruction upon resolution of the joint symptoms.

OCCLUSAL THERAPY

The association between occlusion and TMD remains controversial, as stated earlier. Malocclusions in adults are of little consequence, as skeletal adaptation has already occurred. However, certain dental abnormalities such as missing posterior, crossbite in the occlusion, and excessive vertical or horizontal discrepancy between the upper and lower anterior teeth may play a minor role in TMD (15,19,48).

SURGERY

Surgical procedures are rarely indicated for TMD. Intraarticular steroid injections are used despite lack of supporting evidence. Furthermore, condylar erosion can occur with repeated injections of corticosteroids.

Reestablishment of mandibular mobility and reduction of joint pain has been reported with irrigation of the joint with lactated Ringer solution or normal saline (43).

Simple local anaesthetic injection into the joint space may also provide short-term pain relief (11). Arthroscopy is a more invasive approach than arthrocentesis but allows for direct visualization of the intraarticular surfaces. Its usefulness in the restoration of mouth opening is documented (42,52). However, the comparative efficacy of arthroscopy to restoration of mobility is unknown. Open joint surgery is justified only in extreme circumstances where all else failed and the disability associated with joint disease impacts greatly on the patient's quality of life.

HEADACHE PROBLEMS PRESENTING IN OROMANDIBULAR STRUCTURES

The differential diagnosis of pain in the orofacial area can be complex and challenging. Headache disorders presenting in the lower half of the face can mimic odontogenic pain and musculoskeletal discomfort. Poor decision making can result in unnecessary and sometimes irreversible treatments. Headache disorders commonly seen in the orofacial region include migraine, cluster headache, and chronic paroxysmal hemicrania (CPH).

MIGRAINE

Lovshin first described migraine presenting as facial pain without headache (33). Raskin and Prusiner described ipsilateral tenderness of the carotid and suggested that dental trauma may be a precipitating factor (49). Finally, Moncada and Graff-Radford reported a case of exertional migraine presenting as a toothache that successfully responded to indomethacin (41).

CLUSTER HEADACHE

Cluster headache is a primary headache disorder, although it may coexist with migraine. Periodic migrainous neuralgia is cluster headache presenting as facial pain (5). All patients report pain in the oromandibular region, 53% have toothache, and 47% develop jaw pain (5). Bittar and Graff-Radford reported a series of 42 patients with cluster headache, of whom 65% received unnecessary dental treatments because of the facial presentation (4).

CHRONIC PAROXYSMAL HEADACHE

Patients with CPH can present with primarily tooth pain that radiates to the maxillotemporal regions of the face and that responds to indomethacin (12).

CONCLUSION

The relationship between neurovascular pain and TMD remains elusive. Differences in signs and symptoms point to different pathophysiologic mechanisms, but the frequent coexistence of these clinical disorders suggests a degree

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of physiologic overlap that requires further exploration. A better understanding of the incidence and prevalence of TMD symptoms in normal populations and studies that investigate headache disorders manifesting in the orofacial region are awaited.

The exact pathophysiology of TMD is poorly understood. It remains unclear whether these muscle and joint symptoms in the facial area represent a truly unique disorder or merely a manifestation of a more widespread musculoskeletal pain problem. Until these issues are better understood, management strategies should be conservative with minimal intervention for symptom control.

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