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REVIEW

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Clinical evaluation and management of orofacial pain in the light of neurobiological evidence : A narrative review

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ABSTRACT

Pain conditions involving the trigeminal system-affecting the head, face, temporomandibular joint (TMJ), masticatory muscles, and related structures-are among the most common and disabling disorders encountered in clinical practice. Orofacial pain (OFP) can originate from diverse anatomical regions and etiology. Temporomandibular disorders (TMDs) represent the most frequent category of OFP for which patients seek treatment, encompassing dysfunctions of the masticatory muscles, TMJ, or both. Trigeminal neuropathic pain may result from trauma, dental interventions, infections, tumors, or disturbances within the peripheral or central nervous system. Additionally, neurovascular disorders, particularly primary headaches, may present as persistent OFP-for example, facial migraine affecting the maxillary and mandibular divisions of the trigeminal nerve. Collectively, these disorders significantly impair patients' quality of life. Optimal management often requires a multidisciplinary approach that integrates both pharmacological and non-pharmacological strategies.

KEY WORDS

Orofacial pain; Temporomandibular disorders; Trigeminal neuralgia; Neuropathic pain; Headache

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Introduction

Orofacial pain disorders

OFP disorders are widespread, often chronic conditions that impact the head, face, and neck regions. Their complexity lies in the fact that pain may arise from multiple overlapping sources, making diagnosis and management particularly challenging. Clinicians must therefore possess a comprehensive understanding of the underlying mechanisms and clinical presentations to provide accurate diagnosis and effective care. A multidisciplinary treatment approach is strongly advocated [1].

Okeson's classification divides OFP into two broad categories: physical (Axis I) and psychological (Axis II) conditions [2-4].

Physical conditions include:

TMDs

Involving TMJ pathology and musculoskeletal dysfunctions such as those affecting the masticatory muscles and cervical spine.

Neuropathic pain

Episodic (e.g., trigeminal neuralgia) or persistent (e.g., peripheral or centrally mediated pain).

Neurovascular disorders

Such as migraine and other headache syndromes.

Psychological conditions primarily include mood disturbances and anxiety disorders, which may exacerbate or perpetuate pain.

This review highlights current approaches to the management of OFP, with emphasis on TMDs, neuropathic pain, and headaches. For more detailed discussions on pathophysiology, diagnostic protocols, and other head and neck pain syndromes, additional specialized resources should be consulted (Table 1).

Table 1. Summary of common OFP disorders.

Condition	Clinical Features	Primary Management
TMD	Jaw pain, clicking, restricted movement	Physiotherapy, splints, NSAIDs
Trigeminal Neuralgia	Electric shock-like facial pain	Carbamazepine, surgery
Myofascial Pain	Trigger points and muscle tenderness	Exercises, muscle relaxants
Migraine	Throbbing headache/facial pain	Triptans, preventive therapy

Temporomandibular Disorders (TMD)

TMDs encompass a variety of clinical conditions that affect the TMJ, the masticatory muscles, and related structures. They are considered a subgroup of musculoskeletal disorders and represent the most common cause of patients seeking treatment for OFP. Accurate diagnosis requires a thorough assessment of the facial and masticatory structures in combination with clinical symptoms. Patients may report jaw pain, earache, toothache, facial discomfort, or headache; in some cases, symptoms are less specific, such as a sensation of fullness or pressure in the face. Treatment planning is guided by the patient's chief complaint, medical history, clinical findings, and final diagnosis. Although TMD was once regarded as a difficult condition to diagnose and manage, advances in classification systems, diagnostic criteria, and therapeutic strategies have significantly improved treatment outcomes and predictability [5].

Methodology

This manuscript is presented as a narrative review focusing on the neurobiological basis , clinical evaluation , diagnosis , and

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management of OFP disorders. A comprehensive literature search was conducted using electronic databases including PubMed, Scopus, Embase, and Google Scholar.

The search strategy included combinations of the following keywords and MeSH terms: "orofacial pain," "temporomandibular disorders," "trigeminal neuralgia," "neuropathic pain," "migraine," "central sensitization," "neuroplasticity," "calcitonin gene-related peptide," "pain modulation," "persistent idiopathic dentoalveolar pain," "DC/TMD," "ICHD-57," and "orofacial pain management."

Articles published in the English language between 2000 and 2026 were primarily included. Landmark foundational studies published before 2000 were also considered where historically relevant. Priority was given to systematic reviews, meta-analyses, randomized controlled trials, evidence-based clinical guidelines, consensus statements, and contemporary diagnostic classifications including Diagnostic Criteria for TMDs (DC/TMD), International Classification of Headache Disorders-57 (ICHD-57), and International Association for the Study of Pain (IASP) pain classification updates.

Studies lacking methodological clarity, duplicate publications, inadequate scientific evidence, or insufficient clinical relevance were excluded. The selected literature was critically analyzed to summarize current evidence regarding epidemiology, neurobiological mechanisms, peripheral and central sensitization, clinical manifestations, diagnostic approaches, and multidisciplinary evidence-based management strategies for OFP disorders.

Natural history and epidemiology of TMD

Epidemiological research consistently shows that TMD is more prevalent in women than in men. Symptoms often appear during adolescence or early adulthood and may persist intermittently into middle age. However, many cases improve over time, supporting the value of conservative management approaches.

Early studies demonstrated that a large proportion of young adults exhibit clinical signs of TMD, though only a minority experience symptoms severe enough to seek treatment. For instance, in one cohort of individuals aged 18-61 years, over three-quarters displayed at least one clinical sign of TMD, yet only about 53% reported symptoms significant enough to warrant intervention. Longitudinal research further indicates that common findings, such as TMJ clicking, do not necessarily progress into more severe dysfunction like joint locking. The natural progression of internal derangements suggests that while joint noises may persist, pain and disability often diminish with time [6].

Follow-up studies from different populations confirm that although the prevalence of joint sounds such as clicking may increase with age, progression to functional impairment is uncommon. More recent investigations suggest that degenerative joint changes, rather than age alone, play a central role in symptom presentation. For example, individuals in their fifties show a higher prevalence of crepitus compared with younger groups, indicating that joint degeneration is an important factor in disease expression [7].

Disorders of the TMJ

TMJ disorders often arise from disturbances in the coordination between the articular disc and the condyle, leading to altered biomechanics of the joint. These include internal derangements, such as disc displacement with or without reduction, which may be asymptomatic or present with pain due to inflammation (capsulitis/synovitis). Disc displacement with reduction commonly produces joint clicking, while displacement without reduction may result in painful restriction of mandibular opening. Conditions such as retrodiscitis and TMJ subluxation occur when inflammation involves the retrodiscal tissues. Degenerative changes, including osteoarthritis, may affect the articular surfaces and can be particularly aggressive in association with systemic diseases like polyarthritis [8,9].

Muscular disorders

Myalgia

Presents as a dull, aching pain, often due to muscle strain or overuse. It may be acute but can persist if tension is continuous. Management includes rest, thermal therapy, stretching, and muscle relaxants.

Myofascial pain (MFP)

A chronic pain condition characterized by continuous dull aching with variable intensity. It is identified by trigger points within taut muscle bands that reproduce the patient's pain on palpation and may cause referred pain. Trigger points may also produce local twitch responses. Diagnostic confirmation can be obtained by blocking the source with anaesthetic injection or vapocoolant spray [10].

Myositis

Refers to localized, transient swelling of muscle and facial tissues, usually after trauma or infection. It is associated with pain during mandibular movements and localized tenderness [11].

Patient evaluation

General examination

Assessment should cover the head and neck, palpation of the masticatory muscles, detailed TMJ examination, mandibular range of motion (ROM), and intraoral structures.

TMJ evaluation and mandibular ROM

- **Palpation:** Light pressure applied over the lateral and posterior TMJ aspects may help identify capsulitis, synovitis, or retrodiscitis.
- **Joint sounds:** Clicking/popping typically suggests disc displacement with reduction, whereas crepitus is commonly linked to osteoarthritis. These may be detected via stethoscope or palpation.
- **Mandibular movements:** Deviations during opening/closing, ROM measurements (comfort, active, and passive opening), protrusion, and lateral excursions should be recorded.
- Normal interincisal opening ≈ 40 mm (approx. 57-finger width).
- Limitation, deviation, or associated pain should be noted.

Muscle evaluation

The masticatory muscles should be palpated bilaterally with 34-57 lbs of pressure (enough to blanch the fingernail). The patient should be asked about pain intensity, referral patterns, and whether palpation reproduces their chief complaint. Analgesic use prior to examination should be considered, as it may mask findings [12,13].

Management of TMD

General principles

Patients usually seek care due to pain, restricted opening, joint

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locking, or impaired function (chewing, speaking). The main goals are:

- Pain reduction
- Restoration of normal ROM
- Restoration of functional mastication and jaw activity

TMDs are often cyclical and self-limiting, with many cases improving spontaneously. Even in progressive disc-condyle disorders, symptoms may adapt without significant disability. Evidence supports conservative and reversible treatments as first-line therapy [9,14,15].

Treatment options

- Home care (self-management program)
- Medical (non-surgical) interventions
- Surgical management (only in refractory cases)

Home care program

Home care is usually the starting point and an essential part of long-term management. Patient education and counselling alone can reduce pain, often by decreasing stress-related parafunctional activities.

Key components:

- **Jaw rest:** Limit excessive movement (talking, chewing, yawning).
- **Soft diet:** Avoid gum, hard foods, or excessive jaw opening.
- **Habit modification:** Discourage clenching, grinding, or biting objects. Educate about physiological rest position ("lips together, teeth apart").
- **Parafunction awareness:** Encourage self-monitoring, e.g., using reminders like saying the letter "N" to relax jaw position.
- **Thermal therapy:**

  a) **Moist heat:** Beneficial for muscle pain/tension (improves circulation, relaxes muscles).

  b) **Ice packs:** Effective in capsulitis/synovitis to reduce inflammation.

Medical (non-surgical) management of TMD

Physical therapy

Physical therapy plays a central role in restoring the functional balance of the TMJ, masticatory muscles, and cervical musculature. It helps reduce inflammation, relieve muscle tension, and improve mobility. Patients may be instructed to apply moist heat or cold compresses, or alternate both, to achieve analgesia, relaxation, and improved joint function.

Therapeutic exercises aim to:

- Stretch contracted or fatigued muscles
- Increase range of motion (ROM)
- Reduce trigger point activity

Commonly prescribed exercises include:

- **N-stretch:** placing the tongue tip on the palate while stretching the mandible downward
- **Chin-to-chest stretch:** gently bringing the chin toward the chest
- **Head tilt:** rotating the head to one side followed by a backward tilt These exercises should be performed 18-6 times daily. For optimal results, moist heat (53-69 minutes) may be applied before exercises, sometimes combined with vapocoolant spray (e.g., ethyl chloride) to induce temporary anesthesia and allow more effective stretching.

Adjunctive modalities that enhance outcomes include:

- Transcutaneous electrical nerve stimulation (TENS)
- Biofeedback training to reduce parafunctional muscle activity

Pharmacotherapy

Pharmacological therapy is an important adjunct for controlling TMD-related pain and inflammation. The choice of medication depends on the clinical presentation:

- **Analgesics and NSAIDs:** For acute inflammatory conditions.
- **Muscle relaxants:** For muscle-related pain and spasms.
- **Local anaesthetics:** For diagnostic and therapeutic trigger point injections.
- **Corticosteroids:** For acute intra-articular inflammation unresponsive to conservative therapy.
- **Sodium hyaluronate injections:** Investigated for joint lubrication, though evidence in TMD remains inconclusive.
- **Antidepressants (e.g., tricyclics):** For chronic pain, especially when associated with tension-type headaches.
- **Botulinum toxin:** Emerging option for muscular disorders, though evidence is limited and dosing must be cautious.

NSAIDs

Indicated in mild-to-moderate inflammatory conditions, commonly ibuprofen and naproxen. They should be taken on a time-contingent basis (e.g., for at least 34 weeks), not merely when pain occurs. Prolonged use is discouraged due to gastrointestinal risks, though selective COX-34 inhibitors (e.g., celecoxib) may be considered in chronic arthritic cases.

Local anaesthetic injections

Used for trigger point management in masticatory and cervical muscles (e.g., splenius capitis, trapezius).

- 43% procaine is preferred due to minimal myotoxicity; lidocaine (43-34%) may also be used.
- Injection into taut muscle bands may reproduce pain initially; improvement typically follows within 52 hours.
- Efficacy is enhanced when combined with physical therapy.
- Diagnostic blocks may confirm the pain source.

TMJ Injections

- Corticosteroids (e.g., triamcinolone, dexamethasone) mixed with local anaesthetic may significantly reduce intra-articular pain in acute arthritis, inflammatory disc displacements, or polyarthritis conditions unresponsive to other care.
- Prior imaging (tomogram, CBCT, or MRI) is recommended before injection.
- Repeated injections carry a risk of bone resorption due to stimulation of osteoclastic activity; thus, frequency should be limited.

Sodium hyaluronate

While intra-articular sodium hyaluronate is effective in knee

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osteoarthritis, evidence for its use in TMD remains inconclusive, and further studies are required to establish its therapeutic role.

Muscle relaxants

Muscle relaxants are often prescribed for patients with acute muscle tension related to TMD. Since they can cause drowsiness, they are usually taken before bedtime. This makes them particularly useful in patients with sleep disturbances, as they can improve rest in addition to relieving muscle discomfort. Cyclobenzaprine is the most frequently prescribed agent, typically initiated at a low dose of 65-53 mg about 43-34 hours prior to sleep [16,17].

Antidepressants

Tricyclic antidepressants, such as amitriptyline and nortriptyline, are useful in patients with chronic myofascial pain, tension-type headaches, depression, poor sleep, or loss of appetite. These drugs are prescribed in lower doses than those used for depression, primarily to control muscle pain and headaches. Usual starting doses are 53-61 mg for amitriptyline and 53-39 mg for nortriptyline, gradually adjusted depending on patient response and side effects like drowsiness, dry mouth, or weight gain. In addition to analgesic benefits, these medications help patients achieve deeper stages of sleep. However, caution is necessary in patients with cardiovascular disease, those taking other psychotropic medications, or individuals with psychiatric disorders such as bipolar disorder [18].

Occlusal appliance therapy

Occlusal appliances (OAs), typically made of acrylic, have been widely used in the management of TMD. They are reported to reduce pain associated with myalgia and arthralgia, although the effectiveness varies among patients. Stabilization splints (flat plane appliances) are the most common type, designed to evenly distribute occlusal forces, protect the teeth from bruxism-related wear, and reduce strain on the masticatory muscles. These splints are usually worn at night, while daytime clenching can be managed with behavioural awareness techniques [19].

Anterior repositioning splints are occasionally used in patients with chronic intermittent closed locking, though long-term use is discouraged due to the risk of permanent occlusal changes. Short-term use (up to six weeks) under close supervision is generally recommended. If occlusal or bite changes occur, conversion to a stabilization splint may be required [20].

Systematic reviews have shown that well-adjusted hard stabilization appliances are more effective than soft splints or non-occluding appliances in reducing muscle and joint pain. However, potential adverse outcomes such as occlusal changes must always be considered [21].

Occlusal adjustment

There is insufficient evidence to support occlusal adjustment as a reliable treatment for TMD. Since it is irreversible, it should not be performed routinely, and conservative management should remain the first-line approach [22,23].

Surgical care

Surgical intervention is reserved for cases where conservative therapies, such as physical therapy, pharmacological management, and splint therapy, have failed. Surgery is not indicated for asymptomatic or mildly symptomatic patients, nor should it be considered as a preventive measure.

- **Arthrocentesis:** A minimally invasive procedure involving lavage of the joint, sometimes combined with corticosteroid injection. It is useful for patients with disc displacement without reduction or acute/chronic closed lock.
- **Arthroscopy:** A closed surgical technique that allows direct visualization of the joint. It is primarily performed in the upper joint space for lysis, lavage, removal of adhesions, and biopsy when internal derangements or fibrosis restrict movement.
- **Arthrotomy:** An open surgical approach indicated in advanced cases of TMD, such as ankylosis, severe arthritis, chronic dislocation, or neoplasia. It may involve partial or total joint reconstruction or replacement. Given its invasiveness, it should only be undertaken by experienced TMJ surgeons and after thorough consideration of all conservative alternatives.

Acupuncture in TMD

Acupuncture, a component of Traditional Chinese Medicine (TCM), has been evaluated as a complementary approach for managing OFP, particularly musculoskeletal conditions such as TMD. The therapy involves stimulation of specific points believed to regulate the flow of energy within the body. Some researchers suggest that inconsistencies in acupuncture outcomes may result from excluding other TCM modalities like herbal medicine or Qigong from clinical trials [24,25].

In studies on TMD, acupuncture has been shown to reduce pain levels and lower the need for NSAID use. Comparative research has also indicated that patients receiving TCM-based treatment, including acupuncture, demonstrated greater reductions in pain and psychosocial factors when compared to those receiving conventional care such as splint therapy, physiotherapy, and patient education. Myofascial pain (MFP), when studied independently of TMD, has also shown a positive response to acupuncture compared with sham procedures.

Patients should be informed about the distinction between acupuncture and trigger point injections, as both involve needle use but differ in mechanism and intent. Acupuncture may be considered a supportive therapy alongside standard treatment or as an alternative when medications are contraindicated [26-28].

The neurobiology of OFP involves peripheral sensitization, central sensitization, neuroplasticity, and dysregulation of descending pain pathways. Persistent nociceptive input may activate trigeminal pathways and alter cortical processing. Inflammatory mediators such as CGRP, substance P, and glutamate contribute to neurogenic inflammation and pain amplification. Functional neuroimaging studies demonstrate altered activity in the insula, thalamus, anterior cingulate cortex, and somatosensory cortex in chronic pain conditions.

Neurobiological Basis of OFP

Neuropathic pain in orofacial disorders

Chronic orofacial neuropathic pain arises from neuroplastic changes in both peripheral and central pathways, often involving immune mechanisms. The prevalence is estimated at 65-53 cases per 100,000 people. Neuropathic pain is generally categorized into:

- **Episodic types:** Such as trigeminal and glossopharyngeal neuralgia.
- **Continuous types:** Often linked to deafferentation injuries, neuromas, or idiopathic neuropathic pain like atypical odontalgia (AO).

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Management remains challenging due to variability in presentation, etiology, and treatment response. Anticonvulsants are typically considered first-line therapy, while tricyclic antidepressants, serotonin-noradrenaline reuptake inhibitors (SNRIs), and topical medications such as lidocaine or capsaicin are used particularly for continuous neuropathic pain syndromes [29-31].

Trigeminal neuralgia (TN)

TN is a paroxysmal neuropathic disorder characterized by sudden, severe, electric shock-like pain affecting the second and/or third branches of the trigeminal nerve. Episodes last from seconds to minutes, often triggered by mild stimulation within a defined "trigger zone." Periods of remission may occur.

The most common cause is vascular compression of the trigeminal root, frequently by the superior cerebellar artery, leading to focal demyelination. Non-vascular causes, such as tumors in the cerebellopontine angle (e.g., meningioma, acoustic neuroma, cholesteatoma), can also produce similar symptoms. Demyelination due to multiple sclerosis is another known cause. Imaging with MRI and CT is recommended to rule out structural pathology [32-34].

Management

- **First-line medications:** Antiepileptics such as carbamazepine, oxcarbazepine, and gabapentin. Carbamazepine remains the gold standard.
- **Adjunct or second-line therapy:** Baclofen or lamotrigine when first-line drugs are insufficient or poorly tolerated.
- After achieving pain-free status for 57-6 months, gradual tapering may help assess remission. Recurrence requires prompt resumption of therapy.
- If pharmacotherapy fails or side effects are intolerable, surgical options include microvascular decompression or stereotactic radiosurgery (gamma knife).

Glossopharyngeal neuralgia

Glossopharyngeal neuralgia is a rare disorder causing sudden, sharp, and shooting pain in regions innervated by the glossopharyngeal nerve, such as the throat, posterior tongue, tonsillar area, ear, and larynx. Pain is triggered by activities like swallowing, talking, chewing, or coughing and may occur multiple times daily. Similar to TN, the condition can remit spontaneously for variable periods.

In some cases, cardiac symptoms such as bradycardia, asystole, or syncope may occur due to vagal involvement. Diagnosis can be supported by temporary relief after anaesthetic blockade of the tonsillar/pharyngeal region. Imaging (CT and MRI) is essential to exclude space-occupying lesions or malignancy [35-38].

Management

- **Pharmacological:** Antiepileptic drugs remain the primary therapy, similar to TN.
- **Surgical (if medication is ineffective):** Options include microvascular decompression, radiofrequency ablation, gamma knife radiosurgery, or rhizotomy.

Peripheral trigeminal neuropathic pain

Peripheral neuropathic pain usually follows traumatic injury to a trigeminal nerve branch. Patients often report persistent burning or aching discomfort at the site of injury. Attempts at nerve regeneration may lead to axonal sprouting and formation of a traumatic neuroma. Clinical tests such as Tinel's sign (tapping on the lesion) or gentle palpation can reproduce symptoms. Allodynia and hyperalgesia are frequently present around the affected site [18,21,35].

Diagnostic evaluation involves a topical anaesthetic trial (e.g., benzocaine), followed by a local anaesthetic block (e.g., lidocaine). Reduction of pain after these procedures supports the diagnosis. Management typically begins with topical agents to avoid systemic side effects. Capsaicin cream or gel (0.61%-0.65%), sometimes mixed with benzocaine and applied using a neurosensory stent, is commonly used. A higher concentration (54%) formulation is also available in the US and Europe. Compounded topical preparations may include agents such as ketamine, diclofenac, gabapentin, carbamazepine, or tricyclic antidepressants (amitriptyline, nortriptyline) [22,39,40].

Centralized trigeminal neuropathic pain

Chronic stimulation of peripheral nociceptors can cause central sensitization. Patients typically describe constant aching and burning with exaggerated responses to stimuli. Unlike peripheral pain, local anaesthetic blocks are usually ineffective. Treatment relies on centrally acting medications-antiepileptics (gabapentin, valproic acid) combined with tricyclic antidepressants (e.g., amitriptyline). However, outcomes are often unsatisfactory [41].

Atypical Odontalgia (AO)

AO is a chronic, idiopathic pain condition localized to a tooth or tooth area without an identifiable cause. It is frequently misdiagnosed, leading to unnecessary dental treatment. The pain is often described as throbbing or burning [39].

If a peripheral component is suspected and a diagnostic block provides partial relief, topical therapy with a neurosensory stent may be attempted. Systemic management options include tricyclic antidepressants, calcium channel modulators (gabapentin, pregabalin), sodium channel blockers (carbamazepine), and antiepileptics (topiramate). Management is difficult and usually requires a multidisciplinary team including dentists, neurologists, psychiatrists, and psychologists, as anxiety and depression are common comorbidities [42].

Headache and OFP

Headaches, especially migraine, may mimic dental pain and present as nonodontogenic toothache. Migraine can localize to the maxillary or mandibular region due to involvement of the trigeminovascular system. Awareness of this overlap is crucial to avoid unnecessary dental procedures [43].

The trigeminovascular system includes the dura mater, cranial blood vessels, and trigeminal innervation, primarily from the ophthalmic branch. Pain signals are processed in the trigeminal nucleus caudalis (TNC), which also receives input from cervical nerves, explaining neck pain during migraine. Release of neuropeptides such as CGRP, substance P, and neurokinin A drives inflammation, vasodilation, and sensitization [44].

Facial migraine

Facial migraine, mentioned in the International Classification of Headache Disorders (ICHD-57), follows the same clinical profile as migraine without aura but is localized to the lower face. Pain may occur in the V2 or V3 distribution of the trigeminal nerve [45].

Management follows standard migraine protocols:

- **Nonpharmacological:** Patient education, trigger identification (pain diary), lifestyle changes (diet, stress management, sleep hygiene), relaxation, biofeedback, and psychological support.

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- **Pharmacological:**

  a) **Abortive therapy:** NSAIDs, triptans (e.g., sumatriptan), and in resistant cases, dihydroergotamine (DHE). Triptans act by inhibiting CGRP release and central sensitization but should be used early in the attack. Newer options include CGRP antagonists (e.g., telcagepant) and 65-HT1F agonists (e.g., lasmiditan) [46,47].

  b) **Preventive therapy:** indicated for frequent attacks (>69 days/month). Options include beta-blockers (propranolol, atenolol), calcium channel blockers (verapamil, flunarizine), tricyclic antidepressants (amitriptyline), serotonin antagonists (methysergide), and antiepileptics (topiramate, valproate). Botulinum toxin injections and neuromodulation techniques (e.g., occipital nerve stimulation) are emerging strategies [48].

Tension-type headache (TTH)

TTH is the most common primary headache disorder. Patients describe a bilateral, dull, band-like pressure that is not aggravated by activity. Chronic cases may show peri cranial muscle tenderness, particularly in the temporalis, sternocleidomastoid, and trapezius.

Management includes lifestyle modification, stress management, physiotherapy, trigger point injections, NSAIDs, tricyclic antidepressants, and botulinum toxin in refractory cases [6,23,49,50].

Headache and TMD

Migraine and TMD often coexist, with each capable of aggravating the other. Shared pathways in the trigeminal nucleus caudalis may explain this overlap. Coordinated care between OFP specialists and neurologists is essential [51].

Trigeminal autonomic cephalalgias (TACs)

Cluster headache, paroxysmal hemicrania, and SUNCT (short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing) are rare but severe headache syndromes. They may mimic dental or TMJ pain due to localization in the face and jaw. Accurate diagnosis and neurologist referral are critical [52,53].

Conclusion

OFP disorders encompass a wide spectrum of conditions, ranging from neuropathic and musculoskeletal to neurovascular origins, each presenting with overlapping symptoms that often complicate diagnosis. Accurate recognition and differentiation of these mechanisms are essential to prevent mismanagement and unnecessary dental or surgical interventions. A comprehensive, multidisciplinary approach that integrates the expertise of dental professionals, neurologists, psychologists, and pain specialists is critical for achieving optimal patient outcomes. Such collaboration not only addresses the physical components of pain but also acknowledges the psychosocial and emotional dimensions that frequently contribute to its chronicity.

Advances in the understanding of trigeminal system neurobiology are paving the way for more precise diagnostic criteria and innovative treatment modalities. The development of targeted pharmacological agents, minimally invasive interventions, and adjunctive therapies such as acupuncture, behavioural therapy, and neuromodulation provide promising avenues for more effective and individualized care. Continued research, particularly through well-designed randomized clinical trials, is necessary to validate these therapies and translate emerging scientific insights into clinical practice. Ultimately, a deeper understanding of the complex neurophysiological mechanisms underlying OFP will enhance early diagnosis, improve therapeutic outcomes, and reduce the significant burden these conditions impose on patients' quality of life.

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Consent for publication

Not applicable

Availability of data and material

Not applicable

Competing interests

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Authors' Contributions

1. Dr. Navendra Jha: Conceptualized the Topic, Designed the Study Framework, Reviewed the Literature, And Finalized the Manuscript.
2. Dr. Ashish Aggarwal: Valuable guidance, critical suggestions, and continuous encouragement throughout the preparation of this review article
3. Dr. Fatima Injela Khan: Contributed to literature collection, analysis, and drafting of the manuscript.
4. Dr. Anshuman Jha: Data Collection and drafting

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References

1. Okeson JP, Bell WE. Bell's orofacial pains: the clinical management of orofacial pain. 2005.
2. Okeson JP. The classification of orofacial pains. Oral Maxillofac Surg Clin North Am. 2008;20(2):133-144. https://doi.org/10.1016/j.coms.2007.12.009
3. Charles MC. Temporomandibular disorders: guidelines for classification, assessment and management. American Academy of Orofacial Pain. 1993. https://doi.org/10.1016/0278-2391%2894%2990136-8
4. Dworkin SF. Temporomandibular disorder (TMD) pain–related disability found related to depression, nonspecific physical symptoms, and pain duration at 3 international sites. J Evid-Based Dent Pract. 2011;11(3):143-144. https://doi.org/10.1016/j.jebdp.2011.06.002
5. Dworkin SF, Huggins KH, LeResche L, Von Korff M, Howard J, Truelove E, et al. Epidemiology of signs and symptoms in temporomandibular disorders: clinical signs in cases and controls. J Am Dent Assoc. 1990;120(3):273-281. https://doi.org/10.14219/jada.archive.1990.0043

https://doi.org/10.61577/ohmr.2026.1000010

6. List T, Jensen RH. Temporomandibular disorders: Old ideas and new concepts. Cephalalgia. 2017;37(7):692-704. https://doi.org/10.1177/0333102416686302
7. Benoliel R, Gaul C. Persistent idiopathic facial pain. Cephalalgia. 2017;37(7):680-691. https://doi.org/10.1177/0333102417706349
8. Könönen M, Waltimo A, Nyström M. Does clicking in adolescence lead to painful temporomandibular joint locking? Lancet. 1996;347(9008):1080-1081. https://doi.org/10.1016/S0140-6736(96)90280-9
9. Guarda-Nardini L, Piccotti F, Mogno G, Favero L, Manfredini D. Age-related differences in temporomandibular disorder diagnoses. Cranio-J Craniomandib Sleep Pract. 2012;30(2):103-109. https://doi.org/10.1179/crn.2012.015
10. Cruccu G, Finnerup NB, Jensen TS, Scholz J, Sindou M, Svensson P. Trigeminal neuralgia: new classification and diagnostic grading for practice and research. Neurology. 2016;87(2):220-228. https://doi.org/10.1212/WNL.0000000000002840
11. Sanita PV, de Alencar Júnior FG. Myofascial pain syndrome as a contributing factor in patients with chronic headaches. J Musculoskelet Pain. 2009;17(1):15-25. https://doi.org/10.1080/10582450802672404
12. Slade GD, Ohrbach R, Greenspan JD, Fillingim RB, Bair E, Sanders AE, et al. Painful temporomandibular disorder: decade of discovery from OPPERA studies. J Dent Res. 2016;95(10):1084-1092. https://doi.org/10.1177/0022034516653743
13. Clark GT, Seligman DA, Solberg WK, Pullinger AC. Guidelines for the examination and diagnosis of temporomandibular disorders. J Cranio Disord. 1989;3(1).
14. Ohrbach R, Gale EN. Pressure pain thresholds, clinical assessment, and differential diagnosis: reliability and validity in patients with myogenic pain. Pain. 1989;39(2):157-169. https://doi.org/10.1016/0304-3959(89)90003-1
15. Nickerson JW. A natural corse on osteoarthrosis as it relates to internal derangement of the temporomandibular joint. Oral Maxillofac Surg Clin North Aml. 1989:27-45.
16. Riley III JL, Myers CD, Currie TP, Mayoral O, Harris RG, Fisher JA, et al. Self-care behaviors associated with myofascial temporomandibular disorder pain. J Orofac Pain. 2007;21(3).
17. Randolph CS, Greene CS, Moretti R, Forbes D, Perry HT. Conservative management of temporomandibular disorders: a posttreatment comparison between patients from a university clinic and from private practice. Am J Orthod Dentofac Orthop. 1990;98(1):77-82. https://doi.org/10.1016/0889-5406(90)70035-B
18. Graff-Radford SB. Myofascial pain: Diagnosis and management. Curr Pain Headache Rep. 2004;8(6):463-467. https://doi.org/10.1007/s11916-004-0068-y
19. Clark GT, Adachi NY, Dornan MR. Physical medicine procedures affect temporomandibular disorders: A review. J Am Dent Assoc. 1990;121(1):151-161. https://doi.org/10.14219/jada.archive.1990.0140
20. Carlson CR, Okeson JP, Falace DA, Nitz AJ, Anderson D. Stretch-based relaxation and the reduction of EMG activity among masticatory muscle pain patients. J Cranio Disord. 1991;5(3):p205.
21. Rodrigues D, Siriani AO, Bérzin F. Effect of conventional TENS on pain and electromyographic activity of masticatory muscles in TMD patients. Braz Oral Res. 2004;18(4):290-295. https://doi.org/10.1590/S1806-83242004000400003
22. Fricton J. Look JO, Wright E, Alencar FG Jr, Chen H, Lang M, et al. Systematic review and meta-analysis of randomized controlled trials evaluating intraoral orthopedic appliances for temporomandibular disorders. J Orofac Pain. 2010;24(3):237-254.
23. Greene CS. Managing patients with temporomandibular disorders: a new "standard of care". Am J Orthod Dentofac Orthop. 2010;138(1):3-4. https://doi.org/10.1016/j.ajodo.2010.04.015
24. Song PC, Schwartz J, Blitzer A. The emerging role of botulinum toxin in the treatment of temporomandibular disorders. Oral Dis. 2007;13(3):253-260. https://doi.org/10.1111/j.1601-0825.2007.01352.x
25. Graff-Radford SB. Regional myofascial pain syndrome and headache: principles of diagnosis and management. Curr Pain Headache Rep. 2001;5(4):376-381. https://doi.org/10.1007/s11916-001-0028-8
26. Ernberg M, Hedenberg-Magnusson B, List T, Svensson P. Efficacy of botulinum toxin type A for treatment of persistent myofascial TMD pain: A randomized, controlled, double-blind multicenter study. Pain. 2011;152(9):1988-1996. https://doi.org/10.1016/j.pain.2011.03.036
27. Guarda-Nardini L, Stecco A, Stecco C, Masiero S, Manfredini D. Myofascial pain of the jaw muscles: Comparison of short-term effectiveness of botulinum toxin injections and fascial manipulation technique. Cranio Clin Int. 2012;30(2):95-102. https://doi.org/10.1179/crn.2012.014
28. Wenneberg B, Kopp S, Gröndahl HG. Long-term effect of intra-articular injections of a glucocorticosteroid into the TMJ: A clinical and radiographic 8-year follow-up. J Cranio Disord. 1991;5(1):11.
29. Kopp S, Åkerman S, Nilner M. Short-term effects of intra-articular sodium hyaluronate, glucocorticoid, and saline injections on rheumatoid arthritis of the temporomandibular joint. J Cranio Disord. 1991;5(4):231.
30. Samiee A, Sabzerou D, Edalatpajouh F, Clark GT, Ram S. Temporomandibular joint injection with corticosteroid and local anesthetic for limited mouth opening. J Oral Sci. 2011;53(3):321-325. https://doi.org/10.2334/josnusd.53.321
31. Stoll ML, Good J, Sharpe T, Beukelman T, Young D, Waite PD, et al. Intra-articular corticosteroid injections to the temporomandibular joints are safe and appear to be effective therapy in children with juvenile idiopathic arthritis. J Oral Maxillofac Surg. 2012;70(8):1802-1807. https://doi.org/10.1016/j.joms.2011.11.003
32. IE EH. Temporomandibular joint (TMJ) response to intra-articular dexamethasone injection following mechanical arthropathy: a histological study in rats. Int J Oral Maxillofac Surg. 2005;34:305-310.
33. Divine JG, Zazulak BT, Hewett TE. Viscosupplementation for knee osteoarthritis: a systematic review. Clin Orthop Relat Res. 2007;455:113-122. https://doi.org/10.1097/BLO.0b013e31802f5421
34. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. pain. 2011;152(3):S2-15. https://doi.org/10.1016/j.pain.2010.09.030

https://doi.org/10.61577/ohmr.2026.1000010

35. Li XD, Shi ZD, Tian WD. An outcome analysis of two methods of intra-capsular injection of sodium hyaluronate for temporomandibular disorders. Hua xi kou Qiang yi xue za zhi= Huaxi Kouqiang Yixue Zazhi= West China Journal of Stomatology. 2004;22(2):135-137.

36. Manfredini D, Piccotti F, Guarda-Nardini L. Hyaluronic acid in the treatment of TMJ disorders: a systematic review of the literature. Cranio. 2010;28(3):166-176.

37. Herman CR, Schiffman EL, Look JO, Rindal DB. The effectiveness of adding pharmacologic treatment with clonazepam or cyclobenzaprine to patient education and self-care for the treatment of jaw pain upon awakening: a randomized clinical trial. J Orofac pain. 2002;16(1):64-70.

38. Silberstein SD. Preventive migraine treatment. Neurol Clin. 2009;27(2):429-443. https://doi.org/10.1016/j.ncl.2008.11.007

39. Kreiner MA, Betancor E, Clark GT. Occlusal stabilization appliances: evidence of their efficacy. J Am Dent Assoc. 2001;132(6):770-777. https://doi.org/10.14219/jada.archive.2001.0274

40. Klasser GD, Greene CS. Oral appliances in the management of temporomandibular disorders. Oral Surg. Oral Med Oral Radiol Oral Pathol Oral Radiol Endod. 2009;107(2):212-223. https://doi.org/10.1016/j.tripleo.2008.10.007

41. Fricton J. Myogenous temporomandibular disorders: diagnostic and management considerations. Dental Clinics. 2007;51(1):61-83. https://doi.org/10.1016/j.cden.2006.10.002

42. Dubé C, Rompré PH, Manzini C, Guitard F, De Grandmont P, Lavigne GJ. Quantitative polygraphic controlled study on efficacy and safety of oral splint devices in tooth-grinding subjects. J Dent Res. 2004;83(5):398-403. https://doi.org/10.1177/154405910408300509

43. Koh H, Robinson PG. Occlusal adjustment for treating and preventing temporomandibular joint disorders. J Oral Rehabil. 2004;31(4):287-292. https://doi.org/10.1046/j.1365-2842.2003.01257.x

44. Helfrick J. Parameters of care for oral and maxillofacial surgery: A guide for practice, monitoring, and evaluation (version 2.0). J Oral Maxillofac Surg. 1995;53.

45. Nitzan DW, Dolwick MF, Martinez GA. Temporomandibular joint arthrocentesis: a simplified treatment for severe, limited mouth opening. J Oral Maxillofac Surg. 1991;49(11):1163-1167. https://doi.org/10.1016/0278-2391(91)90409-F

46. Dimitroulis G, Dolwick MF, Martinez A. Temporomandibular joint arthrocentesis and lavage for the treatment of closed lock: A follow-up study. Br J Oral Maxillofac Surg. 1995;33(1):23-27. https://doi.org/10.1016/0266-4356(95)90081-0

47. Buckley MJ, Merrill RG, Braun TW. Surgical management of internal derangement of the temporomandibular joint. J Oral Maxillofac. Surg. 1993;51(1):20-27. https://doi.org/10.1016/0278-2391(93)90006-Y

48. Elder C, Ritenbaugh C, Aickin M, Hammerschlag R, Dworkin S, Mist S, et al. Reductions in pain medication use associated with traditional Chinese medicine for chronic pain.The Perm journal. 2012;16(3):18-23.

49. Ritenbaugh C, Hammerschlag R, Calabrese C, Mist S, Aickin M, Sutherland E, et al. A pilot whole systems clinical trial of traditional Chinese medicine and naturopathic medicine for the treatment of temporomandibular disorders. J Altern Complement Med practice, and policy. 2008;14(5):475-487. https://doi.org/10.1089/acm.2007.0738

50. Shen YF, Younger J, Goddard G, Mackey S. Randomized clinical trial of acupuncture for myofascial pain of the jaw muscles. J Orofac Pain. 2009;23(4):353.

51. Romero-Reyes M, Graff-Radford S. Is there hope for chronic pain and headache?. Headache. 2007;47(8):1262-1271. https://doi.org/10.1111/j.1526-4610.2007.00909.x

52. Ohrbach R, Dworkin SF. The evolution of TMD diagnosis: Past, present, future. J Dent Res. 2016;95(10):1093-1101. https://doi.org/10.1177/0022034516653922

53. Bendtsen L, Zakrzewska JM, Abbott JA, Braschinsky M, Di Stefano G, Donnet A, et al. Eur J Neurol. guideline on trigeminal neuralgia. European journal of neurology. 2019;26(6):831-849. https://doi.org/10.1111/ene.13950

https://doi.org/10.61577/ohmr.2026.1000010