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OROFACIAL PAIN

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ABSTRACT

Diagnosing and treating orofacial pain can be difficult due to its complex history, underlying mechanisms, and associated psychosocial factors like anxiety and depression. Conditions such as burning mouth syndrome (BMS), persistent idiopathic facial pain (PIFP), atypical odontalgia (AO), and trigeminal neuralgia (TN) require early identification by primary care practitioners and prompt referral to specialist care [1]. Neurovascular disorders, including primary headaches, can present as chronic orofacial pain, such as facial migraine, where the pain is localized to the second and third divisions of the trigeminal nerve. These conditions can severely impact the quality of life of those affected. An effective approach to managing orofacial pain disorders should involve a multidisciplinary strategy, combining both pharmacological and non-pharmacological treatments [2].

KEYWORDS Orofacial pain, Trigeminal neuralgia, Myofascial pain dysfunction syndrome, Nervous intermedius neuralgia, TMJ ankylosis.

INTRODUCTION

Orofacial pain disorders are prevalent and debilitating, impacting the head, face, and neck. Their management is challenging for clinicians because of the intricate nature of the orofacial region, where pain may stem from multiple origins [2]. Adopting a biopsychosocial approach, which combines physical therapies, medication, and psychological support, can effectively manage facial pain and minimize its adverse effects on quality of life and daily activities. Implementing early diagnosis and a holistic management approach is crucial to minimizing the negative psychosocial effects of orofacial pain while ensuring that care remains both effective and efficient [1].

PAIN

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage. Pain is a subjective phenomenon.[3]

OROFACIAL PAIN

Pain which can be associated with hard and soft tissues of head , face , oral cavity , or malfunction of nervous system[4].

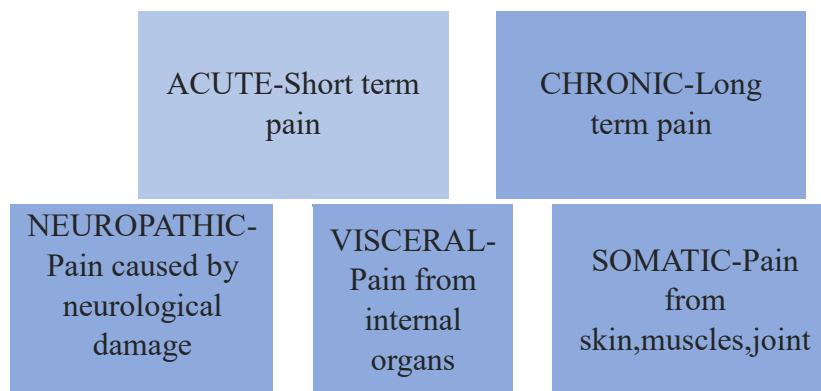
TYPES A.ACUTE OFP

It is primarily associated with teeth and their supporting structures. Most frequently, dental pain is due to dental caries , although a broken filling or tooth abrasion may also cause dental sensitivity. Other oral pains are usually periodontal or gingival in origin.

B. CHRONIC OFP

It is a term used to describe painful regional syndromes with a chronic,

unremitting , pattern

TYPES OF PAIN

Clinically COFP may be subdivided into three main symptomatic classes

1.MUSCULOSKELETAL

☐ Temporomandibular disorders.

2.NEUROPATHIC

- Trigeminal neuralgia
- Glossopharyngeal neuralgia ☐ Geniculate neuralgia

3.NEUROVASCULAR

- Cluster headache
- Migraine
- Paroxysmal hemicrania
- Cranial arteritis
- Tension-type headache

POSSIBLE CAUSES OF FACIAL PAIN:

- 1.Diseases of teeth
- 2.Diseases of periodontium
3. Diseases of oral mucosa
4. Diseases of jaws
5. Diseases of antrum
6. Diseases of salivary glands
7. Diseases of TMJ
8. Diseases of ears
9. Diseases of eyes
- 10.Diseases of sinuses and nasopharynx

DIFFERENTIAL DIAGNOSIS OF OROFACIAL PAIN 1.Intracranial pain disorders

- Neoplasm , aneurysm
- Abscess
- Hemorrhage
- Hematoma
- Edema

2. Neurovascular disorders

- Migraine
- Migraine variants
- Cluster headache
- Paroxysmal hemicranias
- Cranial arteritis
- Tension type-headache

3. Neurogenic pain disorders

- Paroxysmal neuralgias (trigeminal, glossopharyngeal, nervus intermedius)

4. Intraoral pain disorders

- Disorders of pulp
- Disorders of mucogingival tissues
- Disease of tongue
- Diseases of periodontium

5. Temporomandibular disorders

- Diseases of Masticatory muscle
- Diseases of TMJ associated structure

6. Associated structures

- Diseases of Ears, eyes, nose, paranasal sinus, throat, lymph node, salivary gland, neck.

A. MYOFACIAL PAIN DYSFUNCTION SYNDROME

Definition: Myofascial trigger point pain is a regional myogenous pain condition characterized by localized areas of firm, hypersensitive muscle bands known as trigger points. A key feature of this myalgic disorder is the presence of central excitatory effects. Referred pain is common and often mimics the symptoms of a tension-type headache [5].

ETIOLOGY

- Occlusal disturbances
- Intracapsular disorders
- Emotional stress
- Direct or indirect trauma
- Spine pathology
- Stress and strain
- Parafunctional habits like bruxism, clenching

PATHOPHYSIOLOGY

- Injury to TYPE-I muscle fibre
- Metabolic distress at motor end plate
- Activation of muscle nociceptors
- Transmission of pain to CNS

CLINICAL FEATURES

- Preauricular Pain
- Jaw tenderness on function
- TMJ sounds
- Restricted mouth opening
- Facial pain
- Headache [6].

TRIGGER POINTS

These are localized tender areas within taut bands of skeletal muscle and stimulated by macro and micro-traumatic episodes, they refer a characteristic pain pattern to distant group of muscles.

Palpation of trigger points give a **positive jump sign**.

DIAGNOSTIC CRITERIA**LASKIN FOUR CARDINAL SIGNS**

1. Unilateral pain
2. Muscle tenderness
3. Clicking
4. Limited jaw function

NEGATIVE CHARACTERISTICS

1. Absence of clinical radiographic or biomechanical evidence of changes in TMJ
2. Lack of tenderness in TMJ area when palpated through external auditory meatus[5].

DIFFERENTIAL DIAGNOSIS

1. TMJ disorders with disc displacement
2. Odontogenic pain
3. Migraine
4. Trigeminal neuralgia
5. Sinusitis
6. Referred pain from neck
7. Fibromyalgia

DIAGNOSTIC METHODS

1. TMJ arthrography
2. Computed radiography
3. CT scan
4. MRI scan
5. Bone scintigraphy-Nuclear imaging
6. Single photon emission computerized tomography(SPECT)[5].

MANAGEMENT**MEDICATION:** NSAID's

- Aspirin(2 tabs 0.3 to 0.6 gram/4 hour)
- Piroxicam(Cap. 10 to 20 mg /once daily) □ Ibuprofen(200 to 600 mg /3 times a day)

MUSCLE RELAXANT

Methocarbamol (1500mg /4 times a day for 2-3 days)

ANTIDEPRESSANT

Diazepam(5 to 10 mg /2-3 times a day)

Amytriptyline(25 mg /3-4 times a day)

NON INVASIVE METHODS OF TRIGGER POINT INACTIVATION

- Massages
- Acupressure therapy

Relaxation measures: Moist heat application

Ice packs

Fluori-methane sprays

Diathermy

ELECTRICAL CURRENT STIMULATION

- Electro-acupuncture
- Transcutaneous electrical nerve stimulation
- Direct current application to muscle and trigger points[6].

B.HEADACHES

Headaches can be divided into 3 types

PRIMARY HEADACHE

A primary headache occurs without any identifiable underlying cause[8]. It includes:

- Migraine
- Tension type headache
- Cluster headache
- Hemicranias continua

SECONDARY HEADACHE

secondary headache is caused by another condition that leads to traction or inflammation of pain-sensitive structures[9].It includes:

- Post traumatic
- Vascular disease
- CNS infection
- Hypertension , hypoxia

TYPE 3: Cranial neuralgias and central or peripheral causes of facial pain

PRIMARY HEADACHE DISORDERS

Primary headache disorders make up the majority of headache conditions, with migraine and tension-type headache (TTH) being the most common.

I.MIGRAINE

Migraine is the second most frequent type of headache, typically characterized by recurrent throbbing or pulsating pain that is moderate to severe, often affects one side of the head, and lasts 4–72 hours, with symptom-free periods between episodes (episodic).

CLINICAL FEATURES

- Headache
- nausea
- vomiting
- sensitivity to light ,sound, smell
- Approximately one third of patients experience an AURA which is progressing focal neurological symptom with a duration of 5-60 minutes ☐ Unilateral sensory disturbance ☐ Dysphasia[7].

DIAGNOSTIC CRITERIA FOR MIGRAINE Without Aura

- At least five headaches lasting 4–72 hours.
- Throbbing or pulsating pain.
- Unilateral location of pain.
- Intense severity.
- Worsening of the headache with routine physical activities.

With Aura

- At least five headaches, with at least two episodes accompanied by an aura.
- The headache should start with the aura or within 60 minutes after it begins.

Aura Characteristics

- Reversible dysphasic speech disturbance.
- Unilateral sensory symptoms.
- Homonymous visual symptoms.

Additional Features

- Accompanied by nausea or vomiting and either phonophobia or photophobia.
- At least one symptom of the aura gradually progresses over time, with each lasting 5 to 60 minutes[11].

INVESTIGATIONS

Computed tomography Magnetic resonance imaging

MANAGEMENT

FIRST LINE MEDICATION

NSAIDs are widely used, with the most robust evidence supporting:

- acetylsalicylic acid
- ibuprofen
- diclofenac potassium

SECOND LINE MEDICATION

Triptans are most effective when taken early in an attack, while the headache is still mild. If other triptans have failed, or for patients who quickly reach peak headache intensity or cannot take oral triptans due to vomiting, **subcutaneous sumatriptan** may be a useful option. In the case of relapse, patients can repeat their triptan treatment or combine it with fast-acting formulations of **naproxen sodium, ibuprofen lysine, or diclofenac potassium** for improved effectiveness.

Triptans include:

- Sumatriptan 50 mg tablet , nasal spray 5 mg
- Zolmitriptan tablet 2.5 mg, 5 mg nasal spray
- Rizatriptan tablet 5 mg

THIRD LINE MEDICATION

If all triptans fail after an adequate trial period (defined as no or insufficient response in at least three consecutive attacks) or are contraindicated, alternative treatment options are limited. Ditans or gepants may be considered, though their availability is currently restricted. **Lasmiditan** is the only approved ditan for acute migraine treatment, while **ubrogepant and rimegepant are the only approved gepants[10]**.

II. CLUSTER HEADACHE

Cluster headache is a primary headache disorder classified under the trigeminal autonomic cephalalgias (TACs)[12].

EPIDEMIOLOGY

Cluster headache has traditionally been seen as **more common in males**, with a significant male-to-female ratio. However, women with cluster headache often experience more nausea and vomiting during their attacks.

The average age of onset for cluster headache is similar in both genders, typically occurring in the **third decade of life**.

CLINICAL FEATURES

Patients with cluster headache experience multiple attacks of severe, short-lasting headaches.

These headaches are typically:

- excruciating, unilateral, and
- often affect the first division of the trigeminal nerve, primarily in the periand retro-orbital regions and the temple.
- The pain may be mistaken for sinus or dental pain, leading some patients to seek treatment from an otolaryngologist or dentist.

The pain is described as intense, sharp, and burning, often considered worse than childbirth, earning it the nickname “**suicide headaches.**”

- The attack usually intensifies rapidly, reaching severe pain levels, then subsides just as quickly, with a distinct onset and resolution.
- The period during which patients experience cluster headache attacks is referred to as a **bout**, and it typically lasts between 6 and 12 weeks on average[13].

MANAGEMENT

ACUTE: Subcutaneous sumatriptan injection

Sumatriptan or zolmitriptan nasal spray

100 % oxygen inhalation 12-15 L/min

TRANSITIONAL :Block of great occipital nerve using betamethasone and lidocaine with adrenaline

Prednisolone 75-100 mg for 5 days

PREVENTIVE: FIRST LINE: Verapamil 360-960 mg

SECOND LINE: Lithium 0.5-0.8 mmol/L Topiramate 50-200 mg[14].

III. TENSION TYPE HEADACHE

Tension-type headaches (TTH) are recurring episodes of headache that can last anywhere from minutes to weeks. The pain is usually described as pressing or tightening, of mild to moderate intensity, and typically affects both sides of the head. It does not worsen with routine physical activity. Nausea and vomiting are generally absent, although photophobia or phonophobia may occur. These headaches were once referred to by various terms, including psychogenic headache, stress headache, psycho-myogenic headache, and muscle contraction headache[15]. The pain of tension-type headaches (TTH) is typically described as dull, pressure-like, constricting, or giving a sense of fullness in the head.

INCIDENCE

Most prevalent in females when compared to male population.

Incidence of occurrence decreases with decrease in age.

Common precipitating and aggravating factors

- Stress
- lack of sleep
- irregular eating patterns
- alcohol
- menstruation have also been identified as trigger by patients with episodic tension-type headaches (ETTH).

DIURNAL VARIATION

Tension-type headaches (TTH) are often reported to begin at some point during the day and gradually intensify. The headache typically persists throughout the day, remaining relatively unchanged during various activities, although some individuals may experience worsening symptoms by late evening.

INVESTIGATION

Through examination of symptoms.

NON-MEDICATION TREATMENT

The most commonly used non-medication treatments for headaches include :

- Biofeedback
- relaxation training
- self-hypnosis
- cognitive therapy

Acupuncture[15]

MEDICATION

Analgesics: Ibuprofen 400 mg

Ketoprofen 25 mg

Sedating antihistamines: Promethazine-25 mg twice/day

Diphenhydramine-25-50 mg/4-6 hours

Antiemetics: Metoclopramide-10 mg/day

Selective serotonin reuptake inhibitors: Paroxetine-12.5-75 mg/day **PROPHYLAXIS:** Amitriptyline 10-

75 mg 1-2 hours before bed[16].

C. TRIGEMINAL NEURALGIA

Trigeminal neuralgia also known as TIC DOULOUREUX is defined as the sudden, severe, brief, stabbing and recurrent pain within the distribution of one or more branches of trigeminal nerve[18]. Trigeminal neuralgia (TN) is a chronic facial pain disorder that impacts the trigeminal nerve, which is responsible for sensation in the face. For individuals with TN, even mild stimulation of the face can trigger severe, intense pain. Initially, patients may experience brief and mild episodes, but the condition can worsen over time, leading to longer and more frequent bouts of pain.

TN is more common in women than men and is typically seen in individuals over the age of 50.

SYMPTOMS

- Pain in the cheeks, jaw, teeth, gums, or lips.
- Pain typically affects one side of the face.
- Episodes of severe, shooting pain that may feel like an electric shock.
- Pain lasting from a few seconds to several minutes.

• Pain may be localized to a specific spot or spread across a broader area of the face.

TRIGGERS

- Eating
- Drinking
- Smiling
- Brushing your teeth
- Talking
- Touching your face
- Washing your face

CAUSES

- Vascular compression mainly at nerve root
- Demyelinating diseases like multiple sclerosis
- Inflammatory and autoimmune disease like Sjogren's syndrome
- Posterior fossa and cervical malformations
- Eagle syndrome
- Oropharyngeal carcinoma

MANAGEMENT

Management of trigeminal neuralgia (TN) can involve nonsurgical or surgical approaches.

NON SURGICAL MODALITIES

Several medications are used to alleviate pain, including:

- **FIRST LINE DRUGS:** carbamazepine
- **SECOND LINE DRUGS:** Baclofen, Phenytoin, Pregabalin
- **Antidepressants,** such as amitriptyline
- **Muscle relaxants,** such as baclofen

If medications are ineffective, **Botox injections** may be used [18].

SURGICAL APPROACHES

If medications fail to provide relief, surgical options are available. These are divided into two main categories:

1. Open surgery 2. Lesioning procedures

OPEN SURGERY

- Microvascular decompression
- Rhizotomy
- Peripheral neurectomy and nerve blockade ☐ Deep brain stimulation
- Botulinum toxin injection [17].

LESIONING PROCEDURES

Several minimally invasive procedures can be used to treat trigeminal neuralgia (TN), including:

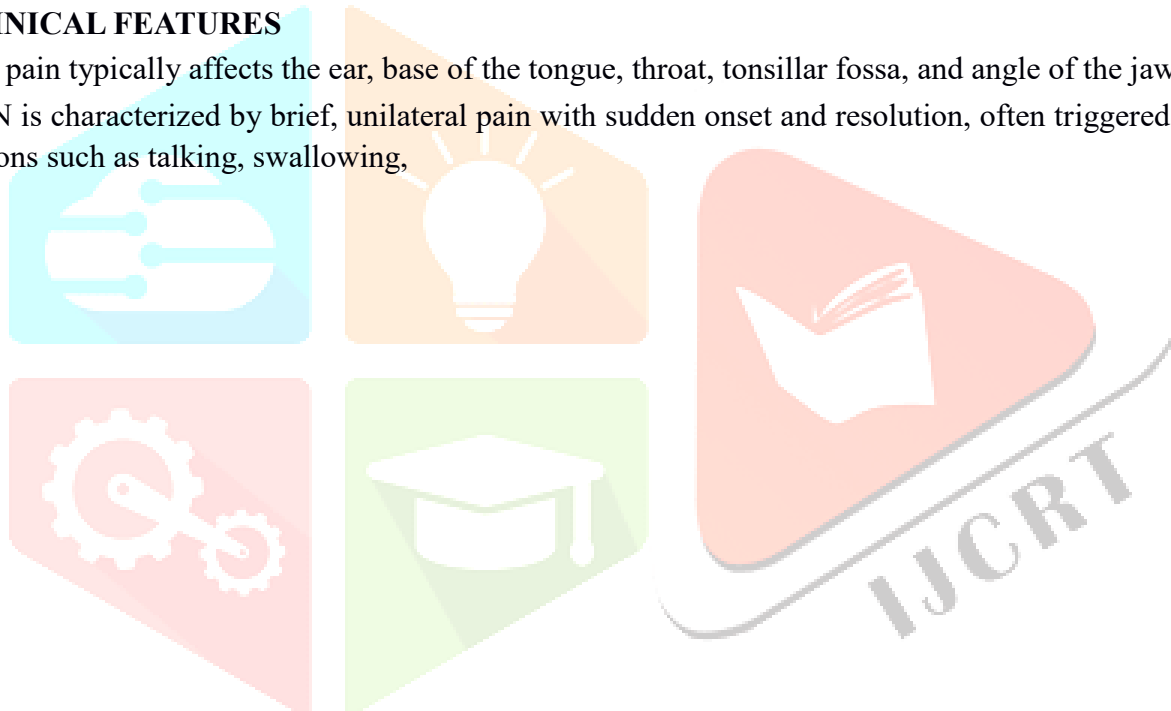
- Percutaneous radiofrequency rhizotomy
- Percutaneous balloon compression
- Percutaneous glycerol rhizotomy
- Stereotactic radiosurgery (Gamma Knife or Cyber-knife) [19].

D.GLOSSOPHARYNGEAL NEURALGIA

Glossopharyngeal neuralgia (GPN) is an extremely rare pain syndrome, distinct from trigeminal neuralgia. It causes electric, shooting pain in the sensory distribution of the auricular and pharyngeal branches of the glossopharyngeal (IX) and vagus (X) cranial nerves.

CLINICAL FEATURES

- The pain typically affects the ear, base of the tongue, throat, tonsillar fossa, and angle of the jaw.
- GPN is characterized by brief, unilateral pain with sudden onset and resolution, often triggered by simple actions such as talking, swallowing,



yawning, coughing, or touching the external auditory canal and the side of the neck.

- Episodes can last from 2 seconds to 2 minutes and may occur up to 200 times per day.
- glossopharyngeal neuralgia (GPN) produces pain on the left side of the body, while trigeminal neuralgia (TN) tends to affect the right side.
- Bilateral pain and associations with multiple sclerosis are more commonly seen in TN.
- Glossopharyngeal neuralgia, like trigeminal neuralgia, typically occurs in individuals who are 50 years old or older.
- In some cases of glossopharyngeal neuralgia (GPN), patients may experience cardiovascular or neurological symptoms, including asystole, bradycardia, hypotension, syncope, seizures, or even cardiac arrest. These symptoms are due to the overlapping connections with the vagus nerve.
- Other rare comorbidities associated with glossopharyngeal neuralgia (GPN) include tinnitus, vertigo, involuntary movements, and neurovegetative phenomena such as tearing, sweating, salivation, and unilateral mydriasis.
- Areas of distribution of glossopharyngeal nerve include the mastoid, back of the throat, posterior third of the tongue, back of the nose, Eustachian tube, middle ear, tonsils, and voice box.
- Patients may experience coughing, hoarseness, and difficulty speaking or swallowing.
- The pain typically starts at the back of the tongue or throat and radiates to the back of the jaw or ear.

DIAGNOSIS

- CT
- MRI
- MRA

MANAGEMENT

Medication: Carbamazepine-200 mg/day

Gabapentin-100 to 5000mg/day in 1 to 4 divided dose

MINIMALLY INVASIVE PROCEDURES

- Pulsed radiofrequency therapy
- Nerve block near styloid process using 2 ml 0.25% bupivacaine
- Percutaneous radiofrequency thermocoagulation

SURGICAL OPTIONS

- Microvascular decompression
- Rhizotomy [20]

E. TEMPOROMANDIBULAR JOINT ANKYLOSIS

Temporomandibular joint ankylosis refers to the fusion of joint components, either through bone or fibrous tissue, leading to restricted mouth opening. This condition can result in challenges with chewing, speaking, and maintaining oral hygiene, while also potentially affecting mandibular growth.

TYPES

1. True temporomandibular joint (TMJ) ankylosis-It directly involves the joint itself
 2. false TMJ ankylosis- occurs outside the joint.
- ☐ Based on the type of tissue that develops within the joint's intracapsular structures, ankylosis can be categorized as:
1. Fibrous
 2. bony
 3. mixed

CLASSIFICATION

Sawhney classified TMJ ankylosis into four types:

- **Type I:** The condylar head is deformed but still recognizable, with fibroadhesions preventing joint movement.

- **Type II:** The deformed condylar head and articular surface fuse mainly at the edges, while the medial part of the condylar head remains intact.
- **Type III:** The ankylotic mass extends to the mandibular ramus and zygomatic arch, with an atrophic and displaced fragment of the anterior condylar head positioned medially.
- **Type IV:** Complete obliteration of the TMJ occurs due to a bony ankylotic mass forming between the mandibular ramus and the cranial base.

CAUSES

- Trauma
- Infections(local or systemic)
- Perinatal trauma from forceps
- Children with age less than 10, Temporomandibular disc trauma, prolonged mandibular immobilization after intracapsular trauma
- Myositis ossificans
- Osteochondroma
- Rheumatoid arthritis
- Radiotherapy
- Surgical treatment of TMJ
- Psoriatic arthritis
- Ankylosing spondylitis

CLINICAL FEATURES

- TMJ ankylosis causes mandibular deformity and growth impairment, leading to difficulties in mastication, swallowing, and speaking.
- Poor oral hygiene due to restricted mouth opening increases the risk of dental caries and periodontal disease.
- Deformed alveolar processes also disrupt tooth eruption and positioning.
- The facial profile of affected individuals is often described as a “bird profile,” characterized by a significantly shortened lower face, retruded mandible with an absent chin, an obtuse cervicomental angle, and an enlarged nasolabial angle.
- Facial asymmetry is evident, with the chin deviated toward the affected side.
- Lip incompetence occurs, with the lower lip trapped behind the upper front teeth.
- As the child grows, facial asymmetry worsens due to restricted mandibular mobility, impaired growth, and abnormal muscle function.
- Prolonged ankylosis can lead to muscle atrophy, while secondary elongation and hypertrophy of the coronoid process further restrict mandibular movement.

TREATMENT

A . Surgical treatment options for TMJ ankylosis include:

1. **Arthroplasty** – Removal of the ankylotic mass to restore joint movement.
2. **Arthroplasty with a free costochondral graft** – Reconstruction using a rib graft [12].
3. **Arthroplasty with temporalis myofascial flap insertion** – Creating a new joint cavity with a muscle flap, often combined with unilateral or bilateral coronoidectomy.
4. **Mandibular distraction osteogenesis** – Lengthening the ramus and body of the mandible on the affected side.
5. **Joint reconstruction with an alloplastic prosthesis** – Using synthetic materials to replace the joint [13,14].
6. **Arthroscopic laser-assisted surgery** – Minimally invasive reshaping of the articular surfaces [15].
7. **Postoperative radiotherapy** – Used to prevent re-ankylosis [5,16].

8. Bilateral arthrotomy – Surgical intervention on both TMJs when necessary.

B . Autogenous Bone Grafts in TMJ Reconstruction

C . Distraction Osteogenesis

D. Alloplastic Replacement of the Temporomandibular Joint[21].

F. FREY SYNDROME

Frey Syndrome is a postoperative condition that typically follows salivary gland surgery, but it can also occur after neck dissection, facelift procedures, or trauma. It is characterized by gustatory sweating (sweating triggered by eating) and flushing in the affected area. It was first described by Lucie Frey in 1923, this condition was originally referred to as auriculotemporal syndrome. It specifically involves sweating and flushing in the preauricular region (near the ear) in response to chewing or salivary stimulation, a phenomenon that occurs due to aberrant nerve regeneration, where parasympathetic fibers meant for the salivary glands inadvertently innervate sweat glands in the skin.

PATHOPHYSIOLOGY

The synkinetic mechanism behind Frey syndrome involves aberrant reinnervation of postganglionic parasympathetic neurons. After nerve damage, these neurons, which normally innervate the salivary glands, mistakenly grow towards and reinnervate nearby denervated sweat glands and cutaneous blood vessels. As a result, the stimulation that would typically trigger salivation (such as chewing) also causes excessive sweating and flushing in the affected area, typically around the preauricular region. This abnormal nerve connection is responsible for the characteristic symptoms of Frey syndrome.

SYMPTOMS

- Flushing
- sweating
- Burning
- Neuralgia
- Itching

DIAGNOSIS

- primarily it is based on clinical history, including a history of salivary gland surgery, trauma, or other relevant procedures.
- confirmatory testing can be performed using the **Minor starch-iodine test**. In this test, iodine is applied to the affected skin area, followed by starch. When the skin sweats, the starch reacts with the iodine, turning a dark blue color, which confirms the presence of gustatory sweating characteristic of Frey syndrome.

MANAGEMENT

- increased skin flap thickness
- local fascia or muscle flaps
- acellular dermal matrix (ADM)
- free fat grafts
- Biomaterials and autologous implantation[22]

G. BELL'S PALSY

Bell's Palsy is an idiopathic, unilateral, and acute facial weakness that follows a pattern consistent with peripheral facial nerve dysfunction. The degree of weakness can be partial or complete, and it typically affects both sides of the face with equal frequency.

FEATURES

- **Unilateral Facial Weakness:** The condition causes sudden-onset facial paralysis, typically affecting only one side of the face.

ETIOLOGY

Idiopathic: The exact cause remains unknown, though there is weak evidence linking it to herpes simplex virus (HSV) infection, which may trigger inflammation of the facial nerve.

SYMPTOMS

- **Mild Pain:** Often felt in or behind the ear, oropharyngeal, or facial numbness.

- Hyperacusis: Impaired tolerance to ordinary levels of sound due to involvement of the stapedius muscle.
- Disturbed Taste: Affected taste sensation on the anterior two-thirds of the tongue due to involvement of the chorda tympani nerve.
- Pain: Severe pain is more indicative of herpes zoster virus infection, such as Ramsay Hunt syndrome, rather than Bell's palsy.

DIFFERENTIAL DIAGNOSIS

Since Bell's palsy is a diagnosis of exclusion, it's important to rule out other potential causes of lower motor neuron facial weakness, including:

- Middle ear infections
- Parotid gland malignancy
- Malignant otitis externa
- Lateral skull base tumors

DIAGNOSIS

The diagnosis of Bell's palsy is typically made clinically, but further investigations may be required to exclude other conditions. Imaging (e.g., MRI) and electrodiagnostic tests (such as nerve conduction studies) may be performed if another cause is suspected.

TREATMENT

- Corticosteroids (e.g., prednisone) are often prescribed to reduce inflammation and improve recovery.
- Antiviral treatment is not routinely recommended unless there is strong suspicion of viral infection, particularly herpes zoster.
- Physical therapy and facial exercises may help with recovery of facial muscle strength.

PROGNOSIS

The majority of patients with Bell's palsy recover fully within 3 to 6 months. However, some may experience residual symptoms such as mild facial weakness or asymmetry. Early intervention with steroids can improve the chances of full recovery[23].

H. POST HERPETIC NEURALGIA

Postherpetic neuralgia (PHN) is the most common long-term complication of varicella-zoster virus (VZV) reactivation, also known as human herpesvirus-3 (HHV-3). PHN occurs when persistent nerve pain remains after the rash of herpes zoster (shingles) has healed.

PATHOPHYSIOLOGY

- Varicella-Zoster Virus (VZV): The virus is responsible for varicella (chickenpox) in childhood. After the initial infection, VZV becomes latent in the dorsal root ganglia.
- Reactivation as Shingles (Herpes Zoster): In immunocompromised individuals or older adults, VZV can reactivate, leading to a painful, blistering rash known as shingles.
- Development of PHN: In some patients, nerve damage caused by VZV leads to chronic neuropathic pain, persisting for months or even years after the rash has resolved.

RISK FACTORS

- Advanced age (higher risk in individuals over 60)
- Severe initial herpes zoster episode
- Delayed antiviral treatment
- Immunosuppression (e.g., chemotherapy, HIV, organ transplantation)

SYMPTOMS

- Persistent neuropathic pain in the affected dermatome, lasting >90 days after rash resolution
- Burning, stabbing, or electric shock-like pain
- Allodynia (pain triggered by light touch)
- Hyperalgesia (increased sensitivity to pain)

PREVENTION

- Shingles vaccine (Recombinant Zoster Vaccine – Shingrix) significantly reduces the risk of herpes zoster and PHN.
- Early antiviral treatment (e.g., valacyclovir, famciclovir, acyclovir) within 72 hours of rash onset may reduce PHN risk.

TREATMENT

- Gabapentin or pregabalin (first-line therapy)
- Tricyclic antidepressants (TCAs) such as amitriptyline
- Serotonin-norepinephrine reuptake inhibitors (SNRIs) (e.g., duloxetine) **Topical treatments:**
- Lidocaine patches
- Capsaicin cream
- Opioids (reserved for severe, refractory cases)[24].

I. NERVUS INTERMEDIUS NEURALGIA

Nervus intermedius neuralgia is an extremely uncommon pain syndrome that is similar to trigeminal neuralgia, but the pain occurs in the distribution of the nervus intermedius. This nerve is a branch of the facial nerve (cranial nerve VII) and is responsible for sensory innervation to parts of the ear, the soft palate, and the mucous membranes of the nasopharynx, among other areas.

SYMPTOMS

- **Pain distribution:** The pain typically affects the ear, soft palate, and throat regions, similar to trigeminal neuralgia but located in the nervus intermedius distribution.
- The pain is described as sharp, stabbing, or electrical, often triggered by specific stimuli, such as chewing, talking, or touching the affected areas.

ETIOLOGY

- **Cross-compression:** The most commonly suggested cause is vascular compression at the central-peripheral myelin junction of the nerve. This compression leads to demyelination of the nerve, resulting in the characteristic sharp, paroxysmal pain seen in the condition.
- **Lesions or tumors near the brainstem** can also potentially contribute to nerve compression or irritation, but this is less common.

DIAGNOSIS

- Diagnosis is typically clinical, with the characteristic distribution of pain and exclusion of other causes such as trigeminal neuralgia or glossopharyngeal neuralgia.
- MRI or CT scans may be used to identify vascular compression, lesions, or tumors affecting the nerve.

TREATMENT

1. Medication:

- Carbamazepine and oxcarbazepine, which are commonly used in the treatment of trigeminal neuralgia, may also be effective for nervus intermedius neuralgia.
- Gabapentin or pregabalin could be used as alternatives in cases of medication intolerance.

2. Surgical Options:

- In cases where medical treatment fails, **microvascular decompression (MVD)** may be considered. This procedure involves relieving pressure on the nerve, which can significantly improve symptoms in patients with vascular compression of the nerve.

3. Other Interventions:

- Botulinum toxin injections and gamma knife radiosurgery may be considered in refractory cases[25].

CONCLUSION

Orofacial pain management can be complex, and clinicians must be familiar with the various causes and characteristics of the different disorders affecting the orofacial region. An orofacial pain specialist, with their expertise and knowledge, is crucial for providing accurate diagnosis and effective treatment. A

multidisciplinary approach is considered ideal for managing orofacial pain disorders. Orofacial pain conditions arise from complex pathophysiology, often accompanied by psychological comorbidities. Chronic orofacial pain can severely impact quality of life and daily functioning. Early diagnosis and referral to secondary care are crucial to ensure evidence-based management is initiated. A biopsychosocial approach to pain management can address the multifactorial causes of orofacial pain while minimizing the economic and health-related burden associated with these conditions.

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