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Disorders of Masticatory Muscle: An Integrated Review

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Abstract

Masticatory muscle pain (MMP) is a painful disorder involving the muscle of mastication, presenting with varied manifestations of jaw pain. These conditions range from the muscle pain being the primary condition or secondary to other diseases. A thorough clinical examination, following careful patient history will facilitate in establishing a tentative diagnosis following prompt treatment therapies. The article mainly empathies with the systematic overview of various type of masticatory muscle disorders in terms of their clinical implications, prevention and management and as well enlightens the importance of accurate identification and differentiation of MMP from primary temporomandibular joint disorders such as those involving pain from osteoarthritis, disc displacement, or jaw dysfunction.

Keywords: Masticatory muscle disorder; Muscle pain; Myalgia; Tendonitis; Myositis; Myospasm; Contractures; Hypertrophy; Neoplasms; Movement Disorders; Systemic/central pain disorders; Fibromyalgia.

Introduction

Masticatory muscle pain (MMP) emanates from the fascia, tendons and skeletal muscles. It is associated with regional and referred pain, characterized by localized muscle tenderness in taut bands of skeletal muscles. The associated muscles may display strings of symptoms like: increased fatigability, stiffness, subjective weakness, pain in movement, and slight restricted range of motion, which may not be related to joint restriction.[1,2]

Major cause of chronic non dental orofacial pain is MMP, accounting for about 12-14% of adult population of which approximately 16% seeks treatment. Pain remains short-lasting and episodic in a vast majority of patients, becoming chronic in only about 20%. It has enlisted risk factors like: gender, age, stress and other psychological factors, phenotype, parafunctional oral habits, physical and psychological trauma.[3]

An assumption of initial pain, remains muscle overuse but its persistence may lead through nociceptive bombardment of central sensitization thereby enhancing the pain sensitivity. Emotional, cognitive distress and affective factors are factors involved in the pain enhancement.[3]

The pain does not stick to any radicular boundary and is felt in pre-auricular area, face, jaws and also radiate to head and neck regions, teeth and ear.

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Normally its unilateral, if bilateral, involvement of one side is more intensely felt. May exhibit alterations by jaw function including eating, jaw movements, yawning, and occasionally talking. It typically reaches a value of 5.0-6.0 on a usual visual analogue scale. Pain presents as aching, stabbing, dull, pressing, pulling, rarely as burning, and can be accompanied by a light paresthesia.[3]

MMP fluctuates over time remains acute, episodic or persistent, chronicity is seen in only about 20% of people. It often leads to high degree of sleep disturbance and somatization accompanied by depression and anxiety.[3]

To help validate an explanatory model for etiology and treatment, understanding the above factors is essentially important. This would in-turn help in coping up with the idea of seeking evidence, after thorough evaluation of pain in the masticatory muscle region.[3]

The classification and diagnostic criteria that has been followed for this review has been taken from" Expanded taxonomy for temporomandibular disorders (Peck et al. 2014)" with an aim to provide a consensus-based classification system and associated diagnostic criteria that have clinical as well as research utility, with an added attempt to enumerate and elaborate the importance of masticatory muscle pain disorders for clinical diagnosis and treatment planning.

Neurophysiology of masticatory muscle pain

The mechanism which underlies the pain in the skeletal muscle is yet to be understood. Activation of nociceptors leads to acute muscle pain. These are specialized receptors potentially damaging tissue stimuli, that may lead to pain perception. Its connected to the central nervous system (CNS) with free nerve endings via unmyelinated (group IV) or thinly myelinated (group III) fibers, which gets activated by trauma and mechanical loading and endogenous inflammatory mediators like-bradykinin (BK), serotonin, and prostaglandin E2 (PGE2).[4]

A nociceptor is only activated when nociceptor terminal is depolarised sufficiently by normal resting membrane potential of nerve. Muscle pain generation requires Adenosine triphosphate (ATP) and protons (H+) particularly which activates nerve endings by binding to membrane specific receptors located in nerve ending, with release of H+ when there is fall in PH. as many painful muscle disorders (e.g., local myalgia, myospasm, myositis) are speculated to be associated with low pH in muscle tissue.[4]

Nerve growth factor (NGF) also plays an important role in inflammatory muscle pain. NGF may lead to only subthreshold excitatory postsynaptic potentials (EPSPs) due to low-frequency firing of action potentials from neural endings, not resulting in conscious pain though but sensitizing second order neuron predisposing to chronic pain.[4]

Masticatory muscle pain model

Model begins with an assumption of the muscles being healthy and functioning normally. Muscles can be interrupted by certain type of events, if it's significant, a muscle response known as protective co-contraction (muscle splinting) occurs.[1]

Mostly as the event being minor, co-contraction resolves normally, thus allowing normal muscle functioning. However in case co-contraction is prolonged initial biochemical and later structural changes occurs, creating local muscle soreness, this may resolve spontaneously or require treatment. Prolong pain input is resulted following no resolution of local muscle soreness, affecting the central nervous system and leading to certain muscle responses.[1]

Usually, masticatory muscle disorders are relatively acute as the muscles returns to normal upon treatment. However, if recognised wrongly and inappropriately managed can pertain to chronic myalgic disorders, with CNS being the important factor and referred as centrally mediated myalgia, often difficult to resolve.[1]

Fibromyalgia, yet another example of chronic musculoskeletal pain disorder, a condition wherein little cause can be found in the peripheral tissue, although the patients suffers with widespread musculoskeletal pain, which is managed quiet differently then the acute muscle disorders.[1]

Thus the mainstay for treatment of MMP remains the rehabilitation of trigger points, with an effort made to reduce all the contributing factors.[5]

Classification of Masticatory Muscle Disorder

Masticatory muscle disorders

1. Muscle pain

A) Myalgia

- Local myalgia
- Myofascial pain

- Myofascial pain with referral

B) Tendonitis

C) Myositis (Centrally Mediated Myalgia)

D) Spasm

2. Contracture

3. Hypertrophy

4. Neoplasm

5. Movement Disorders

A) Orofacial dyskinesia

B) Oromandibular dystonia

6. Masticatory muscle pain attributed to systemic/central pain disorders

A) Fibromyalgia/widespread pain.[6]

Myalgia

As defined according to DC/TMD criteria, it's a pain of jaw origin affecting the jaw movement due to function or parafunction and with provocation testing of masticatory muscles, replication of this pain occurs. The cause has been attributed to infection or direct muscular trauma, muscle overuse due to parafunction or related to stress induced primary myogenous pain.[4]

Three subclasses includes: local myalgia, myofascial pain, and myofascial pain with referral.[7]

The Criteria for diagnosis, includes a positive history for both Pain in the jaw, temple, in front of the ear, or in the ear and pain modified with jaw movement, function or parafunction. During examination being positive for confirmation of pain location, report of familiar pain with atleast one of the following provocation test pain on palpation or during maximum unassisted and assisted opening while examining masseter and temporalis muscle.[8]

- Local Myalgia

Defined as pain localised to immediate site of tissue stimulation and can be localised by palpation.[7,4]

Etiology

It developes as a response to local trauma or locally painful pathologic processes such as injury (protective muscle splinting/co-contraction), acute joint inflammation, delayed post exercise soreness, muscle fatigue or pain from ischaemia, can also result from the alterations of normal biochemical environment.[4,9]

Features

Seen bilaterally, with soreness of muscles of mastication along with pain in cheeck and temple on wide opening, waking and chewing. Its described as stiff, sore aching spasm with tightness and cramping, followed by weakness and fatigue with reduced mandibular range of motion leading to limited opening.[9,10]

Differential diagnosis: myositis, myofascial pain, neoplasm, fibromyalgia.[10]

Treatment

The standard treatment includes proper rest, use of ice/moist heat and NSAIDS, followed by frequent daily active mobilization of neck and jaw muscles until the establishment and maintenance of normal range of movement.[9]

Myofascial pain (myofascial trigger point pain)

It's a muscle disorder that's not completely understood or widely appreciated but it's very common amidst patients of myalgic complaints. It presents as a regional myogenous painful condition , characterised by taut bands of muscle tissues known as "trigger points".[11]

Its painful mechanism begins with constriction of blood vessels, leading to muscle hypertrophy leading to contraction of fibres and nodule formations (taut bands), thus enhancing pain.[12]

Etiology

Is considered to be multifactorial, Travell and Simons attributed following conditions like: trauma, hypovitaminosis, poor physical conditioning, fatigue, and viral infections. Other factors likely to be stress and deep pain input. Since, there is lacking of complete understanding of this condition it's difficult to be specific on etiologic factors.[11]

Clinical characteristics

- Structural dysfunction: with decreased range of movement.
- Pain at rest
- Increased pain with function
- Presence of trigger points: characterised as 2-5mm in diameter, palpable taut bands in ligament, tendon and skeletal muscle.[11,2]

Patients in many instances may not even

acknowledge of trigger points and only be aware of referred pain.[11]

Myofascial pain with referral

The pain of muscle origin "myalgia" along with pain extending beyond the boundary of masticatory muscle such as ear, teeth or eye, with limitation of mandibular movement secondary to pain. Presence of taut bands is also noted (contractures of muscle fibres).[7]

Locations

Trigger point (TrP) of occipital belly of the occipito - frontalis muscle produces referred headache pain behind the eye, TrP involving the temporalis causes pain in the temple area, frontal area, retro-orbital area, and the anterior teeth of the maxilla-leading to teeth sensitivity in affected area, TrP involving the deep masseter muscle causing pain in the area anterior to the ear, TrP involving the splenius capitus muscle causes pain in the frontal, vertex, occipital areas and at the back of the neck and trigger point pain in the trapezius muscle creating referred pain in the temple region.[11]

Diagnosis

Five major criteria and at least one out of three minor criteria are satisfied.[13]

Major criteria includes:

- Localised spontaneous pain or altered sensation in the expected referred area for a given Trp.
- Accessibility of a taut palpable band.
- Exquisite.
- Localised tenderness along taut band.
- Certain reduced degree of measured range of movement.[13]

Minor criteria includes:

- Spontaneous reproduction of perceived pain and altered sensation by pressure on Trp.
- Elicitation of (taut band, local twitch response (LTR) of muscle fibres by transverse 'snapping' palpation or by needle insertion into the TrP.
- Relief of pain by stretching of muscle and injections of Trp.[13]

Differential diagnosis: myalgia, myositis, neoplasia.[13]

Management

Requires an intensive multidisciplinary approach, which includes both surgical and non-surgical modalities.[12]

Non-surgical approach being reassurance, diet modification by elimination of hard chewy foods, limited jaw motion while yawning, avoidance of parafunctional habits, use of anterior splints to eliminate grinding and clenching, followed by rest and supportive pharmacologic therapy directing towards relief of dysfunction.[14]

Physical therapy may include use of stretch, sprays, ischaemic pressure soft pressure continuous massaging along with continuous suppleness exercise. Electrogalvanic and transcutaneous stimulation along with acupuncture, ultrasound, iontophoresis, cryotherapy, biofeedback and cold or soft laser can be further adopted.[12,14]

Pharmacologic therapy may include use of analgesics – such as opioids (morphine, pethidine, codeine) and non–opioid analgesics (salicylates, paracetamol) and anti-inflammatory agents like-salicylates (aspirin), propionic acid (ibuprofen), acetic acid (indomethacin), fenamic acid, oxicam, and aryl-acetic acid derivatives (diclofenac sodium) which acts by relieving pain with or without depressing the CNS. Anxiolytics agents also has been used to alter the patients perception to pain and reaction to supportive therapy.[12]

Trigger point injections are considered as the most efficient means of. A peppering technique is used where is needle is moved in and inside the area of muscle spasm rapidly, thereby inactivating all the foci within trigger point.[15]

Dry needling has also been effectively proposed, as it acts by mechanical disruption of the integrity of dysfunctional end plate.[13]

Followed by psychologic counseling as an individual's reaction to stress is related to their emotional stress.[14]

Tendinitis

Tendinitis has been described as pain of tendon origin, being affected by movement of jaw, function and parafunction and replication of this pain with provocation testing of the masticatory tendon, leading to inflammation exhibited by substance of tendon referring to a clinical syndrome but not to any specific histopathological entity.[16]

Temporal tendinitis is included under masticatory muscle disorders.[16]

Etiology

Chronic parafunctional activities (i.e., intense clenching and bruxing), mandibular trauma, prolonged opening of the mouth, often during dental procedures or intubation for general anesthesia have been attributed. Also, history of direct trauma to the side of the face, such as in a fall on a hard surface, or a sudden movement, such as lateral whiplash from a motor vehicle accident have also been suggested.[16]

Clinical presentation

It presents with inflammation of temporal tendon, with symptoms mimicking to those of an internal derangement of the temporomandibular joint (TMJ) with pain in tmj and ear, temporal headache in association with parafunctional habits and teeth wear. The characteristic ear pressure, exhibits sentation of water caught in ear after swimming or taking shower .This disorder is exhibits a concurrent association with other conditions of craniofacial region like - anterior dislocation of the ipsilateral articular disk of the temporomandibular joint and ernest syndrome.[17]

Differential diagnosis

Anteriorly Dislocated Articular Disk of the Temporomandibular Joint.[17]

Diagnosis

Is based on palpation, dynamic and provocation tests, followed by evaluation of the end-feel that may be indicative of a muscle/tendon restriction.[18]

Management

Treatment for temporal tendinitis is simple and straight forward. It involves both conservative and surgical approaches.[17]

Conservative therapy consists of injecting both temporal tendon heads with 1.8 cc mixture of Marcaine (AstraZeneca, Wilmington, DE) (for prolonged anesthesia) mixed with a suitable anti-inflammatory agent [e.g., (betamethasone, dexamethasone, triamcinolone, and Sarapin (*Sarapin* is a biological medicine derived from the pitcher plant) after anesthetic confirmation of the disorder.[17]

Centrally mediated myalgia (chronic myositis)

It's a disorder originating in the central nervous system, characterised as chronic and continuous, generating symptoms at the periphery of muscle tissue.[19]

This condition is not associated with classical inflammatory signs like redness or swelling, but is rather associated with pain at rest. A constant and prolonged episodes of pain leads to this condition, rather then periodic episodes.[11,19]

Etiology

The involvement of CNS is more rather then the muscle itself, with its greater involvement, antidromic neural impulses are sent out to the muscular and vascular tissues, producing local neurogenic inflammation. The neurogenic inflammation produces pain in these tissues even though the main cause remains CNS, hence the term centrally mediated myalgia.[11]

Environmental risk factors might include viruses, bacteria, ultraviolet radiation, smoking, occupational and perinatal exposures and a growing list of drugs (including biologic agents) and dietary supplements.[20]

Clinical characteristics

The six characteristics very commonly seen are structural dysfunction exhibited as reduced range of mandibular movement, pain at rest, increased pain with function, local muscle tenderness, muscle tightness and contracture.[11]

Diagnosis

Is based on two significant findings in history of the patient, firstly duration of pain, as this condition takes a long time to develop, Typically, the pain will be present for at least 4 weeks and often accounting for several months. Secondly the constancy of pain, patient presents with pain even with jaw at rest.[11]

Further testing must include use of additional non –invasive imaging tools like Muscle imaging MRI, ultrasonography, positron emission tomography (PET) and computed tomography (CT).[21,7,4]

Management

As described by Okeson the main therapeutic modalities for this pathological process includes- reversible occlusal therapy, restriction of movements of the mandible, adoption of dietary habits, with inclusion of pastier foods and interdisciplinary follow-up.[19]

Myospasm (tonic contraction myalgia)

It's a tonic muscle contraction induced by

CNS, characterised as sudden, involuntary, contraction of the muscles affecting the muscles of mastication.[7,5]

Etiology

Its multifactorial, which appears to be related to continuous deep pain input, metabolic local factors within the muscle tissue associated with fatigue or overuse, local muscle condition like change in electrolyte balance, presence of another musculoskeletal disorder.[11,22]

Classification

Classified into jaw closing, involving the masseter and temporalis muscle, characterised by limited mouth opening and acute pain. The jaw opening type involving inferior lateral pterygoid muscle, characterised difficulty in jaw closing after wide opening along with involuntary jaw movements.[22]

Clinical characteristics

Patients with this condition exhibits structural dysfunction, with marked restriction in range of movement determined by muscle spasm or acute malocclusion, with a sudden change in the occlusal contact pattern of the teeth secondary to a disorder, as a result of a myospasm in the inferior lateral pterygoid muscle, followed by pain at rest, increased pain at function, muscle soreness and tightness.[11]

Diagnosis

A thorough history which is evident, with patient reporting sudden onset of pain, tightness, and often a change in jaw position, with difficult mandibular movement. Laboratory testing is also positive for Elevated intramuscular electromyography (EMG) activity when compared to contralateral unaffected muscle.[7,11]

Differential diagnosis: myositis, local myalgia, neoplasm.[10]

Management

The management is directed two ways, firstly reducing the spasm itself whereas other addressing the cause, best treated by pain reduction followed by passive lengthening and stretching of the involved muscle. Manual massage can also be used for pain reduction. Pain can be reduced by use of ice (vapocoolant spray) or injection of local anesthetic (2% lidocaine without vasoconstrictors) into the muscle, with reduction in pain muscle is stretched passively to its full length. In case if the presenting cause is (i.e., deep pain input), attempts should be directed toward elimination of these factors so as to lessen the likelihood of recurrent myospasms. If secondary to fatigue patient is advised rest and establishment of normal electrolyte balance. Even physical therapy techniques have been used for management soft tissue mobilization such as deep massage and passive stretching are the two most important immediate treatments.[11,4]

Contractures

It refers to clinical shortening of the resting length of a muscle, but its ability to contract further is not interefered. It's the most significant consequences of altered passive mechanical properties in muscles. It presents as a potential complications of lesions like cerebral palsy (CP), stroke and spinal cord injury involving the central motar pathway.[7,23,24]

Mechanism

In patients with cerebral palsy (CP), patients after stroke and traumatic brain injury (TBI) patients, Clinical exam and intraoperative inspection clearly reveal that muscle contractures represent a muscle tendon unit that is under extremely high passive mechanical tension.[24]

Classification

It's divided into: myostatic, when a muscle is kept stretched for a prolong time period and myofibrotic, results when there is excessive tissue adhesion between muscle and its sheath. The clinical distinction between the two is difficult but it's essentially important as the way they respond to therapy is different.[11]

Myofibrotic contracture

Resulting from tissue adhesion within muscle or its sheath, following trauma or myositic contraction to the muscle.[11]

Etiology

It occurs as a consequences of inflammatory process leading to fibrous changes in the muscle or its sheath, followed by traumatic mysositis or traumatic process. Radiation therapy, incision through a muscle with fibrotic healing, and disuse for long period (>6 weeks), excessive tissue adhesions within the muscle or its sheath can also being among other causes.[4,11]

Clinical characteristics

It presents with limited mouth opening usually

painless, thus affecting the left condylar movement is not affected, and there is no occurrence of acute malocclusion. The joints gets affected by getting fixed in awkward positions, thus obstructing normal physical activities.[4,11,23]

Diagnosis

Is based on positive history for progressive loss of range of motion, previous muscle injury or long term restricted range of movement. Sometimes the patient may not even be aware of the limited movement due to persistence of pain for a long time.[11]

Treatment

Generally surgical detachment of the involved muscle is suggested as a definitive treatment, often muscles that are surgically detached, often reattaches with time. If the range of motion is maintained by passive exercise, restrictions will not return. Supportive therapy is not indicated as myofibrotic contracture is rarely associated with painful symptoms.[11]

Myostatic contracture

It results when the muscles are stretched for a prolong time period. It may also occur secondary to another disorder.[11]

Clinical Characteristics

It is characterized by painless limitation of mouth opening.[11]

Diagnosis

Is established through patients history with restricted jaw movements that begins secondary to a painful condition that has resolved.[11]

Management

Etiologic factors must be eliminated before effective treatment of the contracture can result.

Definitive treatment is directed towards gradual lengthening of the involved muscles only once the original cause has been eliminated. Lengthening is an attempt to re-establish the original resting length of the muscles and must be done slowly over many weeks. Resting length of the muscles can be re-established by two types of exercise: passive stretching and resistant opening. Supportive therapy is of little use in treatment of myostatic contracture.[11]

Hypertrophy

It refers to muscle enlargement caused by increase in cell size not number. The involved muscles are temporalis, masseter and medial pterygoid muscles. The commonest entity is masseter muscle hypertrophy and hypertrophy of temporalis muscle itself is very rare. It can be either congenital or acquired, resulting from para functional jaw habits such as bruxism.[25,26]

Temporalis muscle hypertrophy

May occur unilaterally or bilaterally, although its occurrence is rare. The isolated unilateral temporalis muscle hypertrophy causes painful soft tissue of temple. In case of bilateral hypertrophy of temporalis facial appearance is greatly affected and is cosmetically disturbing to the patients.[25-27]

Etiology

Includes bruxism, malocclusion, prognathism, dental caries, loss of teeth, temporomandibular joint disease, and the bony deformities produced by craniofacial trauma. In absence of any identifiable triggers, it may be regarded to psychogenic factors.[27]

Clinical presentation

In case of bilateral temporal hypertrophy bilateral swelling is noted while in unilateral temporal hypertrophy hardened temporalis enlargement on clenching of teeth accompanied with increased pain and acute tender swelling.[25,28]

Diagnosis

Definitive diagnosis is achieved through incisional biopsy of the temporal muscle preceded by careful history, clinical examination and laboratory investigations, measuring complete blood count and erythrocyte sedimentation rate. Along with use of imaging modalities like MRI and CT for better demonstration of bony involvement.[29,30]

Differential Diagnosis

Genetic (or possibly genetic) MMH (fiber type 2 hypertrophy), congenital MMH, masticatory muscle myopathy (hypertrophic branchial myopathy), vascular malformation—intramuscular cavernous hemangioma.[27]

Treatment

Although there are variety of treatment regime, conservative way has been traditionally employed, with provision of bite freeing splints. Botulinum

toxin (injections of botulinium toxin A) which acts through cholinergic chemodenervation of motor end plates thereby and inhibiting the gamma motor endings in muscle spindles has also been suggested.[28]

Masseter muscle hypertrophy

It presents as bilateral or unilateral benign enlargement of masseter muscle, thereby increasing the volume of muscle mass.[31]

Etiology

It has been attributed to various factors like emotional stress, chronic bruxism, masseteric hyperfunction and parafunction, and microtrauma.[32]

Clinical features

It manifests mainly after puberty, affecting both males and females, patient may present with characteristic painless swelling over angle of jaw with "square face "leading to unaesthetic appearance. Additional complains may include - pain, headache, muscle stress, trismus and intermittent masticatory claudication and its more pronounced on clenching of teeth in case of bilateral hypertrophy.[31,33,34]

Diagnosis

It can be established through clinical history, physical examination, which includes bimanual intra and extra oral palpation to rule out whether or not the swelling is indeed located in the masseter muscle and complementary imaging resources such as computed tomography (CT), which provides an excellent view of the muscle anatomy and enables reliable measurement of the cross sectional area of muscle tissue to be obtained. Along with histological examination and use of other imaging modalities like Conventional radiography, magnetic resonance imaging, and ultrasonography.[32-35]

Differential diagnosis

Unilateral compensatory hypertrophy, masseter tumour, salivary gland disease, parotid tumour, parotid inflamatory disease, masseter muscle intrinsic myopathy, lipoma, vascular tumours, benign, malignant mandible tumours and metabolic disorders.[31]

Treatment

Its multifaceted, with correction of parafunctional habits, adjustments of dental restorations and occlusal adjustments, non–surgical modality includes reassurance. Use of tranquilisers or muscle relaxants, injection of botulinum toxin type a 40–60 iu per muscle administered by intramuscular route have also been recommended.[31]

Neoplasms

Two neoplasms namely (rhabdomyoma) or malignant (e.g., rhabdomyosarcoma) or metastatic neoplasms which are benign in nature have been associated with masticatory muscles.[4]

Rhabdomyoma

It accounts for 2% of skeletal muscle tumors, which are are particularly rare benign tumors of soft tissue originating from the skeletal muscle cells. It's topographically distinguished as cardiac and extracardiac.[4,37]

Clinical features

The peak incidence is encountered during the fifth and sixth decade, with male predominance of 2:1. It is believed to originate from skeletal muscles of the third and fourth branchial arches and appears as slow growing multifocal solitary mass located deeply located. It may be incompletely demarcated from the surrounding tissue following lobular growth pattern, leading to airway obstruction, hearing loss, dysphagia, hoarseness, and odynophagia.[4,37]

Diagnosis

Both CT and MRI used to determine characteristics, extent of local involvement, and possibility of multifocality, Followed by histopathological evaluation.[4,37]

Differential diagnosis

Granular cell tumors, rhabdomyosarcoma, and other sarcomas of the head and neck.[37]

Treatment

Consists of conservative surgical excision along with improvement in combined chemotherapeutic and irradation regimens.[4,37]

Rhabdomyosarcoma

It is tumor of mesenchymal origin, a soft tissue malignant tumor, arising from cells of skeletal muscle linage that initiate myogenic differentiation but fail to disconnect from their proliferative cycle.[4]

Clinical features

It occurs mostly during the first two decades of life commonly seen in males. Its frequently found in

the head and neck region including other regions as well like genitourinary tract and extremities. Clinically, manifestations may vary from a small cutaneous nodule on the face to an extensive fast-growing facial swelling, which may, be painless or occasionally associated with pain, trismus, paresthesia, facial palsy, and nasal discharge. Most common oral site includes tongue followed by the soft palate, hard palate, and buccal mucosa. Depending on the site of occurrence the head and neck RMS is also divided into three major groups - a) eye and orbits. b) parameningeal. and c) non-orbital, non-parameningeal.[38,39]

Diagnosis

RMS is subdivided according to the international classification as embryonal, botryoid, alveolar, and pleomorphic (anaplastic). Histopathological pattern, cytogenic studies and Immunohistochemistry (IHC) may help in distinguishing between the three entities. Transmission electron microscopy is the gold standard diagnostic test. Other modalities like biopsy excisional and fine needle, CT, MRI may also be helpful.[4,40]

Differential diagnosis

A masseter mass associated with ipsilateral lymphadenopathy is typically consistent with an underlying infectious disease (e.g., epidemic parotitis) or a malignant neoplasm like non-Hodgkin's Lymphoma.[4]

Treatment

A combination of therapeutic approach involving, chemotherapy, surgery and radiation therapy are known to dramatically improve the survival rates.[39]

Movement disorders

Orofacial Dyskinesia/Tardive

It's an involuntary, repetitive stereotyped movement disorder characterised by severe, involuntary, dystonic movements of the facial, oral, and cervical musculature.[41,42]

Etiology

Drug therapy for the treatment of schizophrenia with phenothiazines and parkinsonism with L-dopa has been implicated as one among the cause, other causes includes, biochemical mechanisms along with presence of extrapyramidal disorder.[41,42]

Clinical features

In the early stages of the disorder, fine worm like movements of the tongue can be seen with typical presentation, consisting of tongue protrusion with licking of the lips, sucking and smacking movements, chewing movements, grunting and similar sounds, puffing of the cheeks, forehead furrowing and eye opening and closing. Risk majorly increases in elderly patients with diffuse brain pathosis. Most patients exhibits no specific complaints in early and mild cases. But with orofacial involvement it may lead to cosmetic disfigurement and significantly impair important functions like eating and speaking. It is worsened by anxiety or stress and intentional motor activity.[43]

Diagnosis

Is established by use of past and present use of any neuroleptic drugs, Common examples of such medications are: chlorpromazine (Largactil), haloperidol (Haldol), thioridazine (Mellaril), trifluoperazine (Stelazine), methotrimeprazine (Nozinan, Levoprome, Veractil), perphenazine (Trilafon), and pimozide (Orap).[43]

Differential Diagnosis

Habitual oral muscular activity secondary to ill-fitting dentures, Huntington's disease, and reversible drug induced chorea secondary to L-dopa (used in treatment of Parkinson's disease), amphetamine, or methylphenidate (Ritalin). Other extrapyramidal syndromes are acute dystonic reaction, drug induced parkinsonism and akathisia.[42]

Treatment

It's a reversible condition in most of the patients by discontinuation of the neuroleptic drugs. Reversibility likelihood is greater in young patients and increases with time.[42]

Oromandibular dystonia

Is characterized by involuntary repetitive muscular contraction and being a rare neuromuscular disorder, it affects different parts of the oromandibular region.[44]

Etiology

It is thought to originating from centrally mediated dysregulation of movement, arising as a defect in basal ganglia, especially in the sensory motor regions of the putamen, but still the mechanism of dystonia is not well understood. Due to the defect there is absence of physiological inhibitory control of the basal ganglia over the thalamus and brain stem, which can be demonstrated by long term use of neuroleptics.[44]

Clinical features

Usually seen in older females and may follow a long-term history of movement disorder. Presenting with clinical presentation ranging from abnormal perioral, tongue to oropharyngeal movements, along with dystonia of the neck muscles, eyelids, larynx, and muscles of facial expression. Functional disability may be considerable as Chewing, speech, swallowing, and facial expression is affected leading to malnutrition as well. Oral trauma includes rapid wear or early loss of teeth from persistent grinding, and biting of the tongue. Many patients can suffer from chronic depression and social isolation.[44]

Diagnosis

Based on information from the individual along with a neurological examination. In most situations routine laboratory tests are in the normal range.[45]

Differential diagnosis

Wilsons disease, organic brain injury, cervical spine lesion, stroke, non-reducing disk displacement without reduction.[44]

Treatment

Medications is the first line of therapy use of anti-cholinergic drugs, which would act by reducing muscle spasm, use of benzodiazepines , which would decreases monosynaptic and polysynaptic reflexes and serotonin dopamine receptors antagonists like clozapine and Risperidone which acts by binding to D2 receptors and may help improve dystonia in low dose, along with carbidopa/levodopa in low dose may help dopa responsive dystonia.[44]

Much improvement in symptoms is also yielded by use of BTX-A (botolinum toxin) into muscles of the base of the mouth, muscles of mastication, and extrinsic muscles of the tongue. It is injected using a short 30-gauge needle such as that used with a tuberculin syringe. The location and ease of access makes the masseter and temporalis muscles easy to inject.[11]

Masticatory muscle pain attributed to systemic/ central pain disorders

Fibromyalgia (fibrositis)

It is currently defined as presence of 11/18 tender points and chronic widespread pain (CWP) on examination. Previously it was referred to as fibrositis in the medical literature. As per the American College of Rheumatology, if a patient has perceived wide spread musculoskeletal pain in the four body quadrants for at least 3 months, he/she is suffering from fibromyalgia.[11,46,47]

Etiology

The descending inhibitory system, the hypothala -mic pituitary adrenal (HPA) axis, immune systems and an alteration in the processing of peripheral (musculoskeletal) input by the CNS has been implicated. Yet the etiology is not well – documented.[11]

Clinical features

It involves presentation of at least 11 of 18 designated tender points that do not produce heterotopic pain, patients usually presenting sedentary physical condition. Along with associated clinical characteristics like structural dysfunction, pain at rest, increased pain with function, weakness and fatigue along with presence of tender points.[11]

Diagnosis

Patients experiencing fibromyalgia usually reports with chronic and generalized musculoskeletal pain in numerous sites throughout the body and often present with a sedentary lifestyle accompanied by some degree of clinical depression thus reporting poor sleep quality.[11]

Differential diagnosis Treatment

Condition	Distinguishing Feature from Fibromyalgia
Rheumatoid arthritis	Joint swelling, deformities, elevated ESR, CRP
Systemic lupus erythematosus	Rash, multisystemic inflammation, elevated ESR, ANA
Polymagia rheumatica	Age 60 yrs, severe stiffness when inactive, elevated ESR
Myositis, myopathies	Weakness, elevated muscle enzymes
Ankylosing spondylitis	Back, neck immobility, elevated ESR, abnormal X-rays
Hypothyroidism	Abnormal thyroid function tests
Neuropathies	Weakness, loss of sensation, abnormal EMG, NCV[48]

It should be directed toward the causative and perpetuating factors and should be conservative. In case of associated masticatory muscle disorder, treatment must be directed towards these. Sleep disturbance should be addressed if it's present and use of Low dosages of a tricyclic antidepressant such as 10 to 50 mg of amitriptyline at bedtime can be helpful in reducing symptoms. Cyclobenzaprine (Flexril), 10 mg at bedtime, may also be helpful in assisting in sleep and reducing pain. Techniques such as moist heat, gentle massage, passive stretching, relaxation training, a mild and well controlled general exercise program such as walking or light swimming can also be used as a part of supportive therapy. Along with development of an individual program for each patient.[11]

Conclusion

Masticatory muscle disorders represent the second most common cause of orofacial pain after tooth pain. The treatment of a non-chronic MMP (*Masticatory Muscle Pain*) is simple and most patients achieve good pain relief with a simple conservative treatment. However, as soon as the pain becomes chronic a management approach according to the biopsychosocial model becomes mandatory in order to address simultaneously both the somatic and the non-somatic pain components.

Differential diagnosis also plays an important role since the same symptom (muscular pain) can be the result of different conditions, both local and systemic. The diagnosis must be mainly clinical and should be based upon recent diagnostic criteria.

So a detailed knowledge of various masticatory disorders is a necessity for accurate identification and diagnosis, so that needful can be adopted along with better treatment modalities for improving the quality of life of affected patients.

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