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Muscle Relaxants in Treating Tempromandibular Joint Disorder- An Update

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Abstract

To study about the muscle relaxants are useful in treating the TMJ disorders.Various mechanism behind the muscle relaxants like diazepam,metaxalone,alprazolam,carisprodol and cyclobenzaprine are useful in treating tempromandibular joint disorder.A muscle relaxant is a drug which affects skeletal muscle function and decreases the muscle tone. It may be used to alleviate symptoms such as muscle spasms, pain, and hyper reflexia. The term "muscle relaxant" is used to refer to two major therapeutic groups: neuromuscular blockers and spasmolytics. The muscle relaxants is used in treating tempromandibular disorder are carisprodol, cyclobenzaprine, diazepam, metaxalone, alprazolam and tizanidine. Diazepam appears to reduce spasticity by enhancing the inhibitory effects of neurotransmitter GABA. It also exerts some supraspinal sedative effect. Diazepam has efficacy in patients with spinal cord injury, hemiplegia, and multiple sclerosis. Carisoprodol is a carbamate derivative. Since, tempromandibular disorder is a common disease , this study is aimed to treat the tempromandibular disorder using the muscle relaxants and bring the overall awearness.

Key Words- muscle relaxants, tempromandibular joint disoder, diazepam, tizanidine, carisprodol, Cyclobenzaprine, metaxalone, alprazolam, hemiplegia and reflexia.

INTRODUCTION

Temporomandibular disorders (TMD) result from musculoskeletal dysfunction of the orofacial region affecting masticatory muscles, temporomandibular joints (TMJ), and other associated structures. The main characteristics of these problems are facial and TMJ pain, headache, earache, dizziness, masticatory muscle hypertrophy, limited mouth opening, locked jaw, abnormal teeth wear, joint sounds, and others (1). Dentist must be aware on the proper diagnosis and treatment of temporomandibular disorders, because they repre- sent the second most frequent patients complaints (only less frequent than dental pain) (2). Skeletal muscle relaxants are frequently used to treat these conditions. The muscle relaxants are believed to exert their action either by treating spas- ticity secondary to upper motor neuron syndromes, or muscular pain and spasms secondary to periph- eral musculoskeletal conditions (3). Drugs classified as skeletal muscle relaxants include baclofen, caris prodol, chlorzoxazone, cyclobenzaprine, dantrolene, metaxalone, methocarbamol, orphenadrine, and tizanidine. Some centrally acting muscle relaxants such as phenprobamate and mephenoxalone have antianxiety action that may be related in part to their action (4), these muscle relaxants are useful in treating tempro-mandibular disoders. TMJ disorders are common and often self-limited in the adult population. In epidemiologic studies, up to 75 percent of adults show at least one sign of joint dysfunction on examination and as many as one third have at least one symptom(5,6). However, only 5 percent of adults with TMJ symptoms require treatment and even fewer develop chronic or debilitat- ing symptoms(7).

SYMPTOMS OF TMD

Pain and tenderness on palpation in the muscles of mastication, or of the joint itself (preauricular pain – pain felt just in front of the ear). Pain is the defining feature of TMD and is usually aggravated by manipulation or function,(8) such as when chewing, clenching,(9) or yawning, and is often worse upon waking. The character of the pain is usually dull or aching, poorly localized,(10) and intermittent, although it can sometimes be constant. The pain is more usually unilateral (located on one side) rather than bilateral.(11)It is rarely severe.(12)

Limited range of mandibular movement, which may cause difficulty eating or even talking. There may be locking of the jaw, or stiffness in the jaw muscles and the joints, especially present upon waking.(13)) There may also be incoordination, asymmetry or deviation of mandibular movement.

PATHOGENSIS OF TEMPROMANDIBULAR JOINT DISODER

Inflammation mainly affects the posterior disc attachment (14,15). Several inflammatory mediators play an important role in the pathogenesis of TMJ disorders like tumor necrosis factor alpha (TNFalpha), interleukin-1beta (IL-1beta) , prostaglandin E2 (PGE2), leukotriene B4 (LkB4), matrix metalloproteinases (MMPs), serotonin- 5-hydroxytryptamine (5-HT).(16,17). MMPs are the early marker or detector to determine temporo mandibular joint arthritis(18). Serotonin is the mediator of pain and inflammation is produced in enterocromaffin cells of the gastrointestinal mucosa and absorbed by platelets. It is also produced in the synovial membrane and in the synovial fluid which causes TMJ pain in cases of systemic inflammatory joint diseases (19,20). Inflammation results in tissue response as : vasodilatation, extra vasation, release of mediators, activation of nociceptors, release of neuro peptides as substance P (SP), neuropeptide Y (NPY), which stimulate release of inflammatory mediators like histamine and serotonin and hyperalgesia.

MANAGEMENT OF TMD

Many therapies have been advocated for treating TMD, and many health professionals have found that they are able to help patients improve TMD symptoms. The practitioner managing the patient's therapy should decide which therapies are most cost-effective and evidence-based, and which have the greatest potential to provide the patient with long-term symptom relief. The most cost-effective therapies are the TMD self-management therapies, specifically when use is continuous and adhered to(21,22,23).

MUSCLE RELAXANTS

Muscle relaxants are commonly indicated for the treatment of two different types of conditions: spasticity from upper motor neuron syndromes and muscular pain or spasms from peripheral musculoskeletal diseases or injury such as low back pain. The muscle relaxants in current use have variable mechanisms of action, efficacy and adverse effects. This class of medications is well tolerated, with the most common side effects being drowsiness and nausea. The latency of the liver injury is variable(24).Muscle relaxants make up a heterogeneous group of drugs that mainly exert their pharmacologic effect centrally at the level of the spinal cord, the brainstem, or the cerebrum, and that have an insignificant, if any effect, at the muscle fiber level. Their centrally mediated mechanism of action can exert a clinically significant peripheral therapeutic effect.

MUSCLE RELAXANTS IN TREATING TMJ DISODERS

CYCLOBENZAPRINE

Cyclobenzaprine is probably the most commonly used muscle relaxant for nonspasticity-related muscle pain. Structurally, it resembles tricyclic antidepressants and differs from amitriptyline by only one double bond. Its therapeutic effect is centrally mediated and carries no direct peripheral action on the affected muscles. Its main pharmacologic action occurs at the brainstem and spinal cord levels and is partially explained by a depres- sant effect on the descending serotonergic neurons (25). It is extensively metabolized in the liver and excreted as a glucuronated metabolite through the kidneys. It possesses a fairly long half-life of approximately 18 hours and can continue to accumulate for up to 4 days when administered at a frequency of three times per day. Given its structural similarity to tricyclic anti- depressants as well as potent anticholinergic properties, caution should be exercised when considering its use in the elderly or in patients with heart dis- ease. Likewise, concomitant use with monoamine oxidase inhibitors is absolutely contraindicated because this combination can cause a hyperpyretic crisis or even death. The initial starting dose should be 5 mg three times per day on as needed basis and can be titrated up to 10 mg three times per day per therapeutic effect or side effect. In patients with hepatic or renal insufficiency, it should initially be administered once per day given its relatively long half-life. Of note, one recent study showed equal efficacy of 5 and 10 mg doses, with the smaller dose showing a lower level of sedation (26).The most common side effects are drowsiness, dry mouth, fatigue, and headache, followed by less often occurring adverse effects of diarrhea, dizziness, abdominal pain, nausea, nervousness, blurred vision, and confusion (27).

METAXALONE

Metaxalone is a centrally acting muscle relaxant with an unknown mech- anism of action. It is metabolized in the liver and excreted through the kidneys in the form of metabolites. Typical adult dosing consists of 800 mg three to four times per day. It is recommended to monitor liver function tests after initiation of this agent. Common side effects include drowsiness, dizziness, nervousness, nausea, and headache. The following rare but serious adverse reactions have been reported: leukopenia, hemolytic anemia, jaun- dice, and hypersensitivity reactions.

CARISPRODOL

Carisoprodol is still a commonly prescribed muscle relaxant that should be dispensed with caution owing to the potentially addictive properties of its main metabolite, meprobamate. Carisoprodol produces its muscle relaxant effect by depressing the interneuronal activity at the spinal cord level as well as in the descending tracts of the reticular formation. It is not recom- mended for use in the pediatric age population. This drug is metabolized in the liver with meprobamate as its main metabolite. It is mainly excreted through the kidneys. The usual adult dosage is 350 mg four times per day. The most common side effect is drowsiness. Other central nervous system adverse effects that have been reported include ataxia, agitation, insomnia, and others. Adverse effects such as tachycardia, postural hypotension, nau- sea, erythema multiforme, and eosinophilia have also been seen(28).

DIAZEPAM

Diazepam has commonly been used in the treatment of muscle spasm, especially in the acute setting. It belongs to a group of compounds called ben- zodiazepines, known for their potent anxiolytic, sedative, as well as muscle relaxant effects. Their main mechanism of action is through central potentiation of the inhibitory g-aminobutyric acid (GABA) effect through presynaptic facilitation of GABA release. Benzodiazepines are extensively metabolized in the liver into inactive and in some cases active metabolites. Compounds that lack active metabolites should be used as first-line agents in the elderly and in patients with liver or kidney insufficiency. Such com- pounds are lorazepam, clonazepam, temazepam, and oxazepam. Excretion principally occurs through the kidneys. Some of the common side effects are drowsiness, confusion, ataxia, cognitive impairment, memory loss, agitation, and disinhibition. Of note, withdrawal symptoms may occur after only 4 to 6 weeks of use, and their potential for abuse should be taken into consideration(29).

TIZANIDINE

Tizanidine is a centrally acting muscle relaxant that, through its alpha-2 adrenergic agonist properties, is thought to prevent the release of excitatory amino acids by

suppressing polysynaptic excitation of spinal cord interneurons. Metabolism is through the liver, and excretion is 60% through the kid- neys and 20% through the feces. Tizanidine should be administered through a gradual upward titration from an initial dose of 2 to 4 mg at bedtime up to the maximum of 8 mg three times per day. The bedtime dose can provide an analgesic effect as well as improve quality of sleep owing to the commonly occurring sedating side effect. Other common side effects are daytime drowsiness, hypotension, weakness, and dry mouth. Even though tizanidine's pharmacologic effect is similar to another alpha-2 agonist, clonidine, it possesses only a fraction of its blood pressure–lowering effect. Less commonly reported side effects of tizanidine are palpitations, bradycardia, dizziness, headache, nausea, elevated liver enzymes, and several rare cases of fulminant liver failure that led to death. Serial monitoring of liver enzymes is strongly recommended(30).

BACLOFEN

Structurally, baclofen is related to the centrally occurring inhibitory neurotransmitter GABA. Clinically, it has commonly been used for its muscle relaxant effects in the treatment of spasticity, as well as for its neuropathic analgesic properties in the treatment of trigeminal neuralgia pain. Baclofen is a GABA-B receptor agonist with presynaptic and postsynaptic effects leading to a decrease in the excitatory neurotransmitter release as well as in substance P, which is involved in transmission of nociceptive impulses (15). It is metabolized in the liver and excreted in the urine. Baclofen can be administered orally as well as intrathecally via an implanted pump mech- anism when significant adverse effects preclude further dose escalation to achieve therapeutic effect. Initial dosing of baclofen should be gradual, starting with 5 to 10 mg three times per day. The maximum recommended dose is 80 mg per day in divided doses; however, higher therapeutic doses in cases of refractory spasticity have been used without any significant untoward side effects. Common side effects are weakness, sedation, and dizziness. At higher doses, baclofen can cause seizures, ataxia, and halluci- nations. Abrupt withdrawal should be avoided because it can precipitate seizures and hallucinations.

CONCLUSION

Temporo mandibular disorders are frequent and wide spread in general population.TMJ internal derangement is most frequent type of TMD, and in characterised by several stages of dysfunction involving the condyle- disk relationship. The chief complaint is usually pain, which can manifest itself in different ways: head ache, jaw ache, ear ache, facial pain. In early stages of conditions, treatment may involve eating a soft diet and reducing strain on the jaw with the use of a splint or bite guard. Non steroidal anti-inflammatory drugs or muscle relaxants may be prescribed. Diazepam is the most commonly used muscle relaxant in tempromandibular joint disoders.

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