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Pain Management in Dentistry: A Review and Update

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Abstract

Pain management has progressed a great deal scientifically throughout the last century, in large part as a result of the introduction of the more effective pharmacologic agents and the developmental of a better understanding of the molecular principles that govern their use. There is still a great deal for researches and practitioners to learn about the mechanism and treatments for pain. This review article will discuss regarding the important aspects of the pain control in dentistry

Keywords: Dentistry; Pain; Management; Treatment

Introduction

Pain management is complex under taking; unfortunately we are just being to learn the rules of game. It is the patient's body that does the healing, not the doctor; the most that we can do is providing favorable condition for healing. Patient must understand that it is they, not we who determine final outcome of therapy.

First step in the treatment of any condition is accurate and complete diagnosis, what is problem, structures involved and condition account for it. The diagnosis of pain is pain genesis of modulating conditions of inhibitory and excitory mechanism of physical and emotional reactions that occur in human being as distinct individuals. Pain present to patient is peculiar to him or here we dentists are trained as therapists more than diagnostician's. Dental practitioners are conditioned to treat and our patients are conditioned to accept the treatment [1].

Dental practitioners are more mouth oriented than patient oriented. Dental practitioners do well as long as every thing is obvious when we face problems our condition may fail us. Because when we meet on obscure pain complaints we need to call forth all our best diagnostics capabilities.

Field of orofacial pain includes pain conditions that are associated with the hard and soft tissue of head, neck and all intraoral structures the diagnostic range includes headache, musculo skeletal pain, neurogenic psychogenesic pain, pain from major diseases like AIDS, TB, cancer etc the evaluation and treatment of orofacial pain has evolved into a shared responsibility between dentist and physician with considerable overlap that is distinguished only by the individuals knowledge and training [2].

Classification of Pain

Etiopathogenic classification of pain

A) **Pain due to local causes**

α. Pathologic changes in teeth and jaws

β. TMJ and associated muscles of mastication

χ. Nose and Para nasal diseases

δ. Oral mucosal diseases

ε. Lymph node diseases

ϕ. Salivary gland disease

γ. Diseases of blood vessels

B) **Pain along nerve trunk and central pathways**

α) Trigeminal neuralgia and glassophyrangial neuralgia

β) Migraine and other types of head aches

χ) A typical facial palsy

X) **Referred pain from other organs**

α) Cervical spondalities

β) Angina pectoris

χ) Orophyrangial diseases

δ) Diseases of ENT [3]

Principles of Pain Management

1) Pain therapy is begun with non-narcotic analogies for mild to moderate pain. If these drugs are ineffective intermediate potency opoids such as codeine or its derivatives are combined with them. NSAIDs are effective in many symptoms of bone associated pain are as cartico steroids they also decrease stiffness, selling and tenderness. Opoids and NSAIDs induce rapid change in pain sensation.

2) Treatment of acute pain requires location, origin and cause of pain. Management implies target short-term symptomatic relief; because the goal is to modify pain impulses during the period of tissue healing. NSAIDs can limit pain, swelling and erythema other agents given are muscle relaxants.

3) For severe or chronic pain analgesics are given at regular interval in adequate dose. Medication should never be prescribed as needed basis because pain will not resolve abruptly, oral medication is preferred, especially long acting, unless patients factor prohibit such.

4) Analgesics adjuvant such as tricyclic anti depressants are added to the drug regime if neural environment is suspected. Other adjuncts include anticonvulsants, antiarrthythmics, antihistamines, or phenothizines these agents usually require several days [2].

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Pain Control

One of the most important aspects of practice of dentistry is control or elimination of pain. In past pain has been seen closely associated with dentistry that the word pain and dentistry have become synonymus. Pain in m in past pain in many instances is considered as necessary element of every day living because it is a warning of trouble, in practice of dentistry we do not consider pain as a warning signal, but as an evil to be conquered [1].

Methods of pain control

Removing the cause: By removing the etiologic factor environmental changes would be eliminated, consequently free nerve ending would not be excited and no impulses would be initiated. It is imperivative that any removal leaves no permanent environmental changes in tissue, since this condition would be able to create the impulses even though the causative factor had been removed. This method clearly affects pain perceptions [4].

Blocking the pathways of painful impulses: This is the most widely used method during minor oral surgeries in dentistry. By this method a suitable, possessing local analgesic properties is injected in to tissue in proximity to the nerves involved. The local anesthetic solution prevents depolarization of nerve fibers at the area of absorption thus preventing fibers from conducting any impulses centrally beyond that point. As long as solution is present in the nerve in sufficient concentrate to prevent depolarization, the block will be in effect.

Pharmacotherapy of pain: This is a corner stone in the treatment of pain. Analgesic is the most frequently used in the treatment of pain. Several others classes of pharmaceutical agents are used for palliative and cause related therapy. Some drugs bind to known receptor, which are cellular components with which natural body chemicals (ligands) interact to produce physiologic responses. Some drugs (agonists) mimic rather closely the action of natural substance. Other prevents these actions, antagonists or blocking agents.

E.g., morphine (agonist) bind to certain receptor (mu and kappa) that normally interact with natural endorphins (ligands) this interaction is prevented by nalaxone (blocking agents).

The antidepressant drugs, L-Monoamine oxidases (MAO) inhibitor acts to increase concentration of serotonin, dopamine and non-epinephrine in CSF by educing the natural breakdown through an inhibitory action on the enzyme MAO.

Aspirin produces anti-inflammatory afferent by the action of enzyme cyclo-oxygenase, which normally metabolizes arachidonic acid into prostaglandin as a result of local injury.

Drugs give both intentional and intentional effects when two or more drugs are combined their interaction should be understood some combinations are incompatible. Some drugs potentate action of others to determine proper dose one should familiar with drugs half life and plasma concentrate.

Many things to be known about medications are: indications, contraindications, incompatibility, mode of action mode of administration, safety toxicity complications idiosyncrasy, anaphylaxis and other unwanted reactions [5].

Analgesic Agents

As a general rule the objective of analgesic should not be to eliminate pain altogether. Pain has some value in monitoring progress in patient's condition. It helps and guides the patients when his actions are excessive or abusive the main objective of analgesic is to make pain tolerable to the patient [6].

Types

Non-narcotic analgesics

Narcotic analgesics

Adjuvant analgesics

Non-narcotic analgesics

These include aspirin and NSAIDS, which have analgesic, anti-pyretic, anti-platelet, anti-inflammatory actions. They differ from narcotic analgesic in that they presumably prevent the formation of prostaglandin E1 by inhibitory action on enzyme, cyclo oxygenase. They don't produce tolerance, physical dependence or addiction they have ceiling effect where by increases dose beyond peak point dose not increase analgesic effect, but may effect duration of analgesic.

Acetaminophen and chloride magnesium tri-salicylate are included in non-narcotic group they lack anti platelet and inflammatory action. They are used in treatment of mild to moderate pain and chronic pain. Aspirin and NSAIDS are contraindicated with anticoagulant therapy and other coagulation deficiency condition [7].

Narcotic analgesic

Includes morphine and morphine like drugs they act by

1. Depressing nociceptive neurons while stimulate non-nociceptive cells.
2. It elevates the threshold for painful stimuli
3. It alters the emotional reaction to pain
4. It produces sleep which also elevates the threshold

Morphine inhibits release of bradykinin when mediated by neural mechanism.

Addition of codeine to NSAIDS increases analgesic effect.

Narcotics are useful in managing severe acute pain and a=chronic cancer pain but contraindicated in chronic orofacial pain. These causes constipation, there fore stool softener and laxatives should be administered. They should be administered at regular time schedule

Clinician should be watch full for

1. Tolerance =larger dose are required to obtain satisfactory analgesic effect.
2. Physical dependence= withdrawal symptoms severe the condition
3. Addiction=compulsive caring for the drug and the need to use it for effects other than pain relief.

These enhance the analgesic effect of other medications [8].

Adjuvant analgesic or have independent analgesic activity in certain situations. These include tricyclic antidepressants antihistamine, caffeine, steroids, phenothizines and anticonvulsants.

Anesthetic Agents

Used for diagnosis as well as pain management it can be used topically and in injection form.

Topical

These are available as solution, spray, and ointments. Water-soluble ointment containing topical anesthetic and germicide used for managing dental Alveolitis.

Analgesic balms are agents that give soothing palliative relief of inflammatory pain of superficial deep categories when applied locally to exposed tissue.

Aloe Vera juice is an ancient remedy for superficial pain; balsam of pura; eugenol and guaiacol are other well-known balms. These are useful in controlling of pain from exposed/ulcerated cuteness and mucogingival tissue, exposed dentin and acute alveolitis [9,10].

Injectable local anesthetic (LA)

Varity of LA is available in different concentration with or without vasoconstrictor. Long acting LA such as bupivacine HCl, (marecine) are useful, even though they have higher risk of toxicity proper dosage, technique adequate precaution readiness of emergency are essential for safety and effectiveness of all LA.

Extreme causation is required when vassopressor agents are used in patients receiving MAO inhibitor or anti-depressant of triptyline type because severe prolonged hypertension may result therefore most unwanted reaction with LA is critical intravenous injection.

Injection of 0.02% morphine sulphate around peripheral nerve has been repeated to yield LA that is equal to bupivacine in onset and duration small dose of one mg. or less of morphine does not produce systemic effect [11].

Anti-inflammatory agents

In addition to anti-inflammatory analgesics there are several nonsteroidal medication that are mainly for their anti-inflammatory effect they are mild analgesic and antipyretic acts by inhibition of prostaglandin's biosynthesis. They do not alter disease but suppress symptoms of inflammation.

Corticosteroids exert potent anti-inflammatory effect by inhibiting prostaglandin biosynthesis; their suppressive effect on inflammation may mask infection. These are contraindicated in systemic fungal infection Herpes simplex infection [1].

Muscle relaxants

These are used to controlle myogenous pain. The muscle relaxants are the anti cholinergic the examples are succinyl choline and methocarbamol. The disadvantage is that patient cannot remain ambulatory or safety to continue his usual activity, can be used for hospitalized patients under supervision [12].

Antidepressants

Tricyclic anti depressant agents increases the avalibity of serotonin and non-epinephrine in the CSF. The dimethylated tricyclic drugs make serotonin proportionally more available and induce some sedative effect. The mono methylated tricyclics makes non-epinephrine proportionally more available and induces some CNS stimulation. It has been demonstrated that low dose of amitriptyline 10 mg. just before sleep can have analgesic effects on chronic pain after several weeks of use.

MAO inhibitor increases the available serotonin, dopamine, non-epinephrine in CSF by inhibiting their breakdown and may induce hypertension crisis. Therefore use of all anti depressant should be under adequate medical supervision.

Many sedatives and tranquilizing agents are available some, of which are having the muscle relaxants action. Major tranquilizers like phenothizines are use full in pain controlle by decreasing modulating effect on anxiety and apprehension. Major tranquilizers such as meprobromate and diazepam have advantage of fewer side effects. There muscle relaxation is useful but drug tolerance, dependence and addiction are common side effects occurring. If these are used it should be prescribed for limited period or different drugs should be used periodically [13].

Vocative agents

Neurovascular pain may be influenced by alpha androgenic blocking action of ergotamine tartrate, which causes stimulating effect on smooth muscles of peripheral and cranial vessels. It is available with or without addition of caffeine. Caffeine enhances vaso-constricting effect

Vocative agents are contraindicated in coronary heart diseases, pregnancy and increased blood pressure [14].

Non-epinephrine blockers

Guanethiine and reserpine appears to block the uptake of non-epinephrine by sensitized axons used in treatment of orofacial pain by blocking satellite ganglion

These are commonly used in rheumatoid arthritis [15].

Antimicrobials

These are introduced systemically after culture and sensitivity. This decreases pain by decreasing the inflammation that is their enzymes and byproducts [16].

Antiviral agents

Acyclovier and Vidarabine used in HSV1 and HSV2. These are also effect against Varicella Zoster virus that causes herpes zoster [17].

Antihistamines

Counteract vasodilating action of histamine by blocking histamine receptor, useful in allergic responses in Neurovascular pain [18].

Anticonvulsive agents

Phenotoin sodium is an anticonvulsant and is capable of suppressing pain in about 20% of paroxysmal neuralgia carbamazapine (tegretol) gives pain relief in 70% of trigeminal and glassophyrangial neuralgia [19].

Neurolytic agents

95% of ethyl alcohol is used to destroy peripheral nerve. Sometimes phenol is added to, it provides long-term temporary relief. The disadvantages are local fibrosis, not used commonly. Injection of 0.3 ml of glycerol into retrogasserion space for treatment of trigeminal neuralgia reported that 90% patient pain free. No dysesthesia or anesthesia, dolorosa observed, glycerol acts on demylinated axons assumed to be involved in triggering neuralgia [20].

Uricosuric agents

Probenecid is used in the treatment of gout. It is a uricosuric and renal blocking agent that inhibits reabsorption of urates in the tubules of kidney. But increase excretion of uric acid' the serum urate level is lowered. It induces exacerbations of acute gout. Acute symptoms are

controlled by colchicines and therefore the combination is used in chronic gout [21].

Diet

L tryopthan an amino acid is main dietary supplement brain and spinal cord serotoninergic neurons are actively in nociceptive receptors as well as in analgesic effect of opiates. Increased activity of serotonin inter neuron is associated with analgesia and enhanced drug potency. Case was reported where pain relief was not possible even after 30 mg of intravenous morphine. But pain is controlled by 4 grams of L tryptophan per day for several weeks.

Adequate dosage: [22]

1. L tryptophan 4 grams of per day

2. Low protein, low fat, high carbohydrate

3. Vitamin B-6 10-25 mg/day

4. Four weeks or more continuous therapy is required.

Physical Therapy

Modalities

This is done by an instrument or device these are sensory stimulants ultrasound, electrogalvenic stimulation (ECG) and deep heat [23].

Sensory stimulation

1. Cutaneous stimulation
2. Transcutaneous stimulation
3. Percutaneous stimulation

Cutaneous stimulation

Stimulation of skin is used for pain control from ancient time. The effect occurs through stimulation of thick myelinated cutaneous afferent, A-beta neurons chiefly.

Different forms-Pressing/Rubbing skin directly over lesion and also by adding stimulating substances like alcohol menthol ointment.

Use of mechanical vibrator reduces pain in one third of patients.

Hydrotherapy-direct spray of water over lesion also reduces pain [24,25].

Conclusion

Through rational drug-prescribing habits and education of both patients and caregivers, effective regimes can be designed to increase pain control while decreasing untoward drug side effects. A lucid understanding of pharmacology and drug actions as invaluable because knowledge of real or potential drug interactions can assist in designing regimes that will be most use full in treating patients with acute or chronic debilitating pain syndromes.

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