The Journal of Dental Panacea 2022;4(3):106–111

Content available at: https://www.ipinnovative.com/open-access-journals

The Journal of Dental Panacea

Journal homepage: https://www.jdentalpanacea.org/

Review Article

The management of dental pain using pharmaceutical drugs-A review

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ARTICLE INFO

Article history:
Received 25-05-2022
Accepted 10-06-2022
Available online 30-09-2022

Keywords:
Dental pain
analgesics
Pharmaceutical management
NSAIDS
Opioids

ABSTRACT

Pain is an undesirable sensation that any person would never want to experience. Dental pain is unbearable as the inflammation of the dental pulp is encased by hard dentin and cementum unlike other parts of the body and that deteriorates the normal lifestyle of the person experiencing it. Patients experience extreme pain till the commencement of any dental treatment. Though the right treatment procedure relieves the cause of the pain, post-operative pain exists till the complete healing occurs. This proves to us the importance of dental analgesics. There are many analgesics available and not everything will be appropriate. Proper selection of analgesics helps in the betterment of the patient's status. This article reviews the commonly available analgesics and their pharmacological aspects which help in managing dental pain.

1. Introduction

Pain is a subjective symptom that indicates a need to act quickly, and it is frequently coupled with other subjective feelings like fear, anger, and discomfort. Different patient-related variables influence the nature and degree of pain expression. Gender, age, physiological parameters, substance misuse history, neuropathic and other disease, and psychological profile of individual people are all elements that influence the patient's interpretation of pain.[1,2] "A painful sensory and emotional experience related with, or mimicking, actual or potential tissue injury," according to the International Association for the Study of Pain.

Following various interventional procedures and dental illnesses, dental pain (toothache or odontalgia) is a typical subjective complaint of dental patients. Dental discomfort is one of the most prevalent reasons for people seeking emergency dental care in the United States (about 12 %).[3] Odontogenic pain is a multi-step process that begins with dental tissue destruction and is accompanied by a variety of neural stimuli as a result of neurovascular, neuroinflammation, and morphologic reactions.[2,4]

When it comes to prescription rates, clinical efficacy, cost-effectiveness, and safety profile, analgesics are one of the most important pharmacological classes in dentistry practise. There are various approaches to developing treatment algorithms and guidelines for dental pain therapy based on this amount of priority in dental clinical practise. Nonetheless, prescribing analgesic medicines for dental indications is fraught with difficulties, lowering therapeutic success and raising the risk of major side effects.[2]

In this review, the management of dental pain using pharmaceutical drugs is explained.

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https://doi.org/10.18231/j.jdp.2022.022

1.1. Odontogenic pain

Pain caused in tooth region is known as Odontogenic pain. It is caused mainly because of the following causes:[5]

1. Pulpal Dentinal hypersensitivity Pulp disease (pulpitis)
2. Pathology in periapical region (acute abscess)
3. Gum/periodontal disease
4. Cracked tooth syndrome
5. Dental trauma

2. Classification of Drugs

Classification of analgesics is broadly classified as Opioid analgesics and Non-opioid and non-steroidal (NSAIDS)

*2.1. **Opioid analgesics:**[6,7]*

1. Natural opium alkaloids:
   (a) Morphine
   (b) Codeine
2. Semisynthetic opiates:
   (a) Diacetylmorphine (Heroin)
   (b) Pholcodeine
3. Synthetic opioids:
   (a) Pethidine (Meperidine)
   (b) Fentanyl, Alfentanil, Sufentanil, Remifentanil
   (c) Methadone
   (d) Dextropropoxyphene
   (e) Tramadol

2.2. Non-opioid and non-steroidal analgesics (NSAIDS):[8]

2.2.1. Non selective COX inhibitors

1. Salicylates:
   (a) Aspirin
2. Propionic acid derivatives:
   (a) Ibuprofen
   (b) Naproxen
   (c) Ketoprofen
   (d) Flurbiprofen
3. Fenamate:
   (a) Mephenamic acid
4. Enolic acid derivatives:
   (a) Piroxicam
   (b) Tenoxicam
5. Acetic acid derivatives:
   (a) Ketorolac
   (b) Indomethacin
   (c) Nabumetone
6. Pyrazolone derivatives:
   (a) Phenylbutazone
   (b) Oxyphenylbutazone
7. Selective COX-2 inhibitors:
   (a) Celecoxib
   (b) Etoricoxib
   (c) Parecoxib
8. Preferential COX-2 inhibitors:
   (a) Nimesulide
   (b) Diclofenac
   (c) Aceclofenac
   (d) Meloxicam
   (e) Etodolac

2.3. Analgesic - antipyretics with poor anti-inflammatory action

1. Para aminophenol derivative:
   (a) Paracetamol (Acetaminophen)
2. Pyrazolone derivatives:
   (a) Metamizole((Dipyrone)
   (b) Propyphenazone
3. Benzoxazocine derivative:
   (a) Nefopam

3. MOA of Analgesics

3.1. NSAIDS

The cyclo-oxygenase enzyme has two structurally different variants (COX-l and COX-2). COX-1 is a naturally occurring protein in normal cells, whereas COX-2 is increased in inflammatory cells. The most plausible mechanism of action for NSAID-mediated analgesia is inhibition of COX-2 activity. Some NSAIDs block the lipoxygenase pathway, which can lead to allogenic metabolites being produced. NSAIDs' interference with G-protein-mediated signal transduction could be the basis of an analgesic mechanism unrelated to prostaglandin production inhibition. There's a growing body of evidence that NSAIDs have a central mechanism of action that works in tandem with the peripheral mechanism. This impact could be caused by interfering with prostaglandin production in the CNS. Endogenous opioid peptides or inhibition of serotonin release could also be involved in the central action (5-hydroxytryptamine; 5-HT). A mechanism involving excitatory amino acid inhibition or activation of N-methyl-D-aspartate receptors has also been proposed.[9]

3.2. Opioids

The most often prescribed painkillers are opioids and nonsteroidal anti-inflammatory medications (NSAIDs). By engaging with mu, delta, or kappa opioid receptors, opioids mimic the activities of endogenous opioid peptides. The opioid receptors are linked to G1 proteins, therefore opioids have primarily inhibitory effects. They open calcium-dependent inwardly-rectifying potassium channels while closing N-type voltage-operated calcium channels. Hyperpolarization and a decrease in neural excitability ensue as a result of this. They also reduce intracellular cAMP, which regulates nociceptive neurotransmitter release (e.g. substance P). The main mechanism of analgesic and anti-inflammatory effects of NSAIDs is inhibition of prostaglandin synthesis by cyclooxygenase. Aspirin inhibits cyclo-oxygenase irreversibly, while other NSAIDs inhibit it reversibly.[10]

3.3. Commonly used drugs for dental pain:[11]

Acetaminophen is an antipyretic analgesic used to treat mild to moderate pain. It has no anti-inflammatory properties. Excessive doses can cause permanent liver damage, while long-term use can cause kidney toxicity. Paracetamol is a good example.

NSAIDs - Have analgesic, antipyretic, and anti-inflammatory properties. NSAIDs have been shown to be effective in the treatment of a variety of dental pains. Nonselective NSAIDs inhibit both COX-1 and COX-2, resulting in GI bleeding and ulceration. Aspirin and ibuprofen are two examples.

Opioids - Opioid analgesia is caused by opioid receptor activation, which inhibits neuronal activity. In the treatment of acute dental pain, opioids are usually coupled with acetaminophen. Morphine and tramadol are two examples.

3.4. Factors affecting selection of analgesics for dental pain:[2,12]

The selection of analgesic medicines in the treatment of dental pain is influenced by a number of factors, including:

1. Mechanism of pathophysiological pain: This is a determining element in analgesic selection. Cancer metastases, postoperative dental pain, nerve root infiltration, neuropathic pain, and other mechanisms are among them.
2. The age of the patient: The choice of analgesic is also influenced by the patient's age. Analgesics are administered differently in youngsters and the elderly than in adults. Due to immature metabolism processes, the use of a number of analgesics in children is restricted. Due to diminished potential of metabolism and/or excretion, which reflects in pharmacokinetics and pharmacodynamics of medications, the aged frequently require a dose restriction of analgesics.
3. **Administration route:** The analgesic's bioavailability and pharmaceutical formulation are determined by the patient's overall health, disease features, and bioavailability. Analgesics should be taken orally whenever possible. Pharmaceutical formulations with controlled release are better for chronic pain than those with rapid release**.**
4. **Patients-related features.** Several factors can influence the effectiveness of analgesic treatment in dental patients. Dental physicians should think about the placebo effect carefully. The potential renal and hepatic toxic effects, as well as gastrointestinal disturbances that may affect the pharmacokinetic and safety profile, should be addressed first by dental experts.

4. Management of Dental pain in Adults

In patients who are adults, Pain management in dentistry is usually a surprise and is largely dependent on the particular patient's sensitivity to pain, the patient's expectations, the pathophysiological mechanism of pain, and the choice of analgesic medicines. Pain reduction is a critical precondition for interventional dentistry therapy because it establishes a trusting and comfortable relationship between patients and their dentist.[13] Almost all dental operations are accompanied by pain of varying severity, nature, and duration, and pain management is an important element of dental treatment, whether it is done before or after the surgery.[14] In order to achieve a good clinical outcome and a successful dental clinical therapy, effective pain treatment is required during dentistry healthcare. The use of local anaesthetic ensures the control of patient discomfort during the patient preparation phase, prior to the commencement of dentistry procedures.[15] Clinical evidence demonstrates that local anaesthetic relieves pain during the intraoperative dental phase and quickly relieves postoperative pain, therefore dentists should consider appropriate pain management at all stages of dental treatment. Due to prostaglandins' interaction with tetrodotoxin-resistant receptors, which lowers nerve responses to local anaesthesia, the effect of local anaesthetic is lessened in the oral pathological process.[16] Dental doctors should focus on the disease, the patient, and lastly the various nonpharmacological and pharmacologically effective therapy choices for effective dental pain management. To comprehend the cause of disease and anticipate the patient's health status, the dental practitioner should first analyse the pathological process of dental tissue. Determining the genesis of the disease process, particularly the final inflammatory response, is critical.[17] Premedication with NSAIDs such as ibuprofen or indomethacin has been shown to considerably increase the level of alveolar nerve block anaesthesia in dental

treatments (78 %and 62 %, respectively) when compared to placebo (32 %).[18] A pain response develops throughout the healing process of soft tissue injuries, necessitating pain treatment techniques. Preoperative administration of medication, including analgesic drugs, is suggested in dental surgical procedures to reduce postoperative discomfort and the need for postoperative painkiller. 118 Analgesics to Alternative Therapies for Pain Relief The dynamic process of creating a logical treatment map, which is built by the methodology of conceptualization to visualise the relationship between patient symptoms, dental interventions, therapeutic treatment, and the patient's needs and expectations, is the foundation of an effective dental pain treatment strategy. Furthermore, there is misinformation accessible claiming that naproxen sodium has higher analgesic efficacy to ibuprofen at post-dose intervals of 1 to 12 hours.[19–21] Para-aminophenol derivatives, such as paracetamol, are useful analgesics for use in dentistry (acetaminophen). Individual paracetamol administration is only suggested in moderate cases of dental pain when NSAIDs are contraindicated. Otherwise, clinical evidence suggests that ibuprofen 200–512 mg is preferable to paracetamol 600–1000 mg in reducing postoperative pain. The usage of a combination of ibuprofen and paracetamol as a pain reliever is a novel method. When taken 6 hours after dental intervention, this combination is more effective than solo analgesics. The most common doses of respective analgesics administered in clinical practise are 400 mg for ibuprofen and 1000 mg for paracetamol, according to the data.[22] When solo NSAID analgesics or a combination of NSAID and paracetamol are ineffective, a combination of opioids and NSAIDs is indicated. The analgesic impact of this medication combination is greater than twice the dose of either analgesic when taken separately.[2,23]

5. Management of Dental Pain in Children

Effective pain management is a normal routine method in clinical paediatric care and is required in the modern paradigm of health care. It is widely believed that the underlying mechanism of pain in infants and children is very similar to that in adults, with the exception of neonates, who have certain physiological changes in pain, such as slower and less precise pain conduction but no major abnormalities in pain perception.[24] Over the previous two decades, modern pain management for children has made significant progress in terms of medical problems, surgical interventions, and the postoperative period. The interventional pharmacological and nonpharmacological approaches to advanced pain treatment are the two primary avenues. NSAIDs and other analgesics administered via various routes of administration (i.v. bolus administration, continuous infusion, rectal, transdermal, and other routes of administration); local anaesthetics, epidural anaesthesia, and peripheral nerve blockade are all part of the interventional pharmacological approach. Breathing techniques, hypnosis, transcutaneous electrical nerve stimulation, guided imagery, acupuncture, relaxation, and other nonpharmacological measures include health education for children and psychological approaches to release the perception of fear and other behavioural problems in children patients, as well as breathing techniques, hypnosis, transcutaneous electrical nerve stimulation, guided imagery, acupuncture, relaxation, and other techniques to relieve pain.[25] Children's pain management strategies are based on numerous principles that reflect the distinctions in pain treatment between children and adults. Preventing pain should be the goal of pain relief strategies, as this provides higher treatment success before invasive procedures. Typically, this begins with pre-planning the kid and family in order to lessen fear and anxiety before to intervention, as well as the use of patient-controlled analgesia (PCA). In the case of significant surgical procedures, the treatment of anticipated pain in children can be continued with oral analgesics, depending on the patient's needs.[2]

6. Significant Drug Interactions of Analgesics

When NSAIDs are used with the anticoagulant and antiplatelet effects of warfarin and clopidrogel, substantial interactions occur, resulting in enhanced effects and an increased risk of bleeding. Acetaminophen is an appropriate choice in this case, but only at the lowest feasible dose and for short-term treatment. In contrast to diclofenac or acetaminophen, which had no effect on aspirin's antiplatelet activity, ibuprofen use in patients taking cardio protective aspirin did not interfere with its antiplatelet activity, despite studies showing reduced cardio protective benefits and increased gastrointestinal risk, in contrast to diclofenac or acetaminophen, which had no effect on aspirin's antiplatelet activity.[26] Furthermore, individuals receiving daily aspirin for cardiovascular disease prevention should avoid chronic ibuprofen usage, and the FDA recommends taking ibuprofen at least 8 hours before or after the immediate release of aspirin to avoid potential platelet function interactions.[27] The use of NSAIDs in combination with warfarin or corticosteroids may increase the risk of gastrointestinal bleeding. When used in conjunction with biphosphonates, they also increase the risk of gastrointestinal ulcers. Coadministration of NSAIDs enhances the effects of antidiabetic sulfonylureas. With the use of NSAIDs, there is a decrease in methotrexate renal extraction, which can lead to toxicity. Lithium serum concentrations are also elevated, necessitating the use of a non-NSAID analgesic. Fluconazole has also been proven to boost celecoxib levels by inhibiting its metabolism. NSAIDs interact with ACE inhibitors, diuretics, Ca-channel beta Blockers, and beta-blockers, resulting in reduced antihypertensive effects.

Short-term use does not represent a significant risk in healthy people, but if the medication is to be continued for a long time in hypertensive patients, especially the elderly, cautious selection and constant monitoring is essential. Antacids have been proven to reduce the effects of NSAIDs. In addition, NSAIDs have been observed to interact with SSRIs (selective serotonin reuptake inhibitors) to increase the risk of bleeding, especially upper gastrointestinal and postoperative bleeding.[28,29] There are extremely few medication interactions with acetaminophen. As a metabolic inducer, carbamazepine may lower acetaminophen drug levels. When used with alcohol or medicines that affect the liver, the risk of liver toxicity increases. Dental practitioners should be aware of these interactions and utilise analgesic medication therapy in carefully researched combinations and within the dosage or interval limits. They should also avoid them when there is a higher risk of toxicity.[30,31] Antipsychotics (phenothiazines), which improve their hypotensive impact, and CYP2D6 inhibitors (cimetidine, chlorpheniramine, fluoxetine, and quinidine), which reduce their effects, including hydrocodone, are examples of narcotic analgesic interactions. When administered with morphine, oxycodone, or methadone, inhibitors or inducers of CYP3A4 generate clinically relevant interactions by mediating opioid toxicity or impairing pain therapy. Furthermore, the effects of SSRIs and MAOIs are more closely linked to meperidine, methadone, tramadol, buprenorphine, oxycodone, hydrocodone, pentazocine, and fentanyl, all of which may contribute to the serotonin syndrome. Barbiturates may augment the sedative effects of other sedatives. Phenytoin also causes an increase in meperidine metabolism. Taking this into account, clinicians should be aware of and monitor patients for drug interactions, as well as strive to avoid polypharmacy if possible.[2,32–34]

7. Conclusion

Prescribing an analgesic for dental pain is a challenge indeed. The dentist should understand the origin of the dental pain by taking detailed case history and the medical history of the patient should be known to prevent drug interactions. In the aspect of drug, one should know the mechanism of action of the drug and the duration of action (half -life). Age of the patient should be considered for proper dosage. If the above factors are taken care of, dental pain relief of the patient will be achieved successfully.

8. Source of Funding

None.

9. Conflict of Interest

None.

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Cite this article: Sophia K, Sebastian SM, Linda Christabel. S, Merrylda Claribel. S, Devi MS. The management of dental pain using pharmaceutical drugs-A review. *J Dent Panacea* 2022;4(3):106-111.