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The role of NSAIDs in implant related inflammation: A Scoping Review

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Abstract

Objective: Non-steroidal anti-inflammatory drugs (NSAIDs) are widely utilised in implant surgery for their analgesic, anti-inflammatory, and antipyretic properties. Their role is pivotal in postoperative pain management and inflammation control, which are critical to patient comfort and surgical outcomes. However, their impact in osseointegration is complex. This review examines the role of NSAIDs in implant surgery: as effective agents for preoperative symptom management and as possible modulator of bone metabolism.

Introduction: Pharmacology is concerned with the study of action of drugs and is a branch that comes under biology and medicine. The two chief concerns are the effect of the drugs on the human system and the effect of the human systems on the ability and metabolism of drugs. NSAIDs exert their effect by inhibiting cyclooxygenase (COX) enzymes, primarily COX-1 and COX-2, thereby reducing prostaglandin synthesis (group of lipid compounds having diverse physiological roles within the body). While this action effectively controls pain and inflammation, it may also influence bone healing. Prostaglandins, particularly PGE_2, play a key role in bone remodelling and osseointegration (process where dental or orthopaedics implant fuses directly with the surrounding living bone) by modulating osteoblast and osteoclast activity. Inhibition of COX-2 during the early healing phase may impair osseointegration, potentially affecting implant stability. Therefore, the use of NSAIDs in implant surgery must be carefully balanced to optimise both analgesia and bone healing.

Inclusion Criteria: Population: Patient going implant surgery, Concept: Comparison of two groups of patients- with NSAIDs and without NSAIDs post dental implant surgery, Context: Manav Rachna Dental College.

Method: Google Scholar, PubMed, databases were being searched.

Conclusion: NSAIDs play a key role in postoperative pain control; however, their impact on osseointegration varies. Selective COX-2 inhibitors may impair bone healing, while short-term use of non-selective NSAIDs appears safer. Careful selection and timing of NSAID therapy are essential to support successful implant outcomes.

Keywords: Analgesics, Anti-inflammatory agents, Non- steroidal, Cyclooxygenase inhibitors, Dental implants, Osseointegration, Prostaglandins.

Introduction

Pharmacology is the branch of biomedical science that explores the interactions between[1] chemical substances and living systems, with the primary goal of understanding how drugs produce therapeutic and adverse effects. It encompasses the study of drug composition, properties, mechanisms of action, pharmacokinetics.

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) are among the most widely prescribed and over-the-counter medications used globally for their anti-inflammatory, analgesic, and antipyretic properties[1]. They play a pivotal role in the management of a variety of conditions involving pain and inflammation, such as arthritis, dental pain, postoperative inflammation, and fever. NSAIDs exert their therapeutic effects primarily through the inhibition of cyclooxygenase (COX) enzymes, which are responsible for the conversion of arachidonic acid into prostaglandins, prostacyclin, and thromboxane (bioactive lipid compounds) that mediate inflammation, pain, and fever. Implant surgery, whether in dental, orthopaedic, or reconstructive fields, is inevitably associated with acute inflammation due to tissue trauma, foreign body response, and prosthetic integration processes. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) are routinely prescribed after implant surgeries (dental, orthopaedic, or cosmetic) to manage inflammation, pain, and swelling that result from surgical trauma. However, NSAIDs can also inhibit the production of PGs at the site of implant placement or fracture, thereby

influencing the bone healing cascade. Given these mixed results, it is important to further investigate how NSAID use influences bone healing and osseointegration after dental implant placement[1].

COX-1 and COX-2 are responsible for converting arachidonic acid, a component of cell membranes, into prostaglandins found in all cells and tissues. The prostaglandins formed under the influence of COX cause sensibilization of nociceptors, increased platelet aggregation, increased tension of uterine muscles, and reduced gastric juice secretion[4]. COX-1 is a constitutive enzyme that is continuously expressed in the endothelium of blood vessels, platelets, the gastric lining, and the kidneys. It contributes to vasodilation and inhibits platelet aggregation by influencing prostacyclin (PGI_2) synthesis, some of the examples of COX-1 NSAIDs; Aspirin, Indomethacin, Naproxen etc[4]. COX-2, on the other hand, is an inducible enzyme. COX-2 plays a key role in promoting vascular permeability and contributes to the development of oedema, as well as the intensification of inflammatory responses and pain[9]. Its expression is unregulated in response to pro-inflammatory cytokines acting through specific receptors, leading to the increased synthesis of prostaglandin E (PGE_2), examples of COX-2 NSAIDs; Celecoxib, Etoricoxib etc. This prostaglandin functions as a critical mediator, often referred to as the "final transmitter," in both febrile and inflammatory processes[4].

Prostaglandins (PGs) produced by bone cells exhibit dual roles in bone metabolism, exerting either stimulatory or resorptive effects depending on the cellular context. In fully differentiated osteoblasts and osteoclasts, PGs tend to have an inhibitory influence. These inhibitory PGs are primarily generated following the induction of cyclooxygenase-2 (COX-2), which is recognized as a key regulator in the differentiation of mesenchymal stem cells into the osteoblastic lineage and plays a critical role in bone repair processes[3]. Non-steroidal anti-inflammatory drugs (NSAIDs) have been shown to disrupt fracture healing and hinder new bone formation, notably reducing bone ingrowth into porous-coated joint implants, thereby impairing osseointegration and the long-term stability of these prosthetic devices[3]. Hypoxic conditions are commonly observed in bone tissue following pathological events such as implant placement and fractures. Evidence from clinical studies on bone cultures indicates that hypoxia plays a key regulatory role in stimulating prostaglandin E (PGE) synthesis by osteoblasts. Consequently, the functional presence of cyclooxygenase (COX) enzymes is considered essential in mediating the inflammatory and reparative processes associated with bone healing[1]. To understand the potential influence of NSAIDs on osseointegration, it is essential to first examine the biological processes involved in bone healing. The initial phase of bone repair is marked by inflammation. At the fracture site, a blood clot forms and leukocytes begin to accumulate within the first 6–8 hours. Inflammatory cells, especially platelets, release numerous cytokines and growth factors, including bone morphogenetic proteins (BMPs), platelet-derived growth factor (PDGF), and fibroblast growth factor (FGF). Over the first 10 days, the clot organizes into granulation tissue and neovascularization occurs. During this inflammatory phase, COX-2 expression is upregulated in affected tissues. Prostaglandins produced endogenously play a critical role in bone metabolism by regulating the balance between bone formation and resorption during healing[2].

Medical literature indicates that use of NSAIDs during the healing phase post-implant placement may hinder bone regeneration. However, results from experimental models involving periodontitis suggest varied outcomes. Research related to periodontitis in humans shows that NSAIDs might either decelerate alveolar bone loss or have no noticeable effect. Given these mixed results, it is important to further investigate how NSAID use influences bone healing and osseointegration after dental implant placement. Most in vivo animal studies show that NSAIDs hinder bone healing and osseointegration around titanium implants[3]. Meloxicam reduced bone formation and implant-bone contact in rats, while a short-term regimen of diclofenac and parecoxib in rabbits showed no negative effects—though parecoxib was given intravenously. Studies in COX-2 knockout mice revealed impaired bone formation, highlighting COX-2's role in healing. Diclofenac also delayed bone healing in rats compared to meloxicam and controls[3]. Studies on NSAID use after implant placement show mixed outcomes. Flurbiprofen at 100 mg reduced bone loss compared to lower doses and placebo, particularly after implant loading. However, short-term ibuprofen use did not significantly affect marginal bone levels at 3 or 6 months. In contrast, celecoxib, a selective COX-2 inhibitor, was associated with increased bone loss and possible implant failure. These findings suggest that while some NSAIDs may be safe, selective COX-2 inhibitors could impair bone healing and osseointegration in susceptible patients[3]. Therefore, the rationale of this scoping review aims to compare the post-operative outcomes of patients who receive NSAIDs with those who do not, to better understand the implications of NSAID use on pain control, healing, and implant success. The findings are expected to form clinical decision-making and contribute to the development of evidence-based guidelines for post-implant pharmacological management.

Review Question

To evaluate whether NSAIDs interfere with the bone remodelling/ osseointegration process post dental implant placement.

Inclusion Criteria

Population: Patients going dental implant surgery.

Concept: Comparison of patients with NSAIDs and without NSAIDs post implant surgery.

Context: Manav Rachna Dental College.

Types Of Sources

Numerous experimental and quasi-experimental study designs, systematic review, original research, were considered in this scoping review. Additionally, prospective and retrospective cohort studies, analytical cross-sectional studies, case-control studies, and other analytical observational research were taken into consideration for inclusion.

Search Strategy

In this review, a three-step search approach was applied. To find pertinent literature on the subject, a preliminary search of Google Scholar and MEDLINE (PubMed) was conducted. The index keywords used to find the articles and the key phrases in the titles and abstracts of pertinent publications were combined to create a thorough search strategy.

Study Or Source Of Evidence Selection

After the investigation, duplicates were eliminated, and all citations found were gathered and entered the bibliographic program. After a pilot test, two or more independent reviewers assessed the titles and abstracts to determine whether they matched the review's inclusion criteria. Two or more independent reviewers extensively evaluated the complete text of chosen publications pertaining to the inclusion criteria. The scoping review excluded full-text evidence sources that did not fall in accordance with the inclusion criteria. At every step of the selection process, the reviewers spoke or conferred with one or more additional reviewers to settle any differences that came up. The final scoping review provides a detailed report on the search and study inclusion process outcomes.

Author	Year Of Publication	Origin Of Study	Aim Or Purpose Of Study	Study Population And Sample Size	Methodology Or Methods	Intervention & Outcome Measures	Key Finding Related To Research Question
1. Anusha Etikala, Mustafa Tattan et al	2019	USA	To provide an overview of knowledge about NSAIDs and their potential effects on periodontal and implant wound healing	9 human clinical studies (periodontal), 4 animal studies + 2 human studies (implant)	Literature review via PubMed (MEDLINE)	NSAID types (non-selective and selective COX-2 inhibitors) and their impact on healing post periodontal and implant therapy.	NSAIDs, particularly COX-2 inhibitors, may impair bone healing and osseointegration.
2.Gomes et al	2015	Brazil	To review whether NSAIDs impair osseointegration and whether patients who use them chronically can safely receive dental implants	Review of 31 studies (2 clinical trials, 20 animal studies, 9 in vitro studies)	Systematic literature review of studies from 1982–2012 using PubMed, Cochrane Library, and Medline with defined inclusion/ exclusion criteria.	Reviewed the effects of NSAIDs (varied types, doses, durations) on bone healing, implant osseointegration, and osteoprogenitor cell activity.	Selective COX-2 inhibitors negatively affect osseointegration in both animal and in vitro studies; COX-1 inhibitors showed no adverse effects in clinical trials. Caution is advised when using COX-2 inhibitors postoperatively.
3.Luo et al	2018	Australia	To assess the effects of NSAID dosage, duration, and selectivity on osteoblast activity and osseointegration around titanium implants.	13 included studies (in vitro, in vivo, clinical); various animal models and human participants.	Systematic review following PRISMA guidelines; literature search (1999–2018) across 4 databases; inclusion of clinical, in vitro, and in vivo studies focusing on NSAIDs' effects on bone healing.	NSAID type (selective/ non-selective), duration, dosage; outcome measures included bone-to-implant contact, bone area/density, osteoblast activity.	Short-term NSAID use (especially ibuprofen) did not impair osseointegration in human studies, but selective COX-2 inhibitors (e.g., rofecoxib, meloxicam) showed negative effects in animal models with prolonged use.
4.Goodman et al	2002	USA	To evaluate the effects of COX-1/ COX-2 inhibitors vs. COX-2 selectivs. COX-2 selective NSAIDs on bone ingrowth in vivo	8 mature male New Zealand white rabbits	Titanium harvest chambers implanted in rabbit tibiae; tissue histology and immunohistochemistry	Naproxen sodium (non-specific COX-1/COX-2 inhibitor) and Rofecoxib (COX-2 inhibitor) given orally; bone ingrowth, osteoclast (CD51+), and osteoblast (alkaline phosphatase+) measures assessed	Both NSAIDs significantly decreased bone ingrowth; Rofecoxib reduced osteoblast area significantly; both reduced osteoclast-like cells; COX-2 inhibition may delay bone healing

5.Kalyvas & Tarenidou	2008	Greece	To review literature on the effects of NSAIDs on osseointegration, particularly their influence on bone healing and implant success	Various studies reviewed: human, animal (rabbits, rats, mice), and in vitro models	Literature review of experimental studies-both in vitro and in vivo-evaluating NSAID effects on growth factors, bone healing, and implant integration	NSAIDs (COX-2 selective and non-selective) may impair bone healing by inhibiting prostaglandin synthesis, growth factor expression, and osteoblast function; some effects were dose- and time-dependent	NSAIDs may negatively affect early stages of osseointegration; effects are likely temporary and reversible; further human research is needed to confirm clinical significance

Discussion

The objective of this scoping review was to compare and evaluate the effects of NSAIDs in patients osseointegration post implant surgery. In this study five articles were included, which includes original research, systemic and literature review and case study. On analysing the results, it can be concluded that NSAIDs, particularly COX-2 inhibitors do have a negative impact in the bone healing process and osseointegration post dental implant surgery.

Anusha Etikala, Mustafa Tattan[3], et al. evaluated the potential effects of NSAIDs in periodontal and implant wound healing, they conclude NSAIDs, particularly COX-2 inhibitors may impair bone healing and osseointegration. Evidence from animal and limited human studies suggests that NSAIDs, especially selective COX-2 inhibitors like celecoxib and meloxicam, may impair bone formation and negatively influence osseointegration by inhibiting osteoblast activity and reducing bone-to-implant contact. However, quality of evidence from available human clinical studies is poor and there are conflicting results from animal models. Hence in future better clinical studies are needed to more precisely evaluate the potential effects of NSAIDs on dental wound healing.

Gomes[6] et al. concluded that cyclooxygenase-1 (COX-1) inhibitors did not impair osseointegration. The animal studies showed that any drug that is capable of inhibiting COX-2 may impair the osseointegration process. The in vitro studies showed that COX-2 inhibitors are the most potent depressors of osseointegration at the cellular level.

Luo[1] et al. in systematic review evaluated the impact of non-steroidal anti-inflammatory drugs (NSAIDs) on osteoblast activity and osseointegration, with a focus on their use in dental implant procedures. The findings revealed that both non-selective and selective COX-2 inhibitors, such as indomethacin, meloxicam, and rofecoxib, can impair bone healing by reducing prostaglandin E2 (PGE2) production, osteoblast differentiation, and bone-to-implant contact. These effects were particularly evident with long-term use or high doses of selective COX-2 inhibitors. In contrast, human clinical studies included in the review, which mainly involved short-term ibuprofen use postoperatively, did not demonstrate significant adverse effects on marginal bone levels or implant success rates. The study by Arpornmaeklong[9] et al. found that therapeutic doses of indomethacin and celecoxib inhibit early osteoblast proliferation and PGE2 production on titanium surfaces in a dose- and time-dependent manner, with indomethacin having a stronger effect. Although mature cells showed reduced sensitivity, the findings suggest that short-term use of celecoxib may impair early osseointegration, highlighting the need for caution in its clinical use during implant healing.

Goodman[5], et al. in the Journal of Orthopedic Research demonstrates that nonsteroidal anti-inflammatory drugs (NSAIDs) such as indomethacin, ibuprofen, and high-dose aspirin significantly suppress bone ingrowth in histological sections from a DTC chamber model, with a dose-response effect observed for indomethacin and aspirin. These findings align with the mechanism by which NSAIDs inhibit prostaglandin E2 (PGE2) production, a key mediator of osteoblast activity and bone formation, thus impairing bone repair processes. Fracon[8] et al. which noted similar inhibitory effects on long bone fracture healing. This suggests that NSAIDs, particularly non-selective ones, should be used cautiously in clinical settings involving bone healing to avoid compromising outcomes, reinforcing the need for further research into safer alternatives or optimized dosing strategies.

Kalyvas & Tarenidou[2], evidence suggests that NSAIDs, particularly COX-2 inhibitors, can impair early osseointegration by interfering with key biological processes such as osteoblast differentiation, growth factor expression (e.g., BMPs, VEGF), and angiogenesis. Animal studies report reduced bone-implant contact and bone density following NSAID use, especially with prolonged or high-dose administration. However, some clinical studies have shown no significant long-term impact on implant stability with therapeutic doses of NSAIDs. Importantly, the adverse effects seem dose- and time-dependent and may be reversible if NSAID use is limited to the immediate postoperative period. Therefore, while caution is warranted, especially during early healing, further human studies are needed to establish clear clinical guidelines.

The review by Boursinos[7] et al. demonstrates that steroids and conventional non-steroidal anti-inflammatory drugs (NSAIDs) significantly impair fracture healing in animal models. NSAIDs like indomethacin delay callus formation and bone remodeling by inhibiting prostaglandin synthesis, critical for the inflammatory phase of healing. Selective COX-2 inhibitors, including celecoxib and rofecoxib, yield inconsistent results, with some studies showing delayed healing and reduced callus strength when administered early, and others suggesting minimal impact with later use. Limited human studies indicate an association between NSAID use and increased risk of non-union, particularly with high doses, but robust clinical data are scarce. These findings highlight the need for cautious use of these drugs in fracture patients and further randomized human trials to clarify their impact on bone healing.

Conclusion

NSAIDs are effective for managing postoperative pain in implant dentistry but may negatively affect bone healing, especially selective COX-2 inhibitors. Short-term, low-dose use of non-selective NSAIDs appears safer. Clinicians should use NSAIDs cautiously, considering type and duration, to balance pain control with optimal osseointegration. Further clinical studies are needed to guide safe NSAID use after implant placement.

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