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Research Article

Medications in Orthodontics: Promotors, Suppresors and Clinical Considerations

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Abstract

Orthodontic tooth movement (OTM) is a mechanistically controlled biologic reaction which takes place through remodeling of alveolar bone and periodontal ligament. It is controlled by a number of biochemical mediators and can be significantly affected either by systemically or locally administered drugs. Such drugs affecting OTM are commonly known as promoter drugs and suppressor agents. Since the majority of orthodontic patients are under long-term medication, pharmacologic action of various drugs must be well appreciated in order to make clinical-level decisions. A drug history and appreciation of drug–bone interaction enable the orthodontist to anticipate treatment difficulties, prevent unfavorable effects, and optimize patient safeguarding. More research must be done to create clinical guidelines that integrate pharmacological considerations into orthodontics.

Keywords: *Orthodontic Tooth Movement (OTM), Medications in Orthodontics, Bone Remodeling,Prostaglandins and Leukotrienes, NSAIDs and Corticosteroids, Bisphosphonates.*

INTRODUCTION

For optimal orthodontic treatment, there must be appropriate tooth movement. After mechanically applying force to the teeth, the resultant biological reaction is referred to as Orthodontic Tooth Movement (OTM). It is primarily the result of repetitive application of a controlled mechanical force to one or more teeth, causing remodeling of the socket by the formation of pressure and tension zones in the periodontal ligament (PDL) and alveolar bone. Pharmacologic drugs that modulate or interrupt the inflammatory process play a significant role in tooth movement. Some studies have investigated the effect of long-term and short-term drug administration on OTM.

Davidovitch et al. and Yamasaki et al. studies proved that orthodontic tooth movement (OTM) rate is affected by the systemic or local administration of some drugs. A drug refers to a single chemically active entity used in the treatment, prevention, curing, or diagnosis of disease. The World Health Organization (1966) established a definition of a drug as any product or substance taken for changing or investigating physiological systems or pathological conditions to benefit the recipient. Orthodontic tooth movement remodeling has also been termed as an inflammatory response. This periodontal ligament space inflammation then leads to the release of a number of biochemical mediators and signals that are important in the alveolar bone and periodontal ligament remodeling with tooth movement. Additionally,

inflammatory mediators, neurotransmitters, and growth factors are very important in the process. The principal players in this dynamic process are hormones, body forces, cAMP, certain cytokines like interleukin 1, colony-stimulating factors, calcium, collagenase, and prostaglandins, all of which play a critical role in mediating the biological remodeling required to create tooth movement. Theory of tooth movement has the age-old conviction that force application produces differential pressure in periodontal ligament and that it is contained within endosteal marrow space and periodontal ligament cells itself. Drugs act on such molecular mechanisms directly, and they alter periodontal tissue homeostasis.

The widely used drug in orthodontic treatment can be generically grouped into two broad categories:

1. Promoter drugs
2. Suppressor agents

Promoter agents are medications most often used to regulate inflammatory mediators to enhance orthodontic tooth movement. They include prostaglandins, leukotrienes, cytokines, vitamin D, osteocalcin, corticosteroids, thyroid hormones, and parathyroid hormones.

Suppressor agents, on the other hand, are medications that impede bone resorption. These are nonsteroidal anti-inflammatory drugs (NSAIDs), bisphosphonates, estrogens, cholesterol-lowering drug, and fluorides. (2)

PROSTAGLANDINS

Prostaglandins (PGE) are a group of eicosanoids that regulate the formation of cyclic adenosine monophosphate (cAMP) in tissues. Prostaglandins have the potential to modulate various cellular and tissue functions. Prostaglandins are central nervous system neuromodulators as they modulate the neuronal excitability as well as sympathetic neurotransmission in the peripheral nervous system. Specifically, PGE2 and PGI2 enhance the sensitivity of afferent nerve endings and therefore induce pain on account of chemical, mechanical, and thermal stimulation. Wenchen Lee (1990) studied the role of prostaglandin E1 (PGE1) in accelerating orthodontic tooth movement by enhancing bone resorption. Experimental mesial movement of the first molar was induced in seventy-two Wistar rats by the Waldo method. PGE1 was applied locally (5 µg/kg every 12 hours) or systemically (7.5 ng/kg/min), and the histologic changes were examined. Local and systemic treatments enhanced the osteoclasts and Howship's lacunae to a large extent relative to controls, validating PGE1's bone-resorptive stimulatory effect. The systemic treatment produced more intense and sustained resorptive effect relative to local injections. Local injection was characterized by pain behavior and more body weight suppression, whereas systemic infusion yielded more uniform results with no serious adverse effects. The results show that PGE1 stimulates bone remodeling and is capable of successfully reducing orthodontic treatment time, with systemic delivery being better than local injection. The article speaks of the potential of PGE1 as an adjunct to orthodontics. (3)

LEUKOTRINES

Leukotrienes form a distinct class of eicosanoids derived from the metabolism of arachidonic acid through the lipoxygenase pathway. Unlike prostaglandins, which are synthesized through the cyclooxygenase (COX) pathway, leukotrienes are synthesized only by lipoxygenase enzymes. They are very important in mediating inflammation responses, hypersensitivity responses, and disease states like asthma and allergic diseases. Pharmacological treatment of the disorders typically includes leukotriene receptor antagonists that inhibit the action of the receptors and consequently block the effects mediated by leukotrienes. Examples include montelukast and zafirlukast. Mohammed, Tatakis, and Dziak (1989) investigated the role of leukotrienes (LTs), as well as prostaglandins (PGs), in orthodontic tooth movement. 132 Sprague-Dawley rats were subjected to the experiment. Orthodontic appliances were employed to induce a mesial tipping force on maxillary first molars. The rats were placed into groups receiving distilled water, gum arabic, the PG synthesis inhibitor indomethacin, the LT synthesis inhibitor AA861, or both inhibitors concurrently. Movement of teeth was measured at intervals to 14 days, and prostaglandin E1 (PGE1) and leukotriene B4 (LTB4) tissue concentrations were measured. The findings showed maximum inhibition of tooth movement in the indomethacin, AA861, and combination groups from day 7. Of interest, inhibition of LT decreased LTB4 but raised levels of PGE1, showing a compensatory interaction between cyclooxygenase and lipoxygenase pathways. With the presence of elevated prostaglandins, tooth movement was decreased if leukotrienes were inhibited, implying that LTs play an essential role. The research concludes that prostaglandins and leukotrienes are key biochemical mediators for orthodontics, and research into their interactions has the potential to enhance the effectiveness of treatment and patient success. (4)

VITAMIN D3

In this subsequent experimental study, it was noted that vitamin D3 impacts orthodontic tooth movement in rats. Thirty-two male Wistar rats were divided into experiment and control groups with orthodontic appliances applying mesial force to the maxillary first molar. Vitamin D3 was delivered through local periodontal tissue injections in the experiment group, and a vehicle solution was injected into the control group. The amount of tooth movement and histological changes were monitored after 21 days. The results were that the exposed group of vitamin D3 enhanced the tooth movement much more than the controls. Histological examination showed raised osteoclastic activity and bone resorption on the pressure side and increased bone formation on the tension side, implying accelerated remodeling. The

research concluded that vitamin D3 accelerates orthodontic tooth movement by promoting bone turnover and thus becomes a potential aid in orthodontic therapy to decrease the treatment time. (5)

CORTICOSTEROIDS

The literature review of Knop et al. (2012) described the effect of corticosteroids on orthodontic tooth movement. Corticosteroids are broadly used for their immunosuppressive and anti-inflammatory effects and possess the capability to critically impact bone remodeling processes crucial for tooth movement. Experimental studies on animals have yielded disparate evidence: some observed enhanced tooth movement by enhanced resorption of bone and osteoporosis-like alterations after long-term treatment with corticosteroids, while others noted delayed movement by suppression of osteoclast activity, remodeling of collagen, and bone formation. These variations are due to variations in drug dosage, duration, and animals used. Corticosteroid injection can slow down bone turnover of jaws clinically, and accordingly orthodontic treatment may be postponed. Hence, patients receiving orthodontic treatment should be thoroughly screened for corticosteroid therapy, and longer appointment times and periodic radiographic checks should be maintained. It highlights the necessity for further human research to clarify the particular molecular mechanisms whereby corticosteroids influence orthodontic tooth movement. (6)

SUPPRESSOR DRUGS

ESTROGEN

Estrogen controls bone metabolism by suppressing osteoclast differentiation, inhibiting osteoclast survival, and activating osteoblast activity and, consequently, decreasing bone resorption and increasing bone production. Because orthodontic tooth movement (OTM) is dependent on alveolar bone remodeling, tooth movement slows with high levels of estrogen and speeds up with low levels (during menstruation, menopause, or following ovariectomy). Estrogen receptor-α (ERα) is a determinant in the bone response to mechanical loading and is responsive to systemic estrogen. Orthodontically-induced root resorption (OIRR), a universal side effect of treatment, can be mitigated by estrogen because of its bone-sparing influence, although current evidence is mixed. Clinically, since the majority of orthodontic patients are females, fluctuations of estrogen during menstrual cycles, pregnancy, or menopause would be able to affect treatment results. More research needs to be conducted to define the definite role of estrogen in OTM and OIRR. (7)

BISPHOSPHONATES

Examines the impact of bisphosphonates (BPs), commonly employed osteoporosis, osteopenia, and bone cancers drugs, on orthodontic treatment. Bisphosphonates are potent bone resorption inhibitors by inducing osteoclast apoptosis and inhibiting bone turnover, hence hindering orthodontic tooth movement (OTM). Animal and limited human studies provide evidence demonstrating that BP treatment lengthens orthodontic treatment time, but may be beneficial in maximizing anchorage and minimizing post-treatment relapse. Moreover, BPs have been promising in orthodontically induced root resorption reduction and enhancing stability following rapid maxillary expansion and mandibular distraction osteogenesis. However, their long half-life and complications like bisphosphonate-associated osteonecrosis of the jaw (BRONJ) are of concern to the clinician. The existing literature, primarily experimental, strongly supports cautious orthodontic application of BPs and highlights pre-emptive call for long-term prospective human trials prior to therapeutic use in everyday practice. (8)

NSAIDS

Nonsteroidal Anti-Inflammatory Drugs are also referred to as NSAIDS, and are mostly employed for alleviation from pain. NSAIDs are anti-inflammatory, antipyretic, and analgesic, and are employed in the treatment of numerous clinical conditions. Orthodontists should strictly refrain from recommending this medicine to the patient without consulting the dentist during orthodontic treatment. NSAID will inhibit Cyclooxygenase (COX) enzyme during arachidonic acid conversion and thereby inhibit the synthesis of prostanoids. Prostanoids are the eicosanoid hormone subclass and can mediate bone resorption.

EFFECT OF NSAIDS IN ORTHODONTIC TOOTH MOVEMENT

Orthodontic tooth movement (OTM) is a biologic response following regulated mechanical loading, resulting in bone and periodontal ligament remodeling. Pain induced by orthodontic forces is generally treated with nonsteroidal anti-inflammatory drugs (NSAIDs), which exert their action by inhibiting cyclooxygenase (COX) enzymes and, subsequently, prostaglandin (PG) synthesis. Because PGs, particularly PGE2, play a prominent role in mediating bone resorption, NSAIDs will retard OTM. Aspirin, ibuprofen, indomethacin, naproxen, and diclofenac are the drugs that blocked bone resorption and retarded tooth movement. Acetaminophen is an NSAID with minimal anti-inflammatory action and without effect on orthodontic tooth movement and thus is the drug of choice for orthodontic pain relief. COX-2 inhibitors such as celecoxib have no significant or negligible effect on OTM, while others such as rofecoxib exert a considerable inhibitory effect. The drug of choice is likely to be mainly responsible for maintaining the balance between relief from pain without compromising orthodontic progression. (9)

CONCLUSION

The impact of drugs on orthodontic tooth movement (OTM) is of particular concern as most of those undergoing orthodontics are also under medication for systemic diseases or general well-being. These medications, even for entirely disparate orthodontic conditions, generally have systemic effects on bone metabolism, inflammation, and cellular functions, thus influencing tooth movement either directly or indirectly. These changes will modulate the efficacy, speed, and even stability of orthodontic treatment. Certain medications, for example, have been found to slow movement and extend treatment, whereas others may hasten resorption and threaten anchorage or root integrity.

Thus, it is squarely the responsibility of orthodontists to take a full medical and drug history on the first visit and during treatment. Accurate information regarding the pharmacologic action of drugs in common use is inhibitory, stimulatory, or has a neutral effect enables clinicians to predict potential problems and plan prevention of adverse consequences. By incorporating such knowledge into orthodontic treatment, orthodontists are able to make treatment plans customized, prevent unnecessary delays, and ensure patient safety. Finally, drug-orthodontic interaction awareness not only maximizes the success of treatment but also ensures long-term success, stability, and patient satisfaction with orthodontic treatment.

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