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Interappointment pain & flare up during endodontic treatment procedures: An update

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Abstract

It is of utmost importance for a clinician to have a complete understanding of causes and mechanisms behind interappointment pain to prevent or manage the condition adequately. Interappointment pain is caused primarily by microbial injury to the periradicular tissues and to some extent by chemical and mechanical iatrogenic factors. The offending agents cause inflammation in the periradicular tissues resulting in pain and swelling. The extent of inflammation is proportional to the extent of injury caused to the periradicular tissues. Proper diagnosis and management of the flare up must be affected by the dentist. This review highlights the various causes, mechanisms and treatment of flare-ups.

Keywords: Interappointment pain, flare up, endodontic treatment procedures

Introduction

Modern day endodontic therapy aims at painless therapy of the root canal system. The occurrence of postoperative pain of mild intensity is not a rare event despite acceptable standards of endodontic therapy. The development of interappointment pain of moderate to severe intensity, accompanied or not by swelling, constitutes an emergency like situation and very often requires unscheduled visit for treatment. An association has been demonstrated between the presence of apprehension before endodontic treatment and post operative pain. This review will focus on the mechanisms of interappointment pain, causative agents and the host response to injury that can precipitate pain, diagnosis and management of interappointment pain.

Causes of Interappointment Pain

The causative factors of interappointment pain comprise mechanical, chemical and /or microbial injury to the pulp and periradicular tissues, which are induced or exacerbated during root canal treatment. Mechanical and chemical injuries are often associated with iatrogenic factors, but microbial injury is arguably the most common cause of interappointment pain. It has been reported that frequency of interappointment pain has been reported to be significantly higher in teeth with periradicular lesions as compared to teeth with vital pulps and normal periradicular tissues.

Microbial Causes

Interappointment pain is most often a result of imbalance in host-bacteria relationship induced by intracanal procedures. Circumstantial evidence has suggested that certain bacterial species associated with periradicular lesions include *Porphyromonas endodontalis*, *Porphyromonas gingivalis*, *Prevotella* species, *Treponema denticola*, *Tannerella forsythia* (formerly *Bacteroides forsythus*), *Filifactor alocis*, *Dialister pneumosintes*, *Peptostreptococcus micros*, and *Fingoldia* (formerly *Peptostreptococcus magna*). One study revealed that *F. nucleatum*, *Prevotella* species and *Porphyromonas* species were frequently isolated from flare-up cases^[1]. Most of the presumed endodontic pathogens only show virulence or are more virulent when in association with other species. This is because of synergic or additive microbial interactions, which can certainly influence virulence and play a role in symptom causation.

The microbial load is well recognized as an important factor for a microorganism to cause disease. If the host is faced with a higher number of microbial cells than it is used to dealing with, acute exacerbation of the periradicular lesion can occur.

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A virulent clone of a given pathogenic species does not always express its virulence factors throughout its lifetime. If the root canal environmental conditions are in some way altered by intracanal procedures and as a result become conducive to the expression of virulence genes, microbial virulence can be enhanced and inter appointment pain can ensue.

The host resistance to infection is a factor of unquestionable importance dictating whether a disease will develop or not. Herpesviruses have the ability to interfere with the host immune response, which may trigger overgrowth of pathogenic bacteria and/or diminish the host resistance to infection [2, 3]. The possibility exists that active herpesvirus infections in periradicular lesions may initiate or contribute to flare-ups [4].

Some situations during the endodontic treatment can facilitate microorganisms to cause interappointment pain. These include apical extrusion of debris; incomplete instrumentation leading to changes in the endodontic microbiota or in environmental conditions; and secondary intraradicular infections.

(a) Apical extrusion of debris

In asymptomatic periradicular lesions associated with infected teeth, there is a balance between microbial aggression from the infecting endodontic microbiota and the host defenses at the periradicular tissues. If during chemomechanical preparation microorganisms are extruded into the periradicular tissues, the host will face a situation in which it is now challenged by a larger number of irritants than it was before. Consequently, there will be a transient disruption in the balance between aggression and defense, in such a way that an acute inflammatory response is mounted to re-establish equilibrium.

The risks of interappointment pain during the treatment of infected cases can be even higher in the event of overinstrumentation. The incidence of postoperative pain in re-treatment cases with periradicular lesions has been demonstrated to be significantly high. Solvents used during filling removal are also cytotoxic and may contribute to exacerbation of the periradicular inflammation [5].

The quantitative factor comprises the number of microbial cells extruded (microbial load), while the qualitative factor encompasses the virulence of the extruded microorganisms.

Crown-down techniques, irrespective of whether hand or engine-driven instruments are used, usually extrude less debris and should be elected for the instrumentation of infected root canals. Therefore, the quantitative factor is more likely to be under control of the practitioner. On the other hand, the qualitative factor is more difficult to be controlled. If virulent clonal types of pathogenic bacterial species are present in the root canal system and are propelled to the periradicular tissues during instrumentation, even a small amount of infected debris will have the potential to cause or exacerbate periradicular inflammation.

(b) Incomplete instrumentation

Incomplete chemomechanical preparation can disrupt the balance within the microbial community by eliminating some inhibitory species and leaving behind other previously inhibited species, which can then overgrow. If overgrown strains are virulent and/or reach sufficient numbers, damage to the periradicular tissues can be intensified and then result in lesion exacerbation.

(c) Secondary intraradicular infections

Introduction of new microorganisms into the root canal system can occur due to several ways, the most common being a breach of the aseptic chain during treatment [6].

Non Microbial Causes

Non-microbial causes are represented by chemical or physical factors that can inflict damage to the periradicular tissues and thereby can be responsible for the development of interappointment pain. Indeed, non-microbial causes are usually associated with iatrogenic events. Examples of mechanical irritation causing periradicular inflammation include instrumentation (mainly overinstrumentation), and overextended filling materials. In cases of overinstrumentation, the larger the instrument, the larger the damage to the periradicular tissues. Consequently, the intensity of the inflammatory reaction will be high and so will the risk for postoperative pain to develop. Overextended filling materials mechanically compress the periradicular tissues and may induce pain. Examples of chemical irritation include apical extrusion of irrigants or intracanal medications.

Inflammatory Events Leading To Interappointment Pain

Bacteria that suddenly gain access to the periradicular tissues are faced with two immediate lines of defense, represented by the complement system and by phagocytes (neutrophils and macrophages) present in the chronically inflamed tissue. Bacteria may be recognized directly and engulfed by neutrophils and macrophages with receptors for common bacterial components. The encounter of the bacteria with these host defense mechanisms triggers the production and release of chemical mediators of inflammation, which will induce vascular changes in the microcirculation and recruit new phagocytic cells to the site. In response to bacterial challenge, phagocytes can release a variety of mediators, such as cytokines (IL-1, IL-6, IL-8, IL-12, TNF- α), prostaglandins, oxygen derived radicals, leukotrienes (particularly LTB₄), and platelet-activating factor (PAF). In addition to these products of phagocytes, the activation of the complement system by any of the three pathways generates mediators such as C5a and C3a, both of which can activate mast cells, causing them to release histamine. Histamine causes dilation of arterioles and increases permeability of venules. C5a is a powerful chemotactic agent for neutrophils and monocytes. The combined local effects of these mediators result in exacerbation of the inflammatory response, which usually starts immediately after increase in bacterial aggression and may take some hours before signs and symptoms become evident.

Together with the increased hydrostatic pressure secondary to vasodilation, the increase in the vascular permeability leads to a marked out-flow of fluid and its accumulation in the extravascular space. The inflammatory exudate leaving the vessels and accumulating in tissues will elevate the tissue hydrostatic pressure, which results in swelling and pain. When neutrophils and macrophages are strongly activated or challenged, tissue damage can also ensue, as oxygen radicals and lysosomal enzymes are not able to distinguish between host tissues and bacteria. The extreme form of tissue injury by neutrophils responding to bacteria is abscess formation.

A study found a highly significant association between the presence of allergies to various substances (atopy) and frequency of interappointment pain.

Treatment of Interappointment Pain

Hargreaves and Seltzer ^[7] described an integrated approach for the management and control of odontogenic pain. This has been termed the '3D' approach for pain control: Diagnosis, Definitive treatment, and Drugs.

Diagnosis

First of all, rule out the conditions that mimic endodontic pain, an unrelated sinus, TMJ –related conditions, post injection sequelae or another offending tooth. Gather information regarding when the posttreatment symptoms began, are they intermittent or continuous, are they mild, moderate or severe, is there an associated swelling and does anything exacerbate or alleviate the symptoms.

A thorough clinical examination should then be performed. The following conditions should be properly noted: areas of swelling, discoloration, ulcerations, exudation, defective and/or lost restorations, cracked or fractured teeth and apparent changes in occlusal relationships. Clinical testing should include percussion (both in axial and right-angle direction), apical palpation, bite-stick challenge, thermal stimulation (cold and hot if indicated) and periodontal probing, additional radiographs.

Definitive Treatment

Once the diagnosis has been confirmed that in fact it is the recently treated tooth that is responsible for the post-treatment symptoms, definitive effective treatment must be rendered.

Re-Instrumentation

The involved tooth or area should be properly anesthetized prior to any treatment. The access cavity should then be opened and additional anatomy looked for that might have been missed on the initial visit. Enhanced magnification and illumination are beneficial in this regard. Working lengths should be reconfirmed, patency to the apical foramen obtained and a thorough debridement with copious irrigation performed. Remaining tissue, microorganisms and toxic products or their extrusion are arguably the major elements responsible for the posttreatment symptoms. Occasionally, a suppurative exudation may be established through the root canal system. Drainage will allow for the exudative components to be released from the periradicular tissues thus reducing localized tissue pressure. Closed dressing is to be performed thereafter. Leaving the tooth open for drainage allows for re-infection of the canal and therefore not recommended.

Incision and Drainage

If the abscess occurs after the obturation of the root canal system, incision of the fluctuant tissue is perhaps the only reasonable emergency treatment, provided the root canal filling is adequate. In cases of poorly filled canals; and in addition to incision, the filling material should be removed in order to allow for additional pus drainage through the root canal space.

Intracanal Medicaments

Rogers *et al.* ^[8] demonstrated that both dexamethasone and ketorolac when placed in the root canals of vital teeth after pulpectomy procedures showed statistically significant pain relief at the 12-h time period as compared to the placebo group. No adverse reactions were found following their placement within the root canal system.

Occlusal Reduction

Rosenberg *et al.* ^[9] demonstrated that in teeth with pain upon biting, occlusal reduction was effective in reducing postoperative pain. Sensitivity to biting and chewing is perhaps due to increased levels of inflammatory mediators that stimulate periradicular nociceptors. Occlusal reduction may therefore alleviate the continued mechanical stimulation of the sensitized nociceptors.

Drugs

(a) Antibiotics

In a review on the use of systemic antibiotics for the control of post-treatment endodontic pain, Fouad ^[10] concluded that their use is without justification. Current advances in our understanding of the biology of the infectious and inflammatory process, along with the known risks associated with antibiotics, such as the emergence of multiresistant bacterial strains, strongly indicate that the clinician should seriously re-evaluate their prescribing habits.

(b) Non-narcotic analgesics

Non-narcotic analgesics, NSAIDs and acetaminophen have effectively been used to treat the endodontic pain patient. The NSAIDs have been shown to be very effective for managing pulpal and periradicular pain. In patients with known sensitivity to NSAIDs or aspirin, and who have gastrointestinal ulcerations or hypertension due to renal effects of NSAID's, acetaminophen should be considered for post-treatment pain. The results of several double blind placebo-controlled trials in endodontic pain patients indicated that 400 mg ibuprofen, 50 mg ketoprofen, 100 mg flurbiprofen and 30–60 mg ketorolac all produce significant analgesia as compared to placebo.

Pretreatment with NSAIDs for irreversible pulpitis should have the effect of reducing pulpal and periradicular levels of the inflammatory mediator PGE₂.

The parenteral NSAID ketorolac tromethamine, when injected intraorally or intramuscularly, produced significant analgesia in patients with severe odontogenic pain prior to treatment.

The combination of a NSAID and acetaminophen taken together show additive analgesia for treating dental pain ^[11].

(c) Opioid analgesics

For pain that is not controlled by NSAIDs and acetaminophen, narcotic analgesics are required. These may be given in combination with NSAIDs for additive effects. All opioids provide same degree of pain relief provided that they are prescribed at equipotent doses. Pentazocine is an attractive choice for patients having a prior history of opioid abuse as it does not provide significant euphoric effects mediated by conventional mu agonists. Tramadol has proven efficacy in the management of chronic pain. However, its benefit in acute pain management is not as well defined. It should be used with caution in patients with a history of seizure disorder.

Ideally one should maintain regular nonopioid dosing schedule and add an opioid as needed for 'breakthrough' pain ^[12].

(d) COX-2 Inhibitors

Rofecoxib has the advantage of good analgesic efficacy with single day dosing enhancing compliance and minimizing GI side effects. But these must be prescribed with caution in patients on anti-hypertensives, warfarin, pregnancy, patients under the age of 18 years.

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