

# Endodontic Flare – Ups: An Overview

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## ABSTRACT

*Numerous hypothetical mechanisms responsible for pain and swelling in endodontic flare ups are presented, even though these mechanisms may be interrelated. This review article also underlines the various treatment modalities for relief of pain and swelling in such situations, including pre-medication, drainage establishment, relief of occlusion, intracanal and systemic medication.*

## KEYWORDS

*Endodontics, Flare ups*

## INTRODUCTION

**F**lare up means development of pain & swelling during or after endodontic treatment. Various reasons have been attributed to these acute exacerbations of chronic conditions like 1). alteration of local adaptation syndrome. 2) microbial factors. 3) changes in periapical tissue pressure. 4) effects of

chemical mediators. 5) immunological phenomena. 6) and numerous psychological factors.

## AETIOLOGY OF FLARE UPS

### Alteration of local adaptation syndrome

Selye(1) has documented that there is local tissue adaptation to chronic inflammation

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and a violent reaction occurs if a new irritant is introduced. He injected various irritating chemicals to subcutaneous air filled pouches of a rat resulting in formation of a granuloma pouch after sometime indicating chronic inflammation. No reaction developed when same irritant was used but a severe flare up occurred when irritant was changed.

### Microbial Factors

Sundqvist(2) in his study concluded that in all flare up cases *Bacteroides melanninogenicus*, a gram negative anaerobic rod was present which was also endorsed by Griffee *et al*(3) saying that symptomless infected teeth did not contain *Bacteroides melanninogenicus*.

This gram negative rod produces collagenolytic, fibrinolytic enzymes and endotoxins (4) which activates Hageman factor releasing bradykinin, a potent pain mediator.

### Changes in periapical tissue pressure

Mohorn *et al* (5) showed that endodontic therapy causes pressure changes in periapical area in both directions, study carried out on dogs. A positive periapical pressure i.e excessive exudate not absorbed by lymphatic system, presses on nerve endings causing pain. In contrast a negative periapical pressure leads to aspiration of microbes and altered tissue proteins from root canal to periapical area resulting in increased inflammatory response and pain. In such cases no drainage occurs when root canal is opened.

### Effects of chemical mediators

#### Cell mediators

Like histamine, serotonin, prostaglandins, platelet activating factors, leukotrienes etc. are capable of producing severe pain, which are released from cells.

Histamine, present in mast cells, basophils and platelets, is released due to physical injury or some chemical agents, causes increased permeability of local blood vessels(6).

Serotonin found in gut mucosa, platelets and brain causes increased vascular permeability & contraction of smooth muscles like histamine.

Prostaglandins and leukotrienes which are synthesized by leucocytes and present in exudate during inflammation cause increased vascular permeability, enhance chemotaxis, induce pain and fever (7).

Platelet activating factor (PAF) is sourced from basophils, neutrophils, alveolar macrophages and monocytes(8), produces chemotaxis, platelet aggregation and increased vascular permeability(9). PAF causes oedema & hyperalgesia in rats even though it is yet to be established in humans (10).

### Plasma mediators

Plasma derived factors such as Hageman factor (factor xii) has three components a) prekallikrein activation b) triggers clotting c) triggers fibrinolysis.

Plasmin activates prekallikrein activator first, which further initiates circulatory prekallikrein to form kallikrein, which divides kinogen into kinin known as BRADYKININ (11) thus causing vasodilatation, smooth muscle contraction, enhanced permeability and pain induction in inflammation.

Fibrinopeptides, mediators from clotting system induce vascular leakage and promote leukocyte chemotaxis.

Fibrinolytic system involves plasmin which digests fibrinogen and fibrin. Endotoxins enhance bradykinin production and also influence Hageman factor(12).

### Immunological phenomenon

Pulp has capacity to produce antibodies like immunoglobulin IgG, IgM, IgA against dental caries(13) which can migrate to dentin as demonstrated by Thomas & Lever(14). This emphasizes the specific immunological response to carious process. In chronic pulpitis & periradicular periodontitis, macrophages and lymphocytes denote cell mediated and humoral immune response whereas immunoglobulins were detected in periradicular cysts & granulomas(15). Amongst immunoglobulin, IgG is the most commonly present(70-74%) in periradicular lesions(16).Cymrrman *et al*(17) observed cytotoxic and helper T lymphocytes in excised periapical granulomas. IgE present in pulp

& periapical lesions show immediate hypersensitive anaphylactic reactions (18).

### Psychological factors

Anxiety, fear, psychosis, apprehension & previous traumatic dental experience means a lot to dental patients especially during root canal procedures (19). These anxieties aggravate and intensify painful episodes

### MANAGEMENT

Various treatment modalities like pre-medication, relieving occlusion, open drainage, intracanal and systemic medication has been advocated for management of flare ups

### Premedication of pulp chamber and root canals:

Medication of pulp chamber & root canal has been tried to reduce flare ups due to forcing of infected debris to periradicular area in 1st appointment before instrumentation. But Pearson *et al* (20) found out no significant difference in acute exacerbation episodes in premedicated root canals prior to instrumentation in comparison to completely instrumented canals without any premedication.

### Establishing the drainage

Inflammatory oedema results due to chemical mediators whereas suppuration is caused by infections. Drainage relieves pain and swelling dramatically in suppuration cases, by removing intracanal dressing and keeping the access cavity open. Sometimes discharge does not drain, in those cases, soft tissue incision in the most dependent part of swelling is advocated(21). After cessation of discharging exudate, the access cavity should be temporarily closed again, since it does not serve any purpose to leave root canal open to oral microbial flora(22).

### Relief of occlusion

Cohen(23) suggested occlusal relief prior to endodontic therapy whereas Ingle(24), Weine(25) and Grossman(26) are of the opinion that occlusion should be relieved prior to root canal treatment in teeth which are painful to start with. Dorn *et al*(27) advocated

reduction of occlusion whenever the painful symptoms appear.

### Intracanal medicament

Most of the intracanal medicaments like formocresol, eugenol, camphorated monochlorophenol and iodine potassium iodide have been studied (28). None appeared to be particularly effective, nor was there any significant relationship between interappointment pain and the type of therapy used(29).

### Irrigating solutions

Harrison *et al* (30) found out that patients whose canals were not irrigated or irrigated with normal saline experienced more pain in comparison to those patients whose canals were irrigated with 5% sodium hypochlorite and 3% hydrogen peroxide or even 0.5% sodium hypochlorite alone, provided irrigating solution was not pushed to periapical region. However pain of endodontic origin is multifactorial and cannot be attributed to irrigant alone.

### Sulfa compounds

Nygaard ostby(31) reported relief in pain by using sulfa compounds as intracanal medicament whereas Seltzer *et al*(32) study showed that sulfonamides yielded no better results than placebos.

### Corticosteroids

Moskow *et al* have reported that corticosteroids placed in root canal control pain successfully(33). The anti-inflammatory activity of corticosteroids is based partly due to reduction of lysosomal release and partly due to inhibition of free arachidonic acid release from the phospholipids of cell membrane. The main disadvantage of using corticosteroids in endodontic therapy is their interference with phagocytosis and protein synthesis leading to rampant infection & repair impairment.

### Systemic Drugs

Antibiotics are widely used locally and systemically in endodontic cases, but their role in pain reduction is limited. However systemic antibiotics have a definite role in situations where patient exhibits cellulitis,

malaise, fever and toxemia. An appropriate antibiotic to control root canal infections should depend upon culture sensitivity testing. There are no specific studies regarding antibiotics role in reducing or eliminating pain in acute exacerbations during endodontic therapy. Systemic corticosteroids reduce pain & swelling in cases of single sitting flare ups. It was demonstrated by Marshall and Walton in their study (34) by administering 4mg dexamethasone intramuscularly which significantly reduced pain & swelling within 4 hrs after single sitting endodontic therapy.

Non-narcotic analgesics like aspirin is good for mild to moderate pain whereas narcotic analgesics like pentazocine, codeine, morphine are potent to control severe pain(35).

Non -steroidal anti-inflammatory drugs (NSAID) like ibuprofen, fenoprofen, naproxen etc are potent anti-inflammatory agents and are helpful in reduction of swelling & pain(36).

### Patient counseling

Detailing the complete procedure, expected benefits and possible pain responses of root canal treatment to the patient, will help to reduce the patient's anxiety, apprehension & tension because one prefers to know what will happen if he or she undergoes particular procedure (37).

Postoperative instructions like proper scheduling of medicines , application of ice, following the appropriate regimen of taking medicines etc will elevate the patient's pain threshold(38).

### SUMMARY

A number of factors causing pain & swelling in endodontic flare ups have been presented. In addition, the latest treatment modalities are also highlighted.

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