

## REVIEW ARTICLE

# Endodontic Flare Ups – An Overview

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**ABSTRACT**

Flare ups during endodontic treatment is an undesirable occurrence for both patient and clinician. Flare up phenomena is complex and not well understood which involves number of hypotheses for its etiology. Causes are numerous which include chemical, mechanical, microbial injuries to pulp and periapical tissues. This review discuss about the various etiological aspect and the management of flare ups

**Keywords:** Flare-ups, F. nucleatum, Local adaptation syndrome, Oxidation reduction potential, Prevotella species

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**INTRODUCTION**

An ongoing and frequently encountered problem in endodontics is the development of pain and swelling (flare-up) during or after endodontic therapy. The clinical symptomatology may be of such magnitude as to alarm both the therapist and the patient. Severe pain and swelling associated with flare-ups represent the clinical manifestation of complex pathologic changes occurring at a cellular level. There is an increase in evidence pointing to multiple factors like mechanical, mic-

robial, chemical, immunological and psychological components towards development of endodontic flare-up.<sup>1,2</sup>

Flare up is an acute exacerbation of periradicular pathosis after the initiation or continuation of root canal treatment. It would be desirable to understand the phenomenon and to know how to prevent the occurrence and be able to manage this event.

**ETIOLOGY***A. Alteration of the local adaptation Syndrome*

Local tissue adaptation has been shown experimentally by Selye<sup>3</sup> in his experiment. Inflammation is seen in the connective tissue when exposed to an irritant. Inflammation persists if the irritant is not removed; when a new irritant is introduced into the environment then a violent reaction may ensue, suggesting local adaptation to the previous irritant. A similar presentation maybe seen in a tooth with periapical periodontitis. Lesion maybe adapted to the local irritant with or without perceptible pain or swelling. When a new irritant is introduced into the periapical area in the form of intracanal medicament, irrigants; a violent reaction may ensue leading to liquefactive necrosis, indicative of alteration of the local adaptation syndrome.

*B. Changes in periapical tissue pressure*

The experiments of Mohorn et al<sup>4</sup> have indicated that endodontic therapy may also cause a change in the periapical tissue pressure. In teeth with increased periapical pressure, excessive exudate, not resorbed by the lymphatics, would tend to create pain by pressure on nerve endings. When the periapical pressure is less than atmospheric pressure it maybe conceived that the microorganisms and the altered tissue proteins maybe aspirated into the periapical area. Such teeth may not drain from the access of the root canal.

### C. Microbial factors

Microorganisms whether preoperative or postoperatively are the major causative agents in acute periradicular inflammation. Recent study has shown *F. nucleatum*, *Prevotella* and *Porphyromonas* species in flare-up cases.<sup>5</sup>

#### 1. Apical extrusion of infected debris:

Apical extrusion of the microorganisms and/ or their products during chemomechanical procedures may induce acute periradicular inflammation to reestablish the balance between aggression and defence (Figure1). Such response depends on both the number and virulence of extruded microorganisms.<sup>6</sup>

#### 2. Changes in the endodontic microbiota or in environmental conditions

Incomplete chemomechanical preparation induces changes within the root canal system that may favour the overgrowth of certain species. If overgrown bacteria reach sufficient number and express virulence genes, they can induce damage to the periradicular tissues, and a flare up may ensue (Figure2).

Endodontic procedures inevitably cause changes in the root canal environment. Changes in the root canal are inevitable during endodontic procedures. Environmental changes have the potential to induce virulence genes turned on or off. This is seen during incomplete root canal instrumentation and since it is impossible to predict whether environmental changes will lead to turn on or turn off of virulence genes, chemomechanical treatment should be completed in one session.<sup>7</sup>

#### 3. Secondary intraradicular infections:

Secondary infections are caused by microorganisms that are not present in the primary infection and that penetrate the root canal system during treatment (figure 3), between appointments or after the conclusion of the endodontic treatment.<sup>7</sup>

#### 4. Increase of the oxidation-reduction potential:

Oxidation-reduction potential has been theorized as the cause for exacerbation following endodontic procedures. In the presence of oxygen energy yield for the facultative anaerobes is more

marked than under anaerobic conditions. Facultative anaerobes like streptococci are present in the root canal which may resist intracanal procedures and overgrow to increase the Eh potential and induce acute periapical inflammation (figure-2). *F. nucleatum* appears to be associated with the development of the most severe forms of interappointment endodontic flare-ups.<sup>8</sup>

### RISK FACTORS FOR DEVELOPING A FLAREUP

Risk factors are those factors that are associated with an increased risk of developing a post treatment flare-up. They can be divided into patient presenting factors and what procedures were performed by the dentist. In the literature it is evident that the patient presenting factors are much more important than the treatment procedures in the development of endodontic flare-up.<sup>9</sup>

#### PATIENT PRESENTING FACTORS

##### *Patient demographics*

Studies have shown that the highest numbers of flare-ups were seen in females. Age has not shown to be a significant factor.

##### *Systemic conditions*

With the available literature the flare-up occurrence seems to be unrelated to health status of the patient. Flare-up occurrence does not increase with age hence systemic condition may not be a major factor in the development of endodontic flare-up.

##### *Pulp and periapical status*

Teeth with a vital pulp have relatively few flare-ups in contrast teeth with pulpal necrosis has a much higher incidence of flare-ups. The radiographic presence of a periapical lesion, particularly larger lesions, also serves as a risk factor for development of flare-ups. Immunological status of the periradicular tissue may predispose patients to develop a post endodontic

flare-up. Interestingly the presence of a sinus tract virtually ensures that a flare-up will not occur. Although this is indicative of an abscess, apparently the tract functions as a relief valve, releasing pressure, reducing tissue levels of inflammatory mediators, and thereby preventing the sudden increase in pain.

#### *Patient signs and symptoms*

Patient with preoperative swelling or pain is likely to develop interappointment flare-up. It is likely that patient with pain is associated with increased stress levels; this may adversely have its impact on the immune functions.

#### **TREATMENT PLAN**

Factors based on treatment plan whether conventional vs. retreatment, multiple vs. single. Complete vs. incomplete debridement also has to be considered. There is no evidence suggesting retreatment had higher incidence of flare-up than conventional treatment and most studies indicate no difference.

As to single vs multiple visits, a common belief has been that an approach to minimize post(a) treatment pain, particularly with pulp(b) necrosis/apical pathosis, is to extend treatment over more than one visit. Incomplete debridement has been traditionally assumed to be a cause of flare-ups. However, studies have shown this factor to be unrelated to the risk of developing a flare-up.

#### **INTERAPPOINTMENT FLAREUPS**

There are different factors that have been speculated to precipitate the flare-up including immunologic response, infection, or physical tissue damage, or a combination of the three.

These factors which interact with the primed immune cells are most likely to be seen in teeth with apical pathosis when compared to vital pulps and normal periradicular tissue. No significant difference have been noted in terms of gender, age, diameter of lesion, taking analgesics, placebos, or no medication, or preoperative symptomatic or asymptomatic tooth diagnosis on the incidence of flare-ups.

#### **TREATMENT OF INTERAPPOINTMENT PAIN**

Hargreaves and Seltzer 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**

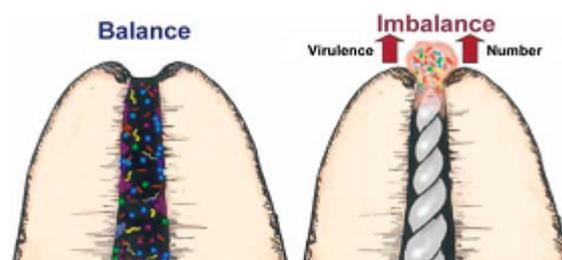
The initial phase of treating the endodontic pain patient is of course diagnosis there are several conditions that have been shown to mimic endodontic or odontogenic pain.<sup>10-11</sup> Obtaining a thorough understanding of the patient’s chief complaint should be the first step in proper management.

#### **DEFINITIVE TREATMENT**

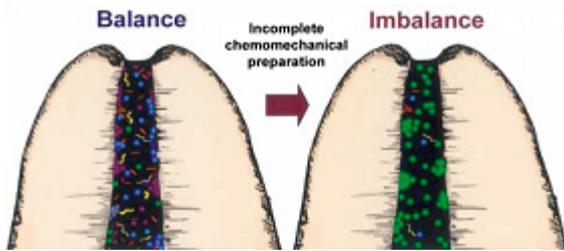
Once the diagnosis has been confirmed that in fact it is the recently treated tooth that is responsible for the post-treatment symptoms, definitive effective treatment must be rendered.

#### *Re-instrumentation*

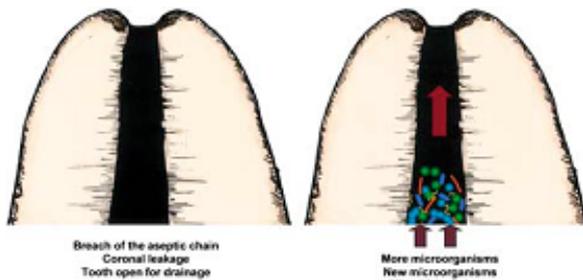
Definitive treatment may involve re-entering the symptomatic tooth. Remaining tissue, microorganisms and toxic products or their extrusion are arguably the major elements responsible for the posttreatment symptoms. Occasionally, a suppurative exudation may be established through the root canal system. Drainage will allow for the exudative components to be released from the periradicular tissues thus reducing localized tissue pressure.<sup>11,12</sup>



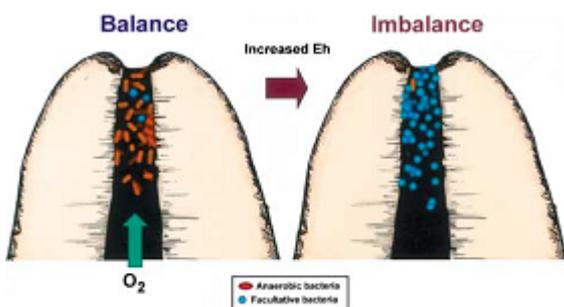
**Figure: 1-** Apical extrusion of infected debris.<sup>27</sup>



**Figure-2:** Changes in the endodontic microbiota or in environmental conditions<sup>27</sup>



**Figure-3:** Secondary intraradicular infections<sup>27</sup>



**Figure-4:** Increase of the oxidation-reduction potential<sup>27</sup>

*Cortical trephination*

Cortical trephination is the surgical perforation of alveolar bone in an attempt to release accumulated periradicular tissue exudate. Chestner et al.<sup>13</sup> reported pain relief in patients with severe and recalcitrant periradicular pain when cortical trephination was performed. Moos et al.<sup>14</sup> found no significant difference in postoperative pain of pulpal origin when treated by either pulpectomy alone or pulpectomy with cortical trephination.

*Incision and drainage (I&D)*

In a case with abscess, the rationale behind incision and drainage is to evacuate pus, microorganisms and toxic products from the periapical tissues. In case of incomplete endodontic treatment, it is recommended to re-enter root

canal to eliminate etiological factors through debridement, irrigation and placement of antimicrobial dressing.

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.

In addition, aggressive incision for drainage has been advocated for any infection with cellulitis, regardless of whether it is fluctuant or indurated.<sup>15</sup>

*Intracanal medicaments*

The use of intracanal steroids<sup>16</sup>, non-steroidal anti-inflammatory drugs (NSAIDs)<sup>17</sup> or a corticosteroid– antibiotic compound<sup>18</sup> has been shown to reduce post-treatment pain.

Rogers et al.<sup>16</sup> 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*

With the limited amount of literature, no benefit has been seen with reducing occlusion to prevent endodontic flare-ups.

Creech et al.<sup>19</sup> and Jostes et al.<sup>20</sup> have shown that routine occlusal reduction for the prevention of post-operative pain was ineffective. Rosenberg et al.<sup>21</sup> demonstrated that in teeth with pain upon biting, occlusal reduction was effective in reducing postoperative pain

**DRUGS**

*Antibiotics*

Fouad<sup>22</sup> has concluded in his review that systemic antibiotics for the control of post treatment endodontic pain is without any justification;

however it is prescribed frequently to treat endodontic pain.<sup>23,24</sup> The advances in understanding biology of infectious and inflammatory process, risks with antibiotics, emergence of multiresistant strains strongly indicate that the clinician should re-evaluate this prescribing habit.

#### *Non-narcotic analgesics*

Non-narcotic analgesics, NSAIDs and acetaminophen have effectively been used to treat the endodontic pain patient. These drugs produce analgesia by actions in both the peripherally inflamed tissues as well as in certain regions of the brain and spinal cord. Ibuprofen 600mg taken every 6 h is optimal for managing pulpal and periradicular pain of inflammatory origin. The combination of a NSAID and acetaminophen taken together show additive analgesia for treating dental pain.<sup>25,26</sup>

### **TREATMENT OF FLARE-UPS IN DIFFERENT CLINICAL SITUATIONS**

#### *Previously Vital Pulps (with or without complete debridement)*

A previously vital pulp will develop into an acute apical abscess. This will occur some time after the appointment and indicates that pulpal remnants have become necrotic and are invaded by bacteria. It is also likely that tissue remnants have become inflamed and are now a major irritant. The working length should be rechecked, and the canal(s) should be carefully cleaned with copious irrigation of sodium hypochlorite they should be opened and debrided, medicated with calcium hydroxide paste, and closed. If not these cases are managed with incision and drainage.<sup>9</sup>

#### *Previously Necrotic Pulps with No Swelling*

In these cases occasionally acute apical abscess may develop which is confined to the bone and can be very painful. The tooth has to be opened, gently recleaned, irrigated with sodium hypochlorite and drainage should be established.<sup>9</sup>

#### *Previously Necrotic Pulps with Swelling*

This situation is unlikely to be a true flare-up, patient reassurance and prescription of analgesics

will suffice. Nothing is gained by opening these teeth and pain will regress spontaneously.<sup>9</sup>

### **POSTOBTURATION EMERGENCIES – Treatment**

Postobturation emergencies are infrequent and are of mild level if present therefore active intervention is seldom necessary. Reassurance with mild analgesics will significantly control patient's anxiety and prevent overreaction. In case of acceptable root canal treatment with acute apical abscess, excision and drainage of the swelling will resolve the swelling in conjunction with reassurance and analgesics. If untreatable then apical surgery has to be considered.

### **CONCLUSION**

Even though it has been demonstrated that a flare-up has no significant influence on the outcome of endodontic treatment, its occurrence is extremely undesirable for both patient and the clinician and can undermine the clinician-patient relationships. Therefore, clinician should employ proper measure and follow appropriate guidelines in an attempt to prevent the development of inter-appointment severe pain and or swelling. Because microorganisms are arguably the major causative agents of flare-up, knowledge about the microbial mechanism involves in the aetiology of these phenomenon is of utmost importance.

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