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Post Endodontic Flare Up- A Dilemma

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Abstract: *Flare-ups can occur after root canal treatment and consist of an acute exacerbations of an asymptomatic pulpal and/or periradicular pathologic condition. The causative factors encompass mechanical, chemical, and/or microbial injury to the pulp or periradicular tissues. Of these factors, microorganisms are arguably the major causative agents of flare up. This review article discusses these many facets of the flare-up: definition, incidence causes, and predisposing factors along with the treatment.*

Keywords: *Acute exacerbation, causes, endodontic treatment, post operative pain.*

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I. Introduction

The primary goal of endodontic treatment is to hermetically seal the entire root canal system by an adequate biomechanical preparation, with no discomfort to the patient and provide condition of periradicular healing. (Udoye and Aguwa, 2010).[1] Some patients experience frequently vexing problem as pain and swelling during or after endodontic therapy -as per European Society of Endodontology[2]. The exact definition of flare up varies from author to author-[3] "**The American Association of Endodontists** defines a flare up as acute exacerbation of peri-radicular pathology after initiation or continuance of RCT."[4]

According to previous published data reported, frequency of post endodontic pain ranges from 1.4 to 16% and sometimes up to 50% in some studies (Ehrmann et al., 2003; Oliveira, 2010).[5,6]

II. Etiology

The origin of the post-endodontic flare-up is polietiological : mechanical, chemical and microbial factors influence its development. Dr. Seltzer has discussed a number of hypothesis thought to be related to etiology of flare up.[7]

1.1 Alteration of the local adaption syndrome.

Alteration in local adaptation syndrome explained by Selye is one of the most accepted theory explaining flare ups in symptomless tooth. He showed that there is a local tissue adaptation to applied irritants. Chronic inflammation persists if irritant is not removed. When endodontic therapy is performed new irritants such as medicaments, irrigating solution or debris is introduced in an inflamed tissue, a violent reaction may ensue and alters tissue proteins or debris may get introduced into the periapical lesion leads to liquefaction necrosis indicative of alteration of local adaptation syndrome.

1.2 Changes in periapical tissue pressure.

The experiments of **Mohorn et al.** have concluded that microorganisms and altered tissue proteins could be aspirated into the periapical area, resulting in accentuation of the inflammatory response and severe pain. This indicates endodontic therapy may also cause localized change in periapical tissue pressure.[8]

1.3 Microbial factors:

Various species of microorganisms proliferate in the apical area of the root canal. Bacteria residing in certain anatomical areas of the root canals such as isthmuses, apical ramifications, irregularities, deltas and dentinal tubules may be left untouched and unaffected by disinfection procedures.[9]

a). **Bacterial Populations In The Root Canal**: The microbiota in flare-ups and refractory or failed cases are different from untreated cases , the former having more of gram negative, facultatives and anaerobes and latter having more gram positive bacteria.[2] In the case of symptomatic apical periodontitis, when the tooth is sensitive to percussion, predominant strains of microorganisms are *Parvimonas micra, Eubacterium, Porphyromonas* (*P. endodontalis, P. gingivalis*) and *Prevotella*. Especially, the BPB (black – pigmented bacteria) have gained much attention.[2,10] **Matusow et al** proposed that a change in oxidation-reduction potential of a root canal environment can be a cause for exacerbation of symptoms after endodontic treatment. In an aerobic environment, the energy yield and growth rate are faster.

b). **Bacteroides Melaninogenicus:**

In all cases of flare-up, an anaerobic gram negative rod, Bacteroides melaninogenicus was found (Grifee et al). Bacteroides melaninogenicus produces enzymes which are both fibrinolytic and collagenolytic. It also produces an endotoxin which activates Hageman factor. The activated Hageman factor initiates production of bradykinin which is a potent pain mediator. In addition, endotoxin can activate an alternate complement system at C3, thereby synergising inflammation through release of vasoactive chemicals.[2] **Fusobacterium nucleatum** appeared to be associated with development of the most severe forms of flare-ups effects.[9]

1.4 Changes in cyclic nucleotides.(cyclic AMP and cyclic GMP)

Increased cyclic AMP levels reduce transmission of nerve impulses through hyperpolarization. Cyclic GMP enhances nerve depolarization, mast cell degranulation and pain transmission.[11] Investigations have shown that there is a relative increase in cyclic GMP over cyclic AMP in cases of painful teeth.[12]

1.5 Immunological phenomena.

Chemical mediators like histamine, serotonin, prostaglandins, leukotrienes, etc play a role in the causation of pain. Histamine & serotonin when release as a result of inflammation acts on the blood vessels and increase vascular permeability. Prostaglandins also increases vascular permeability, induces fever and sensitize pain receptors.[9]

1.6 Mechanical factors

Apical extrusion of infected debris- Comparable study performed by **Reddy and Hicks** showed that cleaning canals with hand endodontic instruments using step-back technique produces 2.58 mg of average amount of extrusion of debris into the periradicular tissues, while using NiTi rotational mechanical instruments with crown-down technique, it is less than 0.5 mg.[13] Over-instrumentation of the root canal and overfilling also responsible for flare up. Incorrectly measured working length and inadequate debridement of degenerated pulp of the root canal are one of the iatrogenic causes of flare up.[10]

1.7 Chemical factors

Irrigation solutions, intracanal medicaments, root fillings and substances, that are in their composition, used in endodontic treatment may cause chemical irritation and post-operation pain and sensitivity after entering the periradicular tissues due to pressure. Some intra-canal medicaments and irrigants like sodium hypochlorite, hydrogen peroxide, eugenol, iodine compounds, prarchlorophenol, formocresol can act as antigens and induce a hypersensitivity response.[9] Langeland et al study shows that corticosteroids are effective in smoothening the pain in teeth with signs of asymptomatic pulpitis. [14,10]

III. Risk Factors Causing Flare-Up After Endodontic Treatment

1. Demographics

El Mubarak et al show results, assessing that post-operative pain was more common among younger patients(18-33 years old) and rare in older patients due to narrowing of diameter of root canal.[10,1] It is established that post-operative pain is more common among women than men comparing the sexual influence to the development of the flare-up.[15]

2. **Condition of the pulp and apical periodontal tissue**

Results of the studies defining the connection between the frequency of flare-up after endodontic treatment, pain intensity and condition of the pulp (viable or necrotic) are controversial: it is established that 47-60% of patients having asymptomatic necrotic pulp experience pain defined from medium to acute during the

first 24 hours after endodontic treatment.[2,6,10]Chance of a flare is 9.64 times higher when the bone destruction is detected on the radiograph.[16] As per Genet et al: bone destruction of 5 mm and more is said to increase the probability of pain occurring.

3. One and/or two visits during the treatment

Existing literature on one and two visits RCT gives conflicting opinions and recommendations. It has been the subject of long-standing debate in the endodontic. Yold et al study summarizes that flare-up rate is 9 times higher after one visit endodontic retreatment compared to retreatment by two – visits.[17]Though statistically insignificant, the occurrence of more flare ups in the two visit RCT than in the one visit RCT in the study by Udoye Christopher el al ,Eleazer and Eleazer (1998) and Albashireh and Al Negrish (2006), but not with those of Oginni and Udoye (2004) and Peters (1980).

III. Treatment

Because all etiological factors cannot be precisely determined, many treatment regimens have empirical advocates. These include: relief of occlusion, medication of the pulp chamber or root canal, establishment of drainage through the root canal or by the excision of the overlying tissues; and various medications applied to the root canal or administered systemically. No specific treatment is universally accepted but may becomes successful because of the placebo effect.

IV. Conclusion

The occurrence of mild to moderate type of pain can occur even after rendering treatment of the highest standards. As evaluated and stated by different studies, minimal to moderate type of pain normally subsides with timeIf endodontic treatment that causes distress to both the patient and operator i.e. flare up, clinicians should employ appropriate measures in an attempt to prevent the occurrence of flare-ups and should be able to treat the efficiently when they do occur.

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4.2 Conflicts of interest
There are no conflicts of interest.

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