Review Article

International Journal of Dental and Health Sciences
Volume 03,Issue 04

ENDODONTIC FLARE UP

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ABSTRACT:

Pain management in Endodontics is of utmost importance for clinician to prevent or manage undesirable conditions.Flare up is one of the complication in endodontic treatment.This review article covers the various incidence,causative factors,hypothetical mechanisms,prevention,different treatment modalities of endodontic flare ups.

Keywords: Endodontic flare ups,irrigants,microorganisms

INTRODUCTION:

International Association for the study of pain defines pain as “unpleasant sensory and emotional experience associated with actual or potential tissue damage”. Pain is the most common reason for dentist consultation. It is a major symptom in many dental condition and can significantly interfere with a person`s quality of life and general functioning .[18] It is often spoken as a protective mechanism since it is usually manifested when an environmental change occurs that causes injury to responsive tissue. In Endodontic, pain may occur preoperatively, Inter appointment or postoperatively.[5] The success of Endodontic treatment is highly related to the elimination or reduction of endodontic pain.

ENDODONTIC FLARE UP

Defined as “Acute exacerbation of asymptomatic pulp or periradicular pathosis after the initiation or continuation of root canal treatment”. A flare up is characterized by severe pain and swelling that may arise following initial debridement of root canals or even after obturation. Requiring an unscheduled visit by patients and active treatment. [6]

According To American Association Of Endodontics, the inter - appointment flare ups has following criteria:

Within few hours to a few days after an endodontic procedure, a patient

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has significant increase in pain or swelling or combination of two.

The problem is of such severity that the patient initiates to contact with dentist.

The dentist determines the problem is of such significance that the patient must come for an unscheduled visit.

At the visit active treatment is rendered that may include incision for drainage, canal debridement, opening the tooth , prescribing appropriate medications or doing whatever is necessary to resolve the problem.[7]

INCIDENCE OF FLARE UP

- Over all incidence of flare- ups is ranges from **1.4% to 16% [1]**
- The incidence of flare-up increases in direct relationship to the severity of the patient's preoperative pathosis and signs/symptoms.
- Lowest frequency : vital pulp without periapical pathosis
  Highest frequency : patients who present with more severe pain and swelling, particularly with pulp necrosis and acute apical abscess. These more severe situations result in a flare-up incidence of close to 20%.[4,7] (Figure 1)

As the severity of pulp pathosis increases, patients are more likely to experience a flare-up

DEMOGRAPHICS

- *Younger age* : Post operative pain was more among younger patients (18-33 years old) according to ElMubarak et al .
- *Older age* : Flare up rarely occur in older patients ,due to the narrowing of the diameter of the root canal therefore less debris is extruded below the apex of the root . blood flow in the alveolar bone resulting in weaker inflammatory response.[8]
- Men : Less common among men
- Women : Pain threshold and toleration depend on sexual hormones and their proportion during different stages of menstrual cycle.Pain feeling is also regulated by hormone cortisol which takes part in mechanisms that are responsible for processing the pain.Its amount excreted in male amount is higher than in females. [8]

GENERAL STATE OF HEALTH

Flare up rate is low in patients using systemic steroids as treatment for systemic diseases. Steroids suppress the acute inflammatory response during the chemo-mechanical preparation of the root canal when mechanical, chemical and or microbial factors irritate the apical periodontal tissue.[8] Torabinejad et al points that patients tendency to allergies is associated with the development of a flare-up after endodontical treatment ,however wolton and fouad study disapproves this hypothesis. [6]

CONDITION OF THE PULP AND APICAL PERIODONTAL TISSUE

It is established that 47-60% of the patients having asymptomatic necrotic pulp experience pain defined from

medium to acute during the first 24 hours after endodontic treatment.

Bone destruction which is visible in dental radiograph is said to be a risk factor of flare-up . Chance of a flare is **9.64 times** higher when the bone destruction is detected.

The connection between size of the bone destruction area and post-operative pain was defined by Gernet et al : bone destruction of 5mm and more is said to increase the probability of pain occurring.[8] (Figure 2)

PRESENCE OF A SINUS TRACT

- 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. [7]

CLINICAL SYMPTOMS

Clinical symptoms before the treatment such as tooth pain when biting, chewing or by itself and sensitivity to percussion . 80% of patients who feel tooth pain before the beginning of the treatment usually feel the pain after treatment. Pain enhances the stress level in the body and effects immune function in a negative way therefore increasing the probability of a flare-up.[8]

TOOTH WHICH IS BEING TREATED

- Glennon et al study results show that temporary pain is felt 1,7 times more often when the canals of the **molar teeth** are treated compared to other teeth types.
- higher frequency of pain in teeth type is determined by the complicated complex anatomy of the root canals and chemo mechanical preparation.[8]

POSTOPERATIVE PAIN AND AMOUNT OF THE VISITS

It is More common after one visit endodontic treatment. Yold et al study summarizes that flare up rate is 4, 9 times higher after one visit endodontic retreatment compared to retreatment by two visits. [8,11,16,17]

INTRACANAL MEDICAMENTS

- Studies show that there is no direct link between usage of intracanal medicaments between visits and frequency of the pain.
- Intracanal medicaments are ineffective in preventing the flare-up which is caused by extrusion of infected debris through the apex of the root during the preparation of the root canal. [8]

ETIOLOGY OF FLARE UPS

MECHANICAL INJURY:

- Over instrumentation
- Inadequate debridement or incomplete removal of pulp tissue
- Apical extrusion of debris
- Secondary intraradicular infections

CHEMICAL INJURY

- Irritants
- Intracanal medicaments
- Overextended filling materials

But microbial injury is the major and the most common cause of inter-appointment pain.

These include

- Porphyromonas endodontalis
- Porphyromonas gingivalis
- Prevotella species
- Treponema denticola
- Tannerella forsythia (formerly Bacteroides forsythus)
- Filifactor alocis
- Dialister pneumosintes
- Peptostreptococcus micros
- Finegoldia (formerly Peptostreptococcus) magna.[1,13] (Figure 3)

PRESENCE OF PATHOGENIC BACTERIA

- A recent study revealed that F. nucleatum, Prevotella species and Porphyromonas species were frequently isolated from microbiota associated with flare-up cases.
- The possibility exists that the bacterial species associated with flare-ups are the same as those involved with primarily infected root canals associated with symptomatic periradicular lesions, although it remains to be confirmed. [13]

PRESENCE OF VIRULENT CLONAL TYPES

- Clonal types of a given pathogenic bacterial species can significantly diverge in their virulence ability.
- A disease ascribed to a given pathogenic species is in fact caused by specific virulent clonal types of that species.
- Thus, presence of virulent clones of candidate endodontic pathogens in the root canal may be a predisposing factor for interappointment pain, provided that conditions are created for them to exert pathogenicity.[13]

MICROBIAL SYNERGISM OR ADDITISM

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 .[13]

NUMBER OF MICROBIAL CELLS

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. This can be accidentally precipitated by endodontic procedures (not necessarily iatrogenic ones) .[13]

ENVIRONMENTAL CUES

A virulent clone of a given pathogenic species does not always express its virulence factors throughout its lifetime.

A great deal of evidence indicates that the environment exerts an important role in inducing the turning on or the turning off of microbial virulence genes .

Studies have demonstrated that environmental changes can influence the behavior of some oral (and endodontic) pathogens, including P. gingivalis, F. nucleatum, P. intermedia and oral treponemes . [13]

HOST RESISTANCE

It is well known that different individuals present different patterns of resistance to infections, And such differences can certainly become evident during individual's lifetime. Hypothetically, individuals who had reduced ability to cope with Infections may be more prone to develop clinical symptoms after endodontic procedures in infected root canals.[13] (Figure 4)

HERPES VIRUS INFECTION

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.

Herpesviruses may induce the release of proinflammatory cytokines by host defense cells. A recent study observed that active infections of periradicular lesions by human cytomegalovirus and/or Epstein–Barr virus were significantly associated with symptoms. Thus, the possibility exists that active herpesvirus infections in periradicular lesions ,May initiate or contribute to flare-ups. The mechanisms behind herpes viruses involvement with symptomatic periradicular lesions remain elusive.

PHOENIX ABSCESS

It is a condition that occurs in teeth with necrotic pulps and apical lesions that are asymptomatic . There is a exacerbation of a previously symptomless periradicular lesion. The reason for this phenomenon is thought to be due to the alteration of the internal environment of the root canal space during instrumentation which activates the bacterial flora.[5]

RECURRENT PERIAPICAL ABSCESS

It is a condition where a tooth with an acute periapical abscess is relieved by emergency treatment after which the acute symptoms return. In some cases the abscess may recur more than once,due to micro organism of high virulence or poor host resistance.

MECHANISMS OF FLARE UPS

Eight microbial and immunological factors are seen to be responsible for flare-ups (Seltzer et al 2004):

1. Alteration of local adaptation syndrome
2. Changes in periapical tissue pressure
3. Microbial Factors
4. Effects of chemical mediators
5. Cell mediators
6. Changes in cyclic nucleotides
7. Immunological phenomenon
8. Psychological factors [2,10]

ALTERATION OF LOCAL ADAPTATION SYNDROME

Selye has documented that there is local tissue adaptation to chronic inflammation and a violent reaction occurs if a new irritant is introduced. He injected various irritating chemicals to subcutaneous air filled pouches of a rat

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.

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. During chemo mechanical preparation micro organisms 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 .[1] (Figure 4)

Changes in periapical tissue pressure

Mohorn et al 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

Chemical mediators can be in form of cell mediators, plasma mediators and in form of neutrophils products. Cell mediators include like histamine, serotonin, prostaglandins, platelet activating factors, leukotrienes etc. are capable of producing severe pain. plasma mediators are present in circulation in inactive precursors form and get activated on coming in contact with irritants,for example : Hageman factor when get activated after contact with irritatnts produce multiple effects like production of bradykinnin and activation of clotting cascade. [2]

CHANGES IN CYCLIC NUCLEOTIDES

Bourne et al have shown that character and intensity of inflammatory and immune response is regulated by **harmones and mediators**. Increased levels of cAMP inhibits mast cells degranulation. Whereas increase in cGMP levels stimulate mast cell degranulation which results in increase in pain. Studies have shown that in flare ups there is increased level of cGMP over cAMP concentrations. [6]

IMMUNOLOGICAL RESPONSE

In chronic pulpitis and periapical diseases presence of macrophages and lymphocytes indicates a both cell mediated and humoral response.

Despite of their protective effect the immunologic response also contributes to destructive phase of reaction which can occur, causing perpetuations and aggravation of inflammatory process.[6]

PSYCHOLOGICAL FACTORS

Anxiety, fear, psychosis , apprehension, previous traumatic dental experience

play an important role in mid-treatment flare-ups .[6]

POSTOPERATIVE PAIN IN RE-TREATMENT CASES

- Flare up rate is high .
- During removal of the root filling material and further instrumentation, filling remnants and infected debris tend to be pushed ahead of the files and to be forced into the periradicular tissues, exacerbating inflammation and causing pain.
- Solvents used during filling removal are also cytotoxic and may contribute to exacerbation of the periradicular inflammation.[1]

INCOMPLETE INSTRUMENTATION

The microbiota associated with primary endodontic infections is usually established as a mixed consortium, and alteration of part of this consortium will affect both the environment and the remaining species.

Potent exogenous forces represented by chemomechanical preparation using antimicrobial irrigants and intracanal medication are needed to eradicate microbial communities from the root canal system. However, 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.[1] (Figure 5)

Furthermore, environmental changes induced by incomplete instrumentation have the potential to induce virulence genes to be turned on.

As a result of the increase in microbial virulence, a previously asymptomatic case may become symptomatic.

Another form of environmental change induced by endodontic intervention refers to the entrance of oxygen in the root canal.

It has been suggested that this can alter the oxidation–reduction potential in the root canal and as a consequence, acute exacerbation can occur .

SECONDARY INTRARADICULAR INFECTIONS

These infections are caused by microorganisms that were not present in the primary infection and have gained entry into the root canal system during treatment, between appointments, or even after the conclusion of the endodontic treatment.

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 .

If the microorganisms that gain access to the root canal are successful in surviving into and colonizing such a new environment, a secondary infection will establish itself and may

be one of the causes of postoperative pain, providing that the newly established microbial species are virulent and reach sufficient numbers to induce acute periradicular inflammation.[1]

MANAGEMENT OF FLARE –UPS

As the etiology of flare ups is multifactorial ,many treatment options have been empirically advocated. Management of flare –ups can be categorized as :

- Preventive
- Definitive

PREVENTION OF FLAREUPS

A history of preoperative pain or swelling particular in case of necrotic and infected pulps,is one of the best predictors of interappoinment flare up. However one should bear in mind that flare up are often unpredictable,because all infected cases have thereotically inceased risk to develop interappoinment flare up.

Guidelines And Procedures To Prevent Or Reduce The Incidence Of Flare Ups:

PROPER DIAGNOSIS:

- Identify the correct tooth causing pain.[5]
- Ascertain whether tooth is vital or non vital.[5]
- Identify if tooth is associated with periapical lesion. [5]
- Determine correct working length [5]
- Inaccurate measurement of the working length may lead to under or over instrumentation and extrusion of debris, irrigant,medicaments or filling materials beyond apex.[5]
- Determine correct working length by Radiographs,Apex locaters. [5]
- Selection of instrumentation technique that extrude less amount s of debris apically.[5]
- 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. [5]
- Complete debridement [5]
- Thorough cleaning and shaping of the root canal system and complete extirpation of vital pulp may decrease the incidence of flare ups.[5]
- Completion of chemomechanical preparation in single visit.[5]
- Use of an antimicrobial intracanal medicament between appointments in the treatment of infected root canal.[5]
- Irrigation Preferably with combination of irrigants such as sodium hypochlorite and chlorohexidine
- Occlusal reduction-The relief of pain provided by occlusal reduction is due to the reduction of mechanical stimulation of sensitized nociceptors.Reduce tooth from occlusion especially if apex is severely violated by over-instrumentation.[12]
- Placement of intracanal medicaments in multi visit root canal treatment ,Calcium hydroxide has been recommended as an

intracanal medicament for prevention or the treatment of flare-up. Chlorhexidine gluconate and iodine potassium iodide are other primary medicaments.

- Not leaving teeth open for drainage.
- Avoid filing too close to the radiographic apex.
- Maintaining the aseptic chain during intracanal procedures**.** [5,7]

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:

1. Diagnosis
2. Definitive treatment
3. Drugs

DIAGNOSIS

Obtaining a thorough understanding of the patient's chief complaint should be the first step in proper management. Gathering information, such as on when the post-treatment symptoms began, are they intermittent or continuous, are they mild, moderate or severe.

CLINICAL EXAMINATION

The following conditions should be properly noted:

1. Areas of swelling
2. Discoloration
3. Ulcerations
4. Exudation
5. Defective and/or lost restorations
6. Cracked or fractured teeth.
7. Apparent changes in occlusal relationships.

DEFINITIVE TREATMENT

The clinician should reassure the patient. 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. 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. 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 post treatment symptoms . [1,3]

1(b) Cortical trephination

Cortical trephination is defined as the surgical perforation of the alveolar bone in an attempt to release accumulated periradicular tissue exudate.

Additionally, in the asymptomatic patient, cortical trephination has been shown to decrease by 16–25% post-operative pain incidence when performed prophylactically.

Moos et al. compared the difference in post-operative pain relief in patients with acute periradicular pain of pulpal origin when treated by either pulpectomy alone or pulpectomy with cortical trephination.There were no

significant differences between the groups. [3]

INCISION AND DRAINAGE (I&D)

In teeth where the endodontic treatment has not yet been completed, it may be advisable to re-enter the root canal system to further eliminate the original etiologic factors via debridement, irrigation and the placement of an 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.

Drainage allows for the exudative components to be released from the periradicular tissues thus reducing localized tissue pressure.

It has been pointed out that leaving a tooth open is the most direct way to allow for re-infection via the oral microbiota .Weine advocated enlarging the apical constriction to at least a size #25 endodontic file to allow for drainage through the tooth.

Antibiotics are usually not indicated in cases of a localized abscess ,but they can be used to supplement clinical procedures in cases where there is poor drainage and if the patient has a concomitant trismus, cellulitis, fever or lymphadenopathy.

Poorly Filled Canals ,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. [3,15]

INTRACANAL MEDICAMENTS

Clinical studies have demonstrated that post-treatment pain is neither prevented nor relieved by medicaments such as formocresol, camphorated paramono chlorophenol, eugenol, iodine potassium iodide, Ledermix, or calcium hydroxide.

However, the use of intracanal steroids , non-steriodal anti-inflammatory drugs (NSAIDs) or a corticosteroid– antibiotic compound.has been shown to reduce post-treatment pain. [1,3,15]

DRUGS

SYSTEMIC ANTIBIOTICS:

Antibiotics recommended only in cases of medically compromised patients at high risk levels and in case of spreading infection that indicates failure of local host responses to control bacterial irritants.

Commonly prescribed antibiotics include penicillin ,erthyromycin or cephalosporin, metronidazole, tinidazole, ornidazole and clindamycin used against anaerobic bacteria.[9,15]

ANALGESICS

Non-narcotic analgesics, NSAIDs and acetaminophen have effectively been used to treat the endodontic pain patient. The combination of a NSAID and acetominophen taken together show additive analgesia for treating dental pain. For pain that is not controlled by NSAIDs and acetominophen, narcotic analgesics are required.These may be given in combination with NSAIDs for additive effects. [9,3,1,15]

PATIENT COUNSELING

Detailed explanations of 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 .[15]

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. [2,15]

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 the patient and the clinician, and can undermine clinician–patient relationships.

Therefore, clinicians should employ proper measures and follow appropriate guidelines in an attempt to prevent the development of interappointment flare-up.

CONCLUSION:

REFERENCES:

1. J.f.siqueira jr. International endodontic journal, 36, 453-463, 2003
2. Endodontic Flare – Ups: An Overview J Oral Health Comm Dent 2010; 4(3): 67-71.
3. Siqueira and f. barnett Endodontic topics 2006, 7, 93-109 inter appoinment pain: mechanisms, diagnosis and treatment.
4. Bayram inc et al incidence of postoperative pain after single-and multi-visit endodontic treatment in teeth with vital and non vital pulp.eur j dent2009 oct;3(4):273-279.
5. Dr. Neeta Shetty MID TREATMENT FLAREUPS IN ENDODONTICS endodontology –A DILEMMA Flare-Ups in Endodontics .
6. A Review IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 9, Issue 4 (Sep.- Oct. 2013), PP 26-31.
7. Anurag singhal et al Flare up in endodontics review JDOR jan march 2012.
8. Egle sipaviciute et al stomatologija ,Baltic dental and maxillofacial journal ,2014,vol.16,no 1.
9. Karl keiser et al Endodontic topics building effective stratergies for the management of endodontic pain 2002.
10. Tharangini raveenthiraraja et al flare up in endodontics : A review INT JOU pharmaceutical and clinical research 2015;7920:148-150.
11. Udoye Christopher et al flare-up incidence and related factors in adults. Jdoh aug 2010.
12. Masoud parirokh et al effect of occlusal reduction on postoperative pain inteeth with irreversible pulpitis and mild tenderness to percussion. joe-vol 39,number 1,jan 2013.
13. Hrikaran et al Clinical and pharamacological management of endodontic flare –up j pharm bioallied sci aug 2012.
14. Ernest h.ehrmann et al Flare-ups in endodotics and their relationship to various medicamnets aust endod j 2007;33:119-130.

15. Samuel seltzer et al flare-ups in endodontics: II therapeutic measures journal of endodontics vol 30 no 7 july 2004.
16. M.gotler postoperative pain after root canal treatment : a prospective cohort study int journal of dentisrtry 2012.
17. Yingying su et al healing rate and post obturation pain of single – versus multiple visit endodontic treatment for infected root canals: a systematic review joe 2010.
18. K Anjaneyulu et al influence of calcium hydroxide on post-treatment pain in endodontics: a systematic reviewjocd vol 17 issue 3 2014..