An overview of etiology of endodontic flare-ups

Anupam Sharma[1,*], Jyoti Mandlik[2], Reshmi Varghese[3]

[1]Professor and Head, [2]Associate Professor, [3]P.G. Student, Dept. of Conservative Dentistry & Endodontoics, Bharati Vidyapeeth Deemed University Dental College & Hospital, Pune

***Corresponding Author:**
Email: dranupamsharma@hotmail.com

Abstract

Endodontic flare-ups are unpleasant events that may occur during endodontic treatment procedures. Various etiological factors may be responsible for pain and inflammation in endodontic flare ups. This article overviews numerous mechanisms thought to be responsible for the flare-ups their incidence and also enumerates various modalities for relief of pain and swelling in these situations, viz. premedication, establishment of drainage, occlusal relief, intracanal medication and systemic management.

Keywords: Endodontic treatment, Flare-up, Intracanal medication.

Introduction

In the course of routine endodontic therapy, emergency treatment may be required to relieve pain, whether or not the originating need for the endodontic care involved an emergency appointment.(1) Some problems during root canal treatment may be post-obturation pain and inter-appointment inflammation. In most cases these problems do not last long, but can be a source of embarrassment to dentist and unease for the patient, especially if the tooth was asymptomatic before commencement of treatment.(2)

Flare-up is a complication during endodontic treatment, defined as an acute exacerbation of asymptomatic pulp or peri-radicular pathology following initiation or continuation of root canal treatment.(2) Other characteristics are occurrence of pain or swelling during endodontic treatment(3) or pain and swelling that may require additional treatment, including further intervention from dentist.(4)

The exact definition of flare-up differs from author to author.(5) The interappointment flare ups is also defined as "Within hours to few days after a root canal treatment procedure, the patient has either pain or swelling or both" The problem must be of sufficient degree that affects patient's lifestyle so that patient contacts the dentist. This may result in: (a) an unscheduled visit and (b) active treatment (incision to drain, canal debridement, opening for drainage).(6) John Harrison, Craig Baumgartner (1983)(7) defined slight pain as any discomfort howsoever brief that does not require medication or any other palliative treatment and which did not delay obturation at the second appointment. Moderate to severe pain was defined as pain requiring medication or palliative treatment or which delayed the obturation of canal system beyond the second appointment. Walton and Fouad(8) defined flare-up "as follows within few days after a root canal treatment procedure, a patient has either pain or swelling or a combination of both". Paul D Eleazer, Kristen R. Eleazer(1998)(9) defined flare ups as either patients reporting pain that was not controlled with over-the-counter medication or an increasing swelling, as reported. The American Association of Endodontists(10) defines a flare up as acute exacerbation of peri-radicular pathology after initiation or continuance of RCT. As definitions of flare up vary, so do reported incidence of such exacerbations (ranging from 1.4% to nearly 45%)

Incidence

The importance of the study of flare-up incidence is its use as a bench mark against which operator's skill can be measured. It then follows that the reason why RCT with low or no flare-up should be the treatment of choice, provided effectiveness and cost remain uncompromised. However, it remains difficult to compare flare-up incidents across studies as flare-up diagnosis varies among studies; more so because both sample populations and case definitions are different.(11)

Studies report grossly varying incidences of flare-ups. These variations may at least partially be the result of examining different factors and conditions as related to flare-ups. For example, Morse et al reported an incidence of nearly 20% flare-ups (swelling as the only criteria) after treatment of asymptomatic teeth with pulp necrosis and chronic periodontitis.(12) In comparison, Barnett and Tronstad in their retrospective study found an incidence of approximately 5.5% flare-ups (pain or swelling) in patients with similar diagnoses (necrosis of pulp with asymptomatic periapical pathosis), but only 1.4% in all patients regardless of diagnosis. Others have reported varying percentages of incidence of flare-ups, again depending upon the factors considered. There are obvious problems with most of these above-mentioned studies; in that many are retrospective, did not have controls, had relatively small sample of patients or had undefined variables.(13)

John Harrison, Craig Baumgartner, David R. Zeike (1983) reported treatment of 245 cases, 76% were free of inter-appointment problems, 17% had mild pain and 6% had pain that requiring palliative treatment.(14) M.

Georgopoulou, P. Anabtassiadis and S. Sykaras (1986) carried out a clinical study to determine the incidence of pain after chemo mechanical preparation of root canals. Their results showed that out of 245 patients in their study 140 (57%) had no pain, 52 (21%) had mild pain, 30 (12%) had moderate pain and 17(7%) reported severe pain.[15]

Etiology

Seltzer discussed a number of hypotheses thought to be related to etiology of flare- ups.

- **Alteration in local adaptation:** There is always some degree of local tissue adaptation to applied irritants. When a new irritant such as a medicament, irrigating solution or debris is introduced in an inflamed tissue a violent reaction may ensue, indicative of alteration in local adaptation syndrome. Ordinarily, connective tissue becomes inflamed when exposed to irritants. Chronic inflammation may persist if the irritant is not removed or if there is local adaptation. However, when a new irritant is introduced to inflamed tissue, a violent reaction may occur.[16] It is of significant clinical interest that violent reactions occur in teeth whose root canals are left open for drainage and in those with long standing asymptomatic periapical lesions. The flare-up can also result from salivary products including secretory IgA, activation of complement system, or from forcing of microorganisms or their products into a previously adapted environment.[17]
- **Changes in periapical tissue local pressure:** Investigations of bone marrow pressure have indicated that various pathologic conditions may produce a wide range of positive pressures. The studies of Mohorn et al have indicated that endodontic therapy may also cause localised changes in the periapical tissue pressure. They concluded from their experiments that microbes and altered tissue proteins could get aspirated into periapical area, resulting in an accentuation of the inflammatory response or even severe pain. Theoretically, such teeth may not drain when the root canal was opened.[17]
- **Microbial factors:** Past studies have failed to uncover a relation between clinical symptoms and presence of specific microorganisms or groups of microorganisms in infected root canals. However, several recent studies have suggested that such relationships may indeed exist.[18]

Prior to 1970's, voluminous data on studies of flora of infected root canals established presence of a considerable variety of microorganisms. These studies were generally performed in both aerobic and anaerobic environs according to the accepted methods of the time. With development of newer techniques for obligate anaerobic culturing, startling findings with respect to anaerobic flora of the root canal have emerged.[19]

From these studies, it seems reasonable to conclude that anaerobic culture techniques produce a far greater spectrum of microbial isolates than purely aerobic techniques. The latter do not seem sensitive enough for detection of entire microbial flora in infected root canals. Furthermore, anaerobes in mixed root canal infections may be responsible for production of various enzymes and endotoxins that inhibit chemotaxis and phagocytosis and interference with antibiotic activity; resulting in the persistence of painful periapical lesions.[20]

Sundqvist et al. found that combinations of bacteria, which included strains of *B.melaninogenicus* or *B.asaccharolyticus;* produced transmissible infections with purulence when inoculated subcutaneously in guinea pigs. It would appear that bacterial synergism is of major importance in maintaining Bacteroides infections.[21] Tanner et al showed that the previously identified species of *B.melaninogenicus* is presently known to contain four distinct bacterial species. They identified a new genus of anaerobic Gram-negative rods, *Wolinella rect;* from endodontically involved teeth with periapical radiolucencies that were associated with pain and/or swelling.[22]

Bacteroides melaninogenicus produces enzymes which are both fibrinolytic and collagenolytic. It also produces an endotoxin which activates Hageman factor.[23] 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. Thus, it appears as if endotoxins elicited from infected root canals may contribute to increasing vasoactive and neurotransmitter substances at the nerve endings of inflamed periapical lesions.

Whether the flora in an infected root canal can change when endodontic treatment is introduced or whether a change in proportions of aerobes to anaerobes can cause clinical exacerbations is conjectural. That the emphasis is on the significance of Gram-negative anaerobes in the production of pain and swelling does not in any way negate the fact that Gram-positive bacteria may also be involved in root canal flareups. It appears that myriad microorganisms may be associated with infectious exacerbations. Teichoic acids as a group of phosphate containing polymers are present in cell walls and plasma membranes of many Gram-positive bacteria. These lipoteichoic acids that are extracted from a variety of lactobacilli, streptococci and bacilli; have been reported to be potent immunogens, producing humoral antibodies IgM, IgG, and IgA. Furthermore, the induced inflammation may release various chemical mediators, which are capable of causing pain.[24]

- **Effects of chemical mediators in flare-ups:**

Cell mediators including histamine, serotonin (5-hydroxytryptamine (5 HT)), Prostaglandins (PGs), platelet-activating factor (PAF), leukotrienes (LTs), various lysosomal components and some lymphocyte

products called lymphokines, all of which are capable of causing pain.(seltzer) Plasma derived factors like Hegeman factor can also cause pain. Hageman factor can also be activated by bacterial endotoxins.[17]

- **Changes in Cyclic Nucleotides:**

Presence of Cyclic AMP has been demonstrated in normal pulps as well as in inflamed pulps. Another cyclic nucleotide, cyclic GMP; is also present in all living systems. Cellular regulations including pain transmission; have been reported to be influenced by the interaction of cyclic AMP and cyclic GMP. Cyclic GMP can be involved in mediating effects of norepinephrine and histamine at certain receptors that are distinct from those associated with the cyclic AMP system. Cyclic GMP has the potential to enhance nerve depolarization and mast cell degranulation. Both of these factors would enhance pain.[17]

Transmitters such as histamine, norepinephrine and serotonin that are elaborated during inflammatory response are capable of elevating cyclic AMP levels in the periapical tissues. In some instances increases of cyclic AMP may reduce the transmission of nerve impulses through hyperpolarization. However, this relationships are not so clear.

Contributing factors

- **Inadequate debridement:** Onset of acute pain or persistence of pain often signals the presence of residual pulp tissue in inadequately instrumented or still undetected canals. Inadequate debridement of degenerated pulp or pulp that is prgressively degenerating allows bacteria and their toxins to remain in the root canal and acts as a continuous irritant.[25]
- **Apical extrusion of infected debris:** Can lead to a transient disruption in the balance between host defence and microbial aggression in a way that host will mobilize an acute inflammation to re-establish the balance. It can be rated as one of the principle causes of post-operative pain.[25]
- **Secondary intra-radicular infection:** It can be caused by microbes not present in primary infection. These usually get entrance in the root canal system between appointments via leaking temporary restorations, fractured tooth or when the tooth is intentionally left open for drainage.[25]
- **Over-instrumentation:** There is a correlation between endodontic over-instrumentation and postoperative pain. The incidence of moderate-to-severe pain is reported to be higher if instrumentation penetrates beyond the apical foramen. With care and attention, gross over instrumentation is avoidable. Gross over instrumentation can also cause acute apical periodontitis,; producing pain that is primarily inflammatory.[25]
- **Overfilling:** The extrusion of sealer or gutta-percha filling or both into periapical tissues of teeth that exhibit no periapical radiolucent areas is more likely to yield higher incidence and degree of postobturation pain than similar teeth that are filled flush or upto1mm short of radiographic apices. However, this is not a universal finding because some clinicians have found no correlation among levels of obturation, extrusion of sealer and intensity of post-obturation pain.[26]
- **One-Appointment Endodontics:** Most patients report little or no spontaneous pain after single-visit root canal therapy; and only 2% may report severe pain.[25]
- **Retreatment:** Endodontists have reported that retreatment cases tend to have a higher incidence of flare-ups. In these cases, host response to extruded filling materials and toxic solvents may be the cause of increased discomfort. Many retreatment cases have associated periapical pathoses alongwith symptoms that increase the likelihood of flare-ups. Technically these cases are the more difficult and time-consuming,; with an increased chance for iatrogenic mishaps.[25]
- **Periapical lesion:** some researchers have noted apical radiolucencies to be related with increased frequency of flare-ups. Pulp of teeth with large periapical radiolucencies have more bacterial strains and may be more infected. These bacteria can cause an acute problems if introduced periapically.[25]
- **Host factors:** Intensity of preoperative pain and amount of patients' apprehension are correlated to degree of postoperative pain. Patients with dental phobias tend to be difficult, because of their low psychophysiologic tolerance. Such patients are best served with some presedation, either oral or intravenous to make their endodontic experience calmer. Other factors that have both positive and negative correlations with flare-ups include patient's age, gender, presence of allergies and tooth position. Race and systemic disease are not associated with increased flare-ups.[26]

Diagnosis

The initial phase of treating the endodontic pain patient is of course diagnosis. For the patient that has recently had an endodontic procedure the diagnosis is often quite simple. However, there are several conditions that have been shown to mimic endodontic or odontogenic pain. Additionally, the current episode of pain may be coming from another tooth, an unrelated sinus or TMJ-related condition or post-injection sequelae. Perhaps the original endodontic diagnosis was incorrect. Obtaining a thorough understanding of the patient's chief complaint should be the first step in proper management.[1]

Gathering information, such as on when the post treatment 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 will assist the clinician in his assessment of the situation. A review of patient's medical and dental history is in order. A thorough clinical examination should be performed. The following conditions should be properly noted: discoloration, areas of swelling, exudation, ulcerations, defective or lost restorations, cracked and/or fractured teeth and any apparent changes in occlusal contacts. Clinical tests should include percussion (both in axial and right-angle direction), apical palpation, bite-stick test, thermal stimulation (cold and hot if indicated) and periodontal probing. The clinician must decide if taking additional radiographs are indicated.[(1)]

Often times, taking additional, properly angulated radiographs may further elucidate the etiology of the current condition. However, interpreting the presence of bone lesions is often difficult. These clinical and radographic tests may reveal that the symptoms as non-odontogenic or as related to another tooth or may in fact be related to recently treated tooth.[(1)]

Treatment

Because all etiological factors cannot be precisely determined, many treatment regimens have empirical advocates for prevention or alleviation of symptoms during root canal therapy. 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. Each treatment has it advocates, but many of the regimens may be successful because of the placebo effect.[(27)]

Conclusion

Endodontic Flare-ups are an undesirable occurrence in the course of endodontic treatment that cause distress to the patient and operator. Clinicians should employ appropriate measures directed at prevention of occurrence of flare-ups and should be able to treat these efficiently when they do occur.

References

1. Weine FS. Endodontic Therapy Fifth edition, Mosby:203-37.
2. Oginni AO, Udoye CI. Endodontic flare ups; comparison of incidence between single and multiple visit procedures in patients attending a Nigerian teaching hospital. BMC Oral Health 2004;4:4.
3. Alacam T, Tinaz AC. Interappointment Emergencies in teeth with necrotic pulps. Journal of Endodontics 2002;28:375-77.
4. Cohen S, Burns RC. Pathway of the pulp, Eighth Edition 2002:61-65.
5. Walton R, Fouad A. Endodontic interappointment flare-ups: a prospective study of incidence and related factors. Journal of Endodontics 1992;18: 172-77.
6. Endodonticinter appointment flare-ups: a prospective study of incidence and related factors. Richard Walton, DMD, MS and Ashraf Fouad BDS, MS Voll. 18, no.4, April 1992.
7. Harrison JW, Baumgartner JC, Zielke DR. Analysis of interappointment pain associated with the combined use of endodontic irrigants and medicaments. Journal of endodontics 1981;7:272-76.
8. Walton R, Fouad A. Endodontic interappointment flare-ups: a prospective study of incidence and related factors. J Endod 1992;18:172-77.
9. Eleazer PD, Eleazer KR. Flare up rate in pulpally necrotic molars in one visit versus two visit endodontic treatment. Journal of Endodontics 1998 Sep;24(9):614-616.
10. Cohen S, Burns RC. Pathway of the pulp, Eighth Edition 2002:61-65.
11. Pickenpaugh I, Reeder A, Beck M, Meyers WJ, Peterson D (2001) Effect of prophylactic amoxicillin on endodontic flare up in asymptomatic necrotic teeth. Journal of Endodontics, 27:53-56.
12. Morse DR, Koren LZ, Eposito JV, et al. Infections flare-ups: induction and prevention, Int J Psychosom 1986; 68(special issue):5-63.
13. Barnett F, Tronstad L. The incidence of flare-ups following endodontic treatment. J Dent Res 1989; 68(special issue): 12-53.
14. Harrison JW, Baumgartner JC, Zielke DR. Analysis of interappointment pain associated with the combined use of endodontic irrigants and medicaments. Journal of endodontics 1981;7:272-76.
15. M. Georgopoulou, P. Ananstassiadis. Pain after chemomechanical preparation. International endodontic journal 1986;19:309-314.
16. Selye H. The part of inflammation in the local adaption syndrome. In: Jasmine G, Robert A, eds. The mechanism of inflammation. Acta Montreal 1953;5:53-74.
17. Samuel Seltzer, Irving J. Naidorf. Flare-ups in Endodontics: 1.Etiological factors. J. of Endodontics 2004
18. Bartals HA, Naidorf IJ, Blechman H. A study of some factors associated with endodontic flare ups. Oral Surg 1968;25:255.
19. Wittgow WC, Saviston CB Jr. Microorganisms from pulp chambers of intact teeth with necrotic pulp. J Endodon 1975;1:168.
20. Griffee M, Patterson SS, Miller CH, Kafrawy AH, De Obarrio JJ. Bacteroidesmelaninogenicus and dental infections. Oral surgery 1982;54:486.
21. Slots J, Gneco RJ. Black pigmented bacteroides species, capnocytophaga species and actinobacillus actinomycetemcomitans in human periodontal disease; virulence factors in colonization, survival and tissue destruction. J Dent Res 1984;63:412.
22. Tanner ACR, Visconti RA, Holdeman LV, Sundqvist GK, Socransky SS. Similarity of Wolinella recta strains isolated from periodontal pockets and root canals. J Endodon 1982;8:294.
23. Mergenhagen SE, Hampp EG, Scherp HW. Preparation and biological activities of endotoxins from oral bacteria. J Infect Dis 1961;108:304.
24. Mid-treatment flare-ups in endodontics: A nightmare for clinicians; Dr. Singhal, Dr. Mohit Sharma: Heal talk 44 Sept-Oct 2010.
25. Cohen S, Burns RC. Pathway of the pulp, Eighth Edition 2002:61-65.
26. Franklin S. Weine: Endodontic therapy: 5th edition:230-235.

27. Dorn SD, Moodnik RM, Feldman MJ, Borden BG. Treatment of the endodontic emergency: a report based on a questionnaire parts 1 and 2. J Endodon 1977;3:94,153.

How to cite this article:

Sharma A, Mandlik J, Varghese R. An overview of etiology of endodontic flare-ups. Ind J Conserv Endod, 2016;1(3):76-80.