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Endodontic Postoperative Pain: Etiology and Related Factors – An Update

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Abstract Endodontic postoperative pain is a common unexpected complication. This problem is not well understood and its incidence varies widely from one study to another. The hypothetical mechanisms for this complication include chemical, mechanical or microbial injury to the periradicular tissue. Instrumentation beyond the apical foramen and apical extrusion of the infected debris seem to be the most important causative factors by stimulation of an acute immune response in the periapical area. The reported incidence of this phenomenon varied from 1.7 to 70%. This wide range of the reported incidence is due to different study designs, pre-operative condition of the teeth, treatment procedure or technique, timing of recording pain experience, index of pain measurement and severity of pain included in the statistical analysis. Although some studies found a correlation between this phenomenon and presence of preoperative pain, presence of periapical lesion, pulpal necrosis, number of visits, technique of instrumentation, age, gender, other studies have failed to find this correlation.. No specific procedure, systemic or localized medication has been found to prevent its occurrence. Knowledge about the etiology of flare-ups and adoption of appropriate preventive measures can significantly reduce the incidence of this highly distressing and undesirable clinical phenomenon.

Keywords: *endodontic flare-ups, postoperative pain, etiology, related factors*

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1. Introduction

Development of pain after endodontic intervention which is known as intra-appointment pain or flare-ups is one of the most common endodontic complications. The unpredictable development of this pain may undermine patients' confidence in the clinician and acceptance of the procedure. The exact definition of endodontic flare-ups varies from one author to another [1,2]. It may be defined as the occurrence of pain and/or swelling following endodontic treatment appointment, requiring an unscheduled visit and active treatment [1]. Endodontic Inter-appointment Emergency is another term used by some authors to describe this complication [2].

Many studies have been conducted in order to identify which factors are associated with this true complication, others have tried to find preventive measures or medicaments for these flare-ups. Unfortunately, the results have varied and there is still no definite procedure or medicament for the prevention of this emergency [3]. This review article tries to improve our knowledge about this type of emergency and this may improve the ability to find possible procedures or medicaments that could help in preventing or minimizing its occurrence. It will also represent the major findings of the previous studies that investigated this stressful condition.

2. Etiology

The factors that may contribute to the sudden appearance of this pain are complex and poorly understood [3]. Most cases of flare-ups occur as a result of acute periradicular inflammation (acute apical periodontitis or acute apical abscess), secondary to intracanal procedures. Acute periradicular inflammation can develop as a result of any type of insult from the root canal space [4].

A number of hypothetical mechanisms that may be responsible for pain and/or swelling during endodontic therapy have been presented by many authors. These include chemical, mechanical or microbial injury to the periradicular tissue.

2.1. Microbial Injury

- **Association with specific microorganisms**:

Most studies on endodontic flare-ups have dealt with the association of certain clinical factors, but there has been little analysis done of the involvement of specific microorganisms [4]. Environmental conditions within the root canal system containing necrotic pulp tissue are conductive to the establishment of several different oral bacterial species, particularly strictly anaerobic bacteria with demanding nutritional requirements [5].

Gomes et al. [6] in 1996 reported that *Prevotella* species were significantly associated with pain.

Black-pigmented anaerobes which includes *Prevotella* and *Porphyromonas* were also the most commonly recovered bacteria from cases with tenderness to percussion [7].

The combined presence of *F. nucleatum* and *Prevotella* species and *Porphyromonas* species is a risk factor for endodontic flare-ups and these organisms, in synergy, are able to worsen a periapical inflammatory lesion [5]. Using the checkerboard DNA-DNA hybridization methodology, Siqueira et al. (2001) found that *Bacteroides forsythus*, *Porphyromonas gingivalis*, *Streptococcus constellatus*, *Prevotella intermedia*, *Fusobacterium nucleatum* and *Fusobacterium periodonticum* are the most prevalent species in cases diagnosed as acute apical abscess [8].

There are some special circumstances in which microorganisms can cause flare-ups:

- **Apical extrusion of infected debris:**

Microbial insult injury caused by microorganisms and their products that egress from the root canal space during mechanical instrumentation to the periradicular tissues is one of the principal causes of inter appointment flare-ups [3]. In asymptomatic chronic periradicular lesions associated with infected teeth, there is a balance between microbial egression from the infecting endodontic micro biota and the host defense in the periradicular tissues. During chemo-mechanical preparation, if the microorganisms are apically extruded, there will be a transient disruption in the balance between extruded irritant and defense in such a way that the host will mobilize an acute inflammation to re-establish the equilibrium [4].

Apical extrusion of the debris seems to be an unavoidable incident in most instrumentation techniques including hand and Niti rotary instrumentation systems with some variety [9,10,11]. Creating a glide path prior to canal instrumentation could reduce the amount of apically extruded debris in curved canals [11].

- **Incomplete bio-mechanical debridement of the root canal space:**

Positive and negative interactions among the members of the microbial community allow the community to be relatively stable and in balance. Ideally, the chemo-mechanical preparation should be completed in one appointment. Incomplete chemo-mechanical preparation can disrupt the balance by eliminating some of the inhibitory species and leaving behind other previously inhibited species, which can then overgrow [5]. If overgrown strains are virulent and/or reach sufficient numbers, damage to the periradicular tissues can be intensified, and this may result in lesion exacerbation [4]. The conditions in which incomplete bio-mechanical debridement could result in post-operative pain include: instrumentation short from the apical constriction; undetectable canals; anatomical complexities of the root canal space such as c-shaped canals, deltas, multiple foramina and furcation accessory canals [2].

- **Secondary intra-radicular infection:**

Secondary intraradicular infections are caused by micro-organisms that penetrate the root canal system during treatment, between appointments or after the end of the endodontic treatment. Introduction of new microorganisms into the root canal system during treatment usually occurs following a breach of the aseptic chain, and the main sources of recontamination include: remnants of dental plaque, calculus or caries on the tooth crown and leaking rubber dam [16]. Microorganisms can also enter the root canal system between appointments, after leakage or loss of the temporary restoration [4].

- **Increase of the oxidation-reduction potential:**

It has been theorized that alteration of the oxidation-reduction potential in the root canal environment can be a cause of exacerbation following endodontic procedures. Entrance of oxygen into the root canal during treatment may favor the overgrowth of facultative bacteria that resisted chemo-mechanical procedures. This theory was based on the microbial isolates from teeth presenting with acute exacerbations during endodontic therapy. All the microbes isolated were aerobic and facultative microbes mainly facultative streptococci and no obligate anaerobes were isolated [12].

- **Viral-bacterial interaction:**

Recent studies have revealed an association between Human Cytomegalovirus and Epstein-Barr virus and the acute exacerbation of periapical lesions. Cytomegaloviurs or Epstein-Barr virus active infections are detected in more than 90% of symptomatic and large periapical lesions [13]. Slots et al. [14] hypothesized that some types of aggressive periapical pathosis develop as a result of a series of interactions among herpes viruses, bacteria, and host immune reactions.

2.2. Mechanical Injury

Instrumentation beyond the apical foramen is one of the iatrogenic causative factors of endodontic post-operative pain [3]. Generally, a distance of 0.5 to 2 mm between the radiographic apex and the end-point of root canal instrumentation is considered acceptable. Actually, what appears to be 0.5 to 2 mm from the radiographical apex may be ending in the periradicular tissue due to x-ray angulations, deviated foramen or superimposed images [15]. Therefore, working length that radio graphically ends 0 to 2 mm short of the apex does not guarantee the avoidance of instrumentation beyond the apical foramen. Radiographic working length measurement should be combined with an electronic determination of the working length [15].

Iatrogenic over instrumentation promotes the enlargement of the apical foramen, which may permit an increased influx of exudates and blood into the root canal [3]. This will enhance the nutrient supply of the remaining bacteria within the root canal. Moreover, in addition to the mechanical injury to the periradicular tissue during over-instrumentation, a significant amount of infected debris will be extruded. This is because over instrumentation usually widens the foramen and infects the periradicular tissues [4].

2.3. Chemical Injury

Irrigation solutions and intracanal medicaments are used within the root canal to clean and aid in disinfecting the dentinal walls. Although these materials are intended to be contained within the root canal, they invariably contact the perapical tissues, either through inadvertent extrusion through the apex or through leaching [16].

Sodium hypochlorite (NaOCl) is the most commonly used irrigating solution in endodontics because of its efficacy for pulpal dissolution and antimicrobial activity [17]. Sodium Hypochlorite is highly toxic at high concentration and tends to induce tissue irritation on contact. In a concentration of 0.5% NaOCl is nontoxic to vital tissues and immediately washed away by the circulating blood [18].

Most complications of the use of Sodium Hypochlorite appears to be the result of its accidental injection beyond the root apex – known sodium hypochlorite accident- which can cause violent tissue reaction characterized by immediate severe pain, rapidly increasing swelling, hemorrhage through the tooth, interstitial bleeding with ecchymosis of the skin and mucosa and, in some cases development of secondary infection and paresthesia [19].

Inadvertent injection of NaOCl beyond the apical foramen may occur when extreme pressure is applied during irrigation or by binding of the irrigation needle tip in the root canal with no release for the irrigant to leave the root canal coronally. If this occurs, the excellent tissue-dissolving capability of NaOCl will lead to tissue necrosis [20].

3. Incidence

The reported incidence of postoperative endodontic pain ranges widely from 1.7% to 70% [21-40]. Although most of the studies are either randomized controlled trials or prospective studies, direct comparison between them is complicated by differences in study design, pre-operative condition of the endodontically treated teeth, treatment procedure or technique, timing of recording pain experience, index of pain measurements and severity of pain included in the statistical analysis (Table 1 and Table 2).

Table 1. Reported incidence of Postoperative Pain (all intensities) by different studies

Author/yr.	Teeth	Incidence(%)	Related factor (s)
Albashaireh (1998) [21]	291	38% multi-visits; 27% single visit	Non-vital pulps
Aqrabawi (2006) [22]	146	8 h: 74%; 1 d: 69%; 2 d: 46%	Hand and rotary instrumentation technique has no diff.
Almeida (2012) [25]	126	3% moderate; 19% mild	No. diff. between NaOCL and chlorohexide irrigation
Byram (2009) [27]	306	69.9% single visit; 69.2% in two visits; 4.6% severe	No diff. between vital and non-vital; no diff. b/n single and multiple visits
Ehrmann (2003) [28]	223	4 h: 37%; 1 d: 23%; 2 d:38%	Ledermix found to be effective in pain control
El Mubarak (2010) [30]	234	12h: 9%	Preoperative pain
Fava (1998) [31]	60	2 days: 2%;	No diff. between $Ca(Oh)_2$ and steroid-antibiotic dressing
Gesi (2006) [32]	256	7 days: 10%	Associated with overfilling
Glennon (2004) [33]	272	64.7% (<10% sever)	Presence of preoperative pain, tooth type, systemic steroid, preoperative swelling
Gotler (2012) [34]	274	63.8% vital; 38.5% necrotic; 48% retreatment	Vital pulps induce more pain
Henry (2001) [35]	41	1 d:56%; 2 d: 33%; 3 d: 27%;	Symptomatic necrotic teeth
Polycarpou (2005) [38]	175	12%	Presence of preoperative pain ; female gender
Risso (2008) [39]	121	10.5% single visit; 23% two visit	Presence of pre-operative pain
Siqueira (2002) [40]	627	Mild pain: 10%, Moderate: 3.3%, Severe: 1.9%.	Previously symptomatic teeth without periradicular lesions
Segura -Egea(2009) [41]	176	37% anterior; 56% posterior	Mandibular >max.; Irreversible pulpitis and acute apical periodontitis
Sadaf (2014) [42]	140	42.9%	Preoperative pain, female gender
Najma (2014) [43]	474	2.53%	Mandibular teeth
Mor (1992) [44]	334	4.2%	Higher in non-vital teeth without apical radiolucency
Torabinejad (1994) [45]	588	6%	Preoperative pain, apprehension, intracanal medication
Ng (2004) [48]	415	40.2% , less than 12% severe	Female, molars, size of periradicular lesion smaller 3mm, single visit
Walton (1993) [49]	80	70% all intensities; low severe	No diff. between administration of antibiotic or non administration

Table 2. Different studies reported incidence of sever postoperative pain –flare ups- with any related factors

Author/yr.	Teeth	Incidence (%)	Related factor (s)
Walton (1992) [1]	946 visits	3.7% flare up	Pulpal necrosis with painful apical pathosis
Alacam (2002) [23]	474	7.17%	More in mandibular
Iqbal (2009) [36]	6580	0.39 %	Presence of a periapical lesion
Al Negrish (2006) [24]	120	2days:11.6% 7days: 3.6%	No. of visits has no effect
Alves Vde (2010) [26]	408	1.71%	Presence of periradicuar radioucency
Oginni (2004) [37]	227	8.1% in multiple visits, 18.3% single visit	Preoperative pain
Eleazer (1998) [29]	402	8% two visits; 3% one visit	Advantage of single visit
Syed (2012) [46]	1328	12h:9%; 24h:8.6%	Old, female, preoperative pain
Udoye (2011) [47]	175	10%	No factors

Table 3. Different studies that compare post operative pain in single-visit and multiple-visits root canal treatment

Study	Finding
Oginni A (2004) [37]	Higher incidence in single visit
Eleazer (1998)[29]	Higher in two visits
Albashaireh (1998) [21]	Higher in multiple visits
Wong AW (2015) [52]	No significant difference in the incidences of post-obturation pain
Bhagwat (2013)[53]	Less in single-visit
Yoldas , Topuz (2004) [54]	Less flare ups in multiple-visits
Jabeen (2014) [55]	Higher incidence of post-obturation pain following the single visit root canal treatment
Byram (2009) [27]; Rao (2014) [50]; Wang (2010) [51] DiRenzo A. (2002) [56]; Al-Negrish (2006) [24]; El Mubarak (2010) [30]; Akbar (2013) [57]; Iqbal (2009) [36]	No difference

4. Related Factors

4.1. Number of Visits

Considerable controversy exists over the question of whether it is preferable to complete endodontic therapy in one appointment or multiple appointments. Many practitioners prefer a two-visit approach to ensure a post-debridement, symptom-free period before canal obturation. Leakage of the coronal filling, re-infection from periapical or periodontal pathogens, failure to kill intra-canal or intratubular bacteria, and ease of treatment for ensuing infections are frequently mentioned in the controversy.

4.2. Retreatment

Retreatment has been suggested as a contributing factor in post-treatment complications. A reported increased incidence of inter-appointment emergencies after retreatment in comparison with the incidence after initial root canal treatment was reported by Torabinejad [2] and Imura[58]. In contrast no difference regarding the incidence of postoperative pain between treatment and retreatment has been found by many authors [59,60].

4.3. Presence of Pre-operative Pain

The presence and severity of pre-operative pain appear to have a strong influence on the development of pain after initial root canal preparation [1,30,33,37,38,39,40,42]. This may be explained by that the virulent clonal types of pathogenic bacteria species that are present in the root canal system are responsible for the pre-operative symptoms. Extrusion of these virulent microorganisms to the periradicular tissue during instrumentation will have the potential to cause or exacerbate periradicular inflammation than non-virulent microorganisms that didn't cause a pre-operative symptoms [4].

4.4. Pulp & Periradicular Condition

Necrotic pulps usually provide an environmental condition that is conductive to the establishment of several different oral bacterial species, particularly strictly anaerobic bacteria [4]. Since microorganisms are the major causative agent of acute peri-radicular inflammation, regardless of whether it develops pre-operatively or post-operatively, it is logical to suggest theoretically that most post-operative pain occurs after instrumentation of necrotic pulps compared to vital pulps. Positive relationship between pulpaly necrotic teeth with painful apical pathosis and flare-up rate was found in most of the studies (Table 4).

4.5. Intra-canal Medicaments

Siquiera et al. (2002) [40] stated that the intra-canal procedures based on the maximum elimination of the irritant including antimicrobial intra-canal dressing are valuable tool to control endodontic infections and theoretically prevent postoperative pain.

Table 4. The relationship between the vitality of the pulp/periradicular condition and the frequency of post operative pain

Study	finding
Mor (1992) [44]	Highest occurrence of inter appointment pain in non-vital teeth unassociated with periapical radiolucency
Walton (1992) [1]	Positive correlation with pulp necrosis with painful apical pathosis
Trope (1991) (62).	Positive correlation with pulp necrosis with painful apical pathosis
Imura (1995) [58]	No significant difference between non-vital and vital teeth but periradicular radiolucency was positively related to flare-ups.
Alacam (2002) [23]	No relation between flare-ups and periapical lesions
Gotler (2012) [34]	Higher incidence in vital pulps than necrotic and retreatment cases
Iqbal (2009) [36]	The presence of a periapical lesion was the single most important predictor of flare-ups.
Bhagwat (2013) [53]	Non-vital teeth with periapical radiolucency exhibited less pain as compared with non-vital teeth without periapical radiolucency.
Byram (2009) [27]	No difference between vital and non-vital teeth
Alves Vde (2010) [26]	Correlation between the flare-up rate and periapical radiolucency.
Henry M (2001) [35]	More post operative pain in symptomatic necrotic teeth

4.5.1. Calcium Hydroxide [$Ca(OH)_2$]

$Ca(OH)_2$ has been found to be quickly and highly effective against some microorganisms related to severe clinical symptoms[63]. Despite the proven antimicrobial activity of $Ca(OH)_2$, many studies showed its limited effect on post-treatment pain after the chemo-mechanical root canal preparation [64,65] (Table 5).

4.5.2. Steroids

According to the findings of the clinical studies performed to correlate the steroid intracanal medicaments and postoperative pain, there is no clear direct preventive effect of these medicaments to endodontic flare-ups. They may have an anti-inflammatory effect but this reduction in inflammation only shows some effect in reducing the severity of this pain after some hours (Table 5).

4.6. Irrigating Solutions

The type of irrigating solution used makes little difference in the incidence of post-operative discomfort, providing that the irrigating solution is not forced beyond the foramen of the tooth [1]. Since the induction of pain in endodontic therapy is multi factorial, it is difficult to attribute a lower pain incidence specifically to the use of any particular irrigant. Bashety K (2010) reported more pain in teeth irrigated using 5.25% sodium hypochlorite when compared to the pain in teeth irrigated using 2% chlorhexidine solution[72]. In contrast, Almeida et al. (2012) found no difference between post operative pain in teeth irrigated with NaOCl or chlorohexidine solutions [25].

4.7. Systemic Medication

4.7.1. Systemic Steroids

Administration of systemic steroids for the purpose of prevention post operative pain was investigated by many authors with variant results. In addition to the oral administration of steroids, local injection around the treated tooth i.e. intraligamentally or supraperiosteal injection are also investigated (Table 6). Marshal JG in his 2002 review stated that the administration of systemic steroids is efficacious as an adjunct to but not a replacement for appropriate endodontic treatment in the attenuation of endodontic post treatment pain. Systemic steroids are also highly effective in those patients who present for treatment with moderate/ severe pain and a clinical diagnosis of pulpal necrosis with associated periapical radiolucency [73].

Table 5. Different studies investigated the relationship between intra-canal medicaments and postoperative endodontic pain

Author	Teeth/diagnosis	Medicament tested	Finding
Siqeira (2002) [40]	627/ necrotic pulps	$Ca(OH)_2$ / para-monochlorophenol paste	Low incidence of flare-ups after dressing with $Ca(OH)_2$ /camphorated para-monochlorophenol paste.
Ehrmann (2003) [28]	Necrotic with acute apical periodontitis	$Ca(OH)_2$; Ledermix; placebo	Ledermix is an effective intracanal medicament for the control of postoperative
Richard (2003) [63]	140	$Ca(OH)_2$ intracanal dressing	Unrelated to the incidence and/or severity of post treatment pain
Anjaneyulu (2014) [65]	Review	$Ca(OH)_2$	No clear evidence of its effect on post-treatment pain
Trope (1990) [62]		$Ca(OH)_2$; Ledermix; placebo	No significant difference among the three intracanal medicaments.
Menakaya (2015) [66]		Mixture of saline/$Ca(OH)_2$ vs. chlorohexidine paste	Postoperative pain was lower in the normal saline/ $Ca(OH)_2$ treatment group
Moskow A. (1984) [67]	50	Dexamethasone solution	Significant reduction of pain at 24 h.
Negm (2001) [68]	988	Corticosteroid antibiotic combination	Complete relief of pain after 24 h. (pain reduction)
Rimmer (1991) [70]	Non vital teeth	$Ca(OH)_2$, Ledermix, formocresol intra-canal med.	Lower flare-up scores with steroid/antibiotic mixtures
Fava (1998) [31]	60 incisors with acute apical periodontitisi	$Ca(OH)_2$, steroid antibiotic combination	No difference in pain incidence and severity between the two intracanal medicaments.
Walton (2003) [69]	140	$Ca(OH)_2$ vs. sterile cotton pellet	$Ca(OH)_2$ was unrelated to the incidence and/or severity of post treatment pain.
Singh (2013) [71]	64 teeth, necrotic pulps and acute apical periodontitis	$Ca(OH)_2$ paste with chlorhexidine gel; 2% chlorhexidine gel; $Ca(Oh)_2$	Chlorhexidine alone and $Ca(Oh)_2$ plus chlorhexidine gave rise to less pain than calcium hydroxide alone or no dressing at all.

Table 6. Different studies investigated the effect of systemic steroids on post operative endodontic pain

Author	Systemic steroid/dose	Finding
Marshal (2002) [73]	4 mg of dexamethasone intramuscular	Significantly reduced both the incidence and severity of pain after 4 hours after single-visit endodontic therapy
Krasner (1986) [74]	0.75 mg dexamethasone or placebo orally	Administrations of oral dexamethasone significantly reduced post-operative pain after 8 and 24 hours
Elieazer (1994) [75]	methylprednisolone intra-ligamentally	Significantly reduced the frequency and intensity of postoperative pain
Merhrvarzfar (2008) [76]	Supraperiosteal injection of dexamethasone	Could be effective in reduction or prevention of postoperative pain during the first 24 h.
Jalalzadeh (2010) [77]	Prednisolone orally (30 mg) before RCT; placebo	Reduced postendodontic pain.

4.7.2. Systemic Antibiotics Prophylaxis

Since the major causative factor of endodontic flare-ups is bacterial infection to the periradicular tissue, it has been thought that prescribing an antibiotic prophylactically could prevent the occurrence of this complication. The use of prophylactic antibiotic for healthy patients for the purpose of prevention of flare-ups is a controversial issue because the over prescribing of antibiotics may lead to bacterial resistance and patient sensitization. Data from controlled clinical trials provide little to no support for the hypothesis that antibiotics reduce post-operative pain (Table 7).

4.7.3. Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

NSAIDs have been used increasingly as analgesics rather than anti-inflammatory agents. If the periapical inflammatory reaction is a major contributor to post-treatment pain, then it is possible that a non-steroidal anti-inflammatory drug may be useful in its management. The use of pre-treatment or post-treatment analgesics may significantly reduce the incidence of flare-ups [45,84,85].

4.8. Technique of Instrumentation

All instrumentation techniques are reported to cause apical extrusion of debris, even when preparation is maintained short of the apical foramen [9,10,11]. The difference resides in the fact that some techniques extrude more debris than other do. Crown-Down techniques using instruments with some sort of rotary action combined with abundant irrigation at least theoretically have the potential to reduce the risk of flare-ups [4,9,91].

Mechanical preparation using rotary instruments appears to generate less post-operative pain than hand instrumentation [92,93]. In contrast Aqrabawi et al. reported no statistically significant difference in post-treatment pain between teeth cleaned and shaped with manual or Niti rotary techniques [22].

Creating a glide path prior to canal instrumentation could reduce the amount of apically extruded debris in curved canals [11].

The use of a patency file during all stages of canal preparation may improve the removal of debris from the last millimeters of the root canal space and it maintains the canal unblocked. Therefore, the drainage of the inflammatory exudates is more easily achieved, decompressing the tissues and allowing for the entry of oxygen, which is lethal for anaerobic flora. It favors the placement of the dressing in direct contact with the living periapical tissues [31]. There was less post endodontic pain when apical patency was maintained in non vital teeth. [94]

Regarding working length determination method, Kara (2014) found no difference in postoperative pain between working length measurement using an electronic apex locator or digital radiography [95].

Table 7. Administration of different prophylactic antibiotics on post operative endodontic pain

Author	Teeth	Antibiotic	Finding
Henry (2001) [35]	Symptomatic necrotic teeth	Penicillin 500 mg capsules for 7 days	Did not reduce pain, percussion pain, swelling,
Walton (1993 [49]		Prophylactic penicillin	No difference between administration or non-administration of antibiotics after 48h.
Fouad (1996) [78]	Pulp necrosis; apical periodontitis	Penicillin and placebo	No difference was found between the groups of administration and non-administration of penicillin
Pickenpaugh (2001) [79]	Symptomatic necrotic teeth	3 mg prophylactic amoxicillin	No effect on the endodontic flare-up
Torabinejad (1994) [45]		Erytrhomycin; Methyl-prednisolone plus penicillin	All are more effective than placebo within the first 48 h following RCT.
Morse (1990) [80]	necrotic pulp / chronic apical periodontitis	single dose - long acting 1 gm tablet of cephalosporin	prophylactic administration of antibiotics significantly reduced the incidence of flare-ups
Abbott (1988) [81]	pulpal necrosis / peri-apical pathosis	Penicillin and erythromycin	Low incidence of flare-ups, with no difference between penicillin or erytrhomycin
Alsomadi (2015) [82]		Ibuprofen 400 mg before; same regimen + amoxicillin	Antibiotic prescription could result in less post endodontic pain

Table 8. Different studies investigate systemic prophylaxis of NSAIDs on post operative pain

Author	NSAID	Finding
Walton and Fouad (1991) [1]		Analgesics and anti-inflammatory drugs found to have a significantly higher incidence of flare-ups
Torabinejad (1994) [45]	Ibuprofen	More effective than placebo in controlling pain
Menhinick (2004) [83]	600 mg ibuprofen and 1000 mg acetaminophen	More effective than ibuprofen alone for the management of postoperative endodontic pain
Monke (2000) [84]	Prophylactic etodolac Ibuprofen and placebo	Ibuprofen was more effective in pain reduction than etodolac and the placebo.
Gopicrishan (2003) [85]	Rofecoxib, ibuprofen, or placebo prophylacticaly	Both rofecoxib and ibuprofen provided better pain relief than placebo at 4 and 8 h.
Nekofar (2003) [86]	Meloxicam; piroxicam; placebo	No significant differences between the three groups
Attar (2008) [87]	Ibuprofen before RCT	Will not reduce postoperative pain
Negm (1989) [88]	Piroxicam and diclofenac	Piroxicam and diclofenac are superior to the placebo in controlling postoperative pain.
Metri (2016) [89]	Diclofenac 30 minutes before RCT	Post-endodontic pain was substantially reduced
Mokhtari (2016) [90]	Indomethacin 25 mg or 400 mg ibuprofen	Ibuprofen and indomethacin significantly reduced the postoperative pain.

4.9. Psychological Factors

Fear of dentists and dental procedures, anxiety, apprehension, and many other psychological factors influence the patient's pain perception and reaction thresholds. Torabinejad and his colleagues [45] noted an association between the presence of apprehension before endodontic treatment and post-operative pain.

4.10. Gender, Age, Tooth Location and Type

The influence of patient age, gender or tooth/arch group on flare-up occurrence reported to be of no significance in many studies [1,58,80].

In contrast, a retrospective study conducted by Torabinejad et al. [2] showed a significant positive correlation of flare-ups with patients aged between 40 and 59 years, female patients and mandibular teeth. Mandibular molar teeth appears to be a greater risk associated with postoperative pain than maxillary [2,23,33,41,42,43,61]. The reason for this may be that the mandible has a cortically thicker plate than the upper jaw, and this may cause the accumulation of exudates, which causes more pressure compared with the maxilla [23].

Females were found to have a higher risk for developing post operative pain in many studies [2,42,61] while other studies didn't find any relationships between gender and post operative pain [33,43].

5. Prevention

Clinical procedures:

Although there are no specific clinical procedure or medication found to prevent endodontic post-operative pain, some clinical tips need to be followed in an effort to minimize its occurrence. These include the following:

- Maintaining the aseptic condition during intracanal procedures: Asepsis is paramount in endodontic therapy to prevent infection in vital cases or introduction of new microbial species in cases of infected necrotic pulps. Thus, clinicians should be aware of the need to perform endodontic treatment under strictly aseptic conditions as some cases of secondary infections may even be more difficult to treat than primary infections and may cause flare-ups, persistent symptoms and /or failure of the root canal treatment [4].
- Selection of instrumentation techniques that extrude less amounts of debris apically.
- Completion of the biomechanical debridement of the root canal space in the first visit: Maximum removal of irritants from the root canal system may reduce the risks of inter-appointment discomfort caused by the remaining virulent microorganisms [3,40].
- Use of an anti-inflammatory or anti-microbial intracanal medicament between appointments in the treatment of infected cases [28,40,68,71]
- Instrumentation to 1-2 mm shorter than the radiographical apex and confirming the working length by electronic apex locators [15,93]. In cases of infected canals exhibiting periapical involvement and pre-treatment symptoms, where the possibility of developing flare-ups is high, the clinician should take maximum care to confine the instrumentation short of the radiographic apex avoiding extrusion of debris from the canal into the periradicular tissues [58].
- Psychological preparation of patients: (particularly those with preoperative pain) will decrease inter-appointment symptoms in the mild to moderate levels [45].
- Avoidance leaving the root canal open for the purpose of drainage. Leaving the tooth open is the most direct way to permit the re-infection of the root canal system in addition to overcome any previous attempts to eradicate microorganisms within root canal system [4].
- To minimize NaOCl accidents, the irrigating needle should be placed short of the working length, fit loosely in the canal and the solution must be injected using a gentle flow rate. Constantly moving the needle up and down during irrigation prevents wedging of the needle in the canal and provides better irrigation. The use of irrigation tips with side venting reduces the possibility of forcing solutions into the periapical tissues [19,20].

6. Conclusion

The reported incidence of endodontic post operative pain varied widely. The severe intensity of pain or flare ups is of low incidence. The etiology of this pain is linked primarily to microbial injury to the periapical area due to extrusion of the infected debris during canal instrumentation. There are no clear evidence to support any technique, intracanal or systemic medication that can prevent the occurrence of this stressful complication. Some local or systemic medications found to control the pain rather than preventing it. However, the clinician should be motivated to follow some guidelines and adopt some clinical procedures that have the potential to reduce the incidence of flare-ups.

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