Pain and flare-up after endodontic treatment procedures
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SUMMARY

Flare-ups can occur after root canal treatment and consist of acute exacerbations of an asymptomatic pulpal and/or periradicular pathologic condition. The causative factors of interappointment pain encompass mechanical, chemical, and/or microbial injury to the pulp or periradicular tissues. Microorganisms can participate in causation of interappointment pain in the following situations: apical extrusion of debris; incomplete instrumentation leading to changes in the endodontic microbiota or in environmental conditions; and secondary intraradicular infections. Interappointment pain is almost exclusively due to the development of acute inflammation at the periradicular tissues in response to an increase in the intensity of injury coming from the root canal system. The mechanical irritation of apical periodontal tissue is caused by overinstrumentation of the root canal and filling material extrusion through the apical foramen. Incorrectly measured working length of the root canal has inherent connection with these causative factors of endodontic flare-up. This review article discusses these many facets of the flare-up: definition, incidence causes and predisposing factors.

Key words: endodontic treatment, flare-up, acute exacerbation, postoperative pain, root canal infection.

INTRODUCTION

The primary aim of endodontic treatment is biomechanical preparation of the root canal (cleaning, shaping and disinfection) and to hermetically seal it with no discomfort to the patient, and provide conditions for the periradicular tissues to heal (1, 2). During endodontic treatment, which follows the treatment protocol adopted by the European Society of Endodontology (ESE), some patients experience flare-ups of endodontic disease (3). A flare-up can be defined as pain and/or swelling of the facial soft tissues and the oral mucosa in the area of the endodontically treated tooth that occur within a few hours or a few days following the root canal treatment, when clinical symptoms (tooth pain when biting, chewing or by itself) are strongly expressed and the patient visits a health care institution sooner than scheduled (1, 4-12). After endodontic treatment the flare-up manifests as pain of various intensity (13). The quantitative evaluation of pain is carried out by using special measurement scales to assess the intensity of the pain. The widely used Visual Analogue Scale (VAS) displays a continuous line with numbers from 1 to 100 placed along the line which represent the intensity of pain (14). The intensity of pain can be measured more accurately when more than one scale is used. Therefore, the Facial Grimace Scale (FGS) – a sequence of five facial grimaces, representing different moods, is used together with VAS (15). When evaluating on the Visual Analogue Scale, the intensity of post-endodontic pain ranges from 5 to 44 points (16), lasts less than 72 hours and responds well to non-steroidal anti-inflammatory drugs and acetaminophen (17). According to research data, the flare-up rate after endodontic treatment varies from 1.4% to 16% (18, 19) and up to 50% in some researches (1, 11, 19). The frequency of post-endodontic pain and flare-up varies in different publications due to differences in study types (prospective and retrospective) and methodology, time of tooth pulp and apical periodontitis diagnosis and the moment the pain was recorded, the clinical experience of the dentist and his/her practical skills (4, 20, 21).

CAUSES OF FLARE-UPS

If during endodontic treatment the periradicular tissues are damaged during the manipulations in the
root canal, then an acute inflammatory response, called a flare-up, begins. Even though the flare-up activates the defensive system of the body which starts fighting the infection, the flare-up brings also about undesirable effects for the patient – pain and swelling (18).

The origin of the post-endodontic flare-up is poli-
etiological; mechanical, chemical and microbial factors influence its development (18, 19, 22-26). Regardless the type of the factor, the flare-up depends on the extent of the periradicular tissue injury, its severity and intensity of the inflammatory response. These factors are interrelated and directly interdependent (18).

Microbial factors
Microorganisms in the root canal system take part in the pathogenesis of asymptomatic apical peri-
odontitis and together with virulent factors they are able to enter periradicular tissues. Various species microorganisms proliferate in the apical area of the root canal. Microbial density in 5 mm of the apical root area may reach up to 106 bacteria, with pre-
dominating anaerobic microorganisms (27). Because of its complicated anatomy (accessory canals, apical deltas) and high bacteria density, the apical root ca-
nal area is said to be “dangerous” for the pathogenic bacteria, the host and the dentist.

In a case of asymptomatic apical periodontitis there is a balance between infectious microflora and defensive mechanisms of human immune system in the periodontal tissues (22). This phenomenon is called "local adaptation syndrome" in the scientific literature (24). During the chemomechanical preparation of the root canal after extrusion of infected debris from apical foramen to periradicular tissues, the inflammation is increased due to imbalance between microorganisms and human immune system caused by irritants get-
ting in the apical periodontal tissues: vessels dilate, their permeability increases and inflammation cell chemotaxis begins (3, 18). Its intensity depends on the virulence of microorganisms and their amount in the periodontal tissues (3). 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 (3). Specificity in anaerobic infections is low and numerous combinations of normally low virulent oral bacterial species have the capacity to induce an acute infection in the root canal and peri-
apical tissues. The low virulence is compensated by the increase in numbers by the growth and multiplica-
tion and by the polymicrobial nature of the primary endodontic infection. The concomitant outgrowth of bacteria through apical foramen into the periradicular tissues cannot be prevented since the bacteria are in an active growing phase, sometimes even stimulated by host factors such as blood components and serum (27, 28). If the root canal is not adequately chemomechani-
cally prepared and between visits is not filled in with intracanal medicaments, the synergistic interaction of microbes in the root canal changes therefore activating virulence genes of pathogenic strains and that causes increased inflammatory response (18, 22). If aseptic rules are not followed during the endodontic treatment, insufficient patient mouth hygiene, working without rubber dam system, uncleansed carious tissue or old non-hermetic filling and secondary infection in the root canal can be a cause of post-operation pain and flare-up. Between visits microorganisms can also enter the root canal through non-hermetic temporary filling or in case of it falling out. After endodontic treatment infection might enter through temporary coronal filling left for longer than two weeks or through non-hermetic and cracked permanent coronal restoration (29).

Mechanical factors
During asymptomatic apical periodontitis root canal system of the tooth is infected therefore micro-
organisms are able to reach the apical third of the root canal, apical foramen and apical deltas. Chemo-
 mechanical preparation is one of the factors causing success of endodontic treatment. During it pieces of debris, necrotic pulp masses, irrigative solutions and microorganisms from root canal access apical periodontal tissues and causes inflammation and post-
operative pain that disturbs healing of periradicular tis-
 sues (30). Despite chosen technique, during mechani-
cal formation of root canal some amount of infected debris are extruded into the periodontal tissues (28, 31). Studies show that minimal amount of extrusion of debris through the apical foramen is reached us-
ing crown-down technique with engine-driven Ni-Ti systems (19, 25, 28, 32). Comparable study performed by Reddy and Hicks shows that cleaning canals with hand endodontic instruments using step-back tech-
nique, average amount of extrusion of debris into the periradicular tissues is 2.58 mg, while using NiTi rotational mechanical instruments with crown-down technique it is less than 0.5 mg (10). While perform-
ing the chemomechanical preparation of root canal it is essential to reach the end point of root canal which is the physiological apex of the root – the conjunction of cementum and dentine. The mechanical irritation of periradicular tissues is caused by overinstrumenta-
tion of the root canal and filling material extrusion through the apical foramen (30). One of the iatrogenic factors causing the flare-up of the endodontic treat-
ment is incorrectly measured working length of the root canal (WL) (33). WL is a distance between the highest chosen point of the coronal part of the tooth and the conjunction of cementum and dentine called the physiological apex of the root which is the place where the chemomechanical preparation and filing of the root canal has to be finished (30, 34). Lange-land estimated that the conjunction of cementum and dentine in the area of apex is localized in the distance of 0.5-3 mm from the visible anatomical apex of the root and moderately in the distance of 1-2 mm from the radiological apex of the root (21). The Brunton et al study results show that when the tip of endodontic instrument working part is withdrawn 1 mm from the radiological apex of the root, the physiological apex of the root is correctly localized only 16% of cases. Hassanien et al determined that when the tip of endodontic instrument working part is 0.5 mm from the radiological apex of the root, WL is too short therefore it does not the conjunction of cementum and dentine; the Welk et al study shows that 19-25% of cases WL was too long and goes below the physiological apex of the root (35). If WL measured is too long, the apical constriction in the area of physiological apex of the root is destroyed, infected debris and filling material of the canal are extruded to the periodontal tissues, periodontal tissues is being mechanically stimulated and exudation and blood enters the canal, therefore microorganisms left in the root canal can multiply and proliferate in the beneficial conditions (18, 20, 36). The apical constriction is not present when roots are not fully formed, also it might be resorbed due to inflammation of periradicular tissues or iatrogenically destroyed by incorrectly measured WL, recapitulation and drainage of apical abscess through the root canal (4). If WL measured is too short, pulp remnants and bacteria are left in the apical third of the canal therefore success and prognosis of endodontic treatment is significantly decreased (30, 36). When the WL of dental root canal is measured radiologically, its accuracy is determined by anatomy of the tooth, place of the apical foramen, curvature of the root canal and the dental radiological examination technique (35). WL measuring by the dental radiograph depends to the condition of the root and periodontal tissues according to Weine: -1 mm from the radiological apex of the root, if no alveolar bone and root resorption is detected; -1.5 mm from the radiological apex of the root, if alveolar bone resorption is detected; -2 mm if alveolar bone and root resorption is detected (37). It is impossible to localize the conjunction area of cementum and dentine according to radiological WL evaluation technique, also there might be a distortion of radiological views and roots and adjacent structures might cover one another, therefore it is essential to combine radiological data with the results of electronic apex locator (AL) (36). Using modern AL, apical constriction area is measured accurately and reliably, eg. Root ZX (J. Morita Co., Tokyo, Japan) accuracy of measuring the working length (+/-5mm) is 97.37%, Raypex 5 (VDW, Munich, Germany) 80-85.59%, Elements – Diagnostic (SybronEndo, Sybron Dental, Orange, CA, USA) 94.28% (34). Accuracy of measuring the working length is determined by the sizes of endodontic instrument, root canal and apical foramen (38).

There are two controversial opinions about the need of recapitulation during the chemomechanical preparation of the root canal (39). Recapitulation is being done by the flexible k-file instrument (#6, #8, #10), which enters the canal deeper than the measured WL, i.e. below the physiological apex of the root (40). Main advantages of this method are avoiding obstruction of the apex with debris and improving access of the irrigation solutions in the apical third of the root canal, however main disadvantage of the recapitulation is mechanical irritation of the periodontal tissues and extrusion of debris and microorganisms into the apical periodontal tissues. The inflammation of the periodontal tissues and post-operation pain can be caused even by extrusion of #10 k-file instrument through the apex (41).

Chemical factors
Irrigation solutions, intracanal medicaments, root fillings and substances, that are in their composition, used in endodontic treatment might be toxin therefore they cause chemical irritation and post-operation pain and sensitivity after entering the periradicular tissues. Pastes that are used with gutapercha for filling the root canal have different level of toxicity by the time they consolidate. The more filling from the root canal is extruded to periodontal tissues, the more intense inflammatory reaction is (18). Some researches show that flare-ups are often after endodontic retreatment of teeth filled with resorcinol – formaldehyde resin (42). Pastes containing formaldehyde are cytotoxic, can cause necrosis after contacting live tissue and extruded into apical periodontal tissues initiate inflammation which causes pain and swelling. If formaldehyde is excluded as by-product during consolidation, periodontal tissues are damaged temporarily, though it is insoluble and might be only surgically eliminated (43).

RISK FACTORS CAUSING FLARE-UP AFTER ENDODONTIC TREATMENT

Some studies show that development of the flare-up is caused by two groups of risk factors after
extensive procedure: 1) risk factors depending on a patient such as demographics, general state of health, condition of the pulp and apical periodontal tissue, clinical symptoms, tooth which is being treated, 2) risk factors associated with therapeutic procedures that are one and/or several visits during the treatment, primary endodontic treatment/retreatment and intracanal medicaments (44).

**Demographics**

Studies on evaluating the probability and intensity of the pain occurring after treatment show that patient is not a significant factor in development of the flare-up (21, 26, 44-46). ElMubarak et al show opposing results, assessing that post-operative pain was more common among younger patients (18-33 years old) (29). Flare-up and post-operative sensitivity 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 and decreased blood flow in the alveolar bone resulting in weaker inflammatory response (26).

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 (21, 29, 44). Pain threshold and toleration depend on sexual hormones and their proportion during different stages of menstrual cycle (1, 8). 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 female (1, 8, 46).

**General state of health**

Flare rate after endodontic treatment procedures is low in patients using systemic steroids as treatment for systemic diseases. Steroids suppress the acute inflammatory response during the chemomechanical preparation of the root canal when mechanical, chemical and/or microbial factors irritate the apical periodontal tissue (21). Torabinejad et al points that patients tendency to allergies is associated with development of a flare-up after endodontical treatment, however Wolton and Fouad study disproves this hypothesis (44, 47).

**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, 3, 6, 7, 19, 23, 24, 30, 44, 48-51). Bone destruction which is visible in dental radiograph is said to be a risk factor of post-operative pain and flare-up (29, 44). Chance of a flare is 9.64 times higher when the bone destruction is detected (33). The connection between size of the bone destruction area and post-operative pain was defined by Genet et al: bone destruction of 5 mm and more is said to increase the probability of pain occurring (46).

**Clinical symptoms**

The next factor determining the post-operative pain is clinical symptoms that were before the treatment such as tooth pain when biting, chewing or by itself and sensitivity to percussion (44). 80% of patients who feel tooth pain before the beginning of the treatment usually feel the pain and after it (13, 17, 21). Pain enhances the stress level in the body and effects immune function in a negative way therefore increasing the probability of a flare-up (44).

**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 (46). Higher frequency of pain in the lateral teeth type is determined by the complicated complex anatomy of the root canals and chemomechanical preparation (21, 29, 46).

**One and/or two visits during the treatment**

Primary endodontic treatment when the pulp is viable or endodontic retreatment when there are no visible clinical symptoms related to the changes in periradicular tissues, chemomechanical preparation and filling of the root canal is done by one visit (13, 52, 53). If the pulp is necrotic and there are radiological changes in periradicular tissues, endodontic treatment is done by two visits: during the first visit the root canal is prepared chemomechanically, filled with intracanal medicaments for maximal root canal disinfection and the crown is hermetically sealed with temporary filling while during the second visit the filling of the root canal is performed (13, 25, 52, 53). Studies show that there is no direct link between manifestation of the post-operative pain and amount of the visits during the endodontic treatment (54, 55). However some studies show controversial results, i.e. that pain is more common after one visit endodontic treatment (56-58). 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 (13).

**Intracanal medicaments**

Antimicrobial intracanal medicaments are essential when controlling the endodontic infection due
to the insufficient amount of microorganisms that are eliminated during the chemomechanical preparation of the root canal (23). While studies show that there is no direct link between the usage of intracanal medicaments during visits and frequency of the pain (21, 28), Harrison et al. studies show contrary that antimicrobial intracanal medicaments reduce postoperative pain caused by microorganisms that are left in the root canal and secondary infection (21). 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 (3). Langeland et al. study shows that corticosteroids are effective in soothing the pain: pain was decreased in few minutes or hours in teeth with signs of asymptomatic pulpitis after the pulp is extirpated and the canal is filled with "Ledermix" paste (59). Corticosteroids supress the inflammatory response due to decreasing the permeability of the vessels and polymorphonuclear cells and leucocytes, as well as phagocytosis and inhibiting the formation of arachidonic acid therefore blocking the cyclooxygenase, lipoxygenase and the synthesis of prostaglandins and leukotriens (45, 59). However together with decreased phagocytosis reaction and protein synthesis corticosteroids also slow down the healing in periradicular tissues (11, 26, 60).

**CONCLUSIONS**

The flare-up rate after endodontic treatment is 1.4-16%. Its origin is polietiological; its development is mostly influenced by mechanical, chemical and microbial factors. These factors are interrelated and directly interdependent. Incorrectly measured working length of the root canal is a mechanical factor causing the damaging effect of the chemical and microbial factors to the apical periodontal tissue. Development of the flare-up after the endodontic treatment procedures is also influenced by demographics, general state of health, condition of the pulp and apical periodontal tissue, clinical symptoms, tooth which is being treated, number of visits during the treatment and intracanal medicaments.

**REFERENCES**


