In the name of God

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An in vivo comparative study of antibacterial efficacy of NaOCl with normal saline followed by 2% IKI solution as final rinse in endodontic treatments

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Abstract

Background: Bacteria play a central role in the development and progression of pulp and periapical disease. It is believed that effective debridement of the root canal system prior to sealing by means of chemomechanical preparation is the key to long-term success of endodontic therapy. In order to achieve this goal, various irrigants are used which Sodium hypochlorite (NaOCl) is amongst the favorite one.

Despite the positive aspects of NaOCl, it also has some known clinical disadvantages which can make someone to search for other alternatives.

There are some advantages for the clinical use of Iodine potassium iodide (IKI) as a root canal disinfectant over NaOCl such as lower toxicity, more tolerable odor and taste, and nonbleaching.

It has acceptable antimicrobial properties, and a 2% solution has lower toxicity to tissue culture cells. Besides, it can penetrate deeper than 1000μm of dentin for 5 minutes.

In spite of its promising results from in vitro studies, published clinical studies have been insufficient. So, the current study was aimed at comparing the antibacterial activity of NaOCl with normal saline followed by 2% IKI solution as final rinse in infected teeth.

Materials and methods: For this purpose, thirty single-rooted teeth with necrotic pulps were selected according to inclusion/exclusion criteria and divided in to two groups randomly. In group I, 2.5% NaOCl was an irrigant during instrumentation and in group II; normal saline was used followed by a 5 minute of 2% IKI final rinse. Bacterial

samples were taken before treatment (S_1), after instrumentation (S_2) and after final rinse of IKI in group II (S_3).

Results: Results revealed that bacteria were present in all initial samples. Both NaOCl and Normal saline were able to significantly reduce the number of CFU from S_1 to S_2 (90 % and 43% respectively). After chemomechanical preparation using NaOCl (group I), 2 of the 15 canals (13.3%) showed negative culture but no cases in group II rendered bacterial free.

Following treatment by using IKI final rinse in group II, an approximate 52.7% increase in bacterial count was seen from S_2 to S_3 although 15% decrease (non-significant) from S_1 to S_3 was also evident. It means that this protocol of using IKI did not offer any additional advantages over irrigation with sole normal saline (15% versus 43% reduction from initial bacterial count which had no statistically significant difference).

Conclusion: 2.5% NaOCl irrigation could not render all canals free of bacteria but was significantly proven superior to either sole irrigation with normal saline or irrigation with normal saline followed by 2% IKI final rinse.

Key words: IKI, NaOCl, Normal saline, Irrigant, Endodontics

1. Introduction

Microorganisms and their by-products are considered to be the major cause of pulp and periradicular pathosis (1-6). Hence, the major objective in endodontic therapy is to disinfect the entire root canal system by eliminating any possible source of infection (7). Moreover, the outcome of endodontic treatment largely depends on the effectiveness of the control or elimination of microorganisms in root canal system (8-10). This goal may be accomplished using mechanical instrumentation and chemical irrigation by various techniques and different irrigants and medicaments.

There is no solid evidence in the literature that demonstrates that mechanical instrumentation alone results in a bacteriafree root canal system, and when the complex anatomy of the root canal system is considered (11,12), this is not surprising.

Further, there is in vitro and clinical evidence that mechanical instrumentation leaves significant portions of the root canal walls untouched (13), so, complete elimination of bacteria by instrumentation alone is unlikely to occur (14).

It is assumed, but not demonstrated, that any pulp tissue left in the root canals can serve as nutrient for any remaining bacteria. Furthermore, tissue remnants also impede the antimicrobial effects of root canal irrigants and medicaments. Therefore, some form of irrigation and disinfection is necessary to remove residual tissue and to kill microorganisms. Chemical treatment of the root canal system can be arbitrarily divided into irrigants, canal rinses, and interappointment medicaments. In this part we will review the various endodontic infections and chemomechanical strategies to manage them.

1.1. Root canal infections versus other infections

Root canal system infections possess some peculiarities that differentiate them from infections in other human sites. Once established, a root canal system infection cannot be eliminated by the host defense mechanisms or by systemic antibiotic therapy. This is explained by the fact that microorganisms present in root canal system infections are in a protected sanctuary, where the absence of a blood supply in a necrotic pulp impedes the transport of defense cells and molecules as well as systemically administrated antibiotics to the infected site (15). On the other hand, although host defense mechanisms and systemic antibiotics are ineffective against microorganisms within the root canal system, if microorganisms gain access to the highly vascularized periradicular tissues, they are usually effectively eliminated and thereby prevented from spreading to other sites. However, if infection spreads into

facial spaces, it may cause life-threatening conditions such as Ludwig's angina and cavernous sinus thrombosis (15).

Due to the anatomical localization of the endodontic infection, it only can be treated through professional endodontic intervention using both chemical and mechanical procedures.

1.2. Microbiology of root canal system

Basically the three most common situations requiring endodontic treatment are as follows (16-19):

- The uninfected pulp (irreversible pulpitis, iatrogenic pulp exposures, trauma, elective pulp therapy for mechanical or restorative dentistry reasons),
- Infected root canal systems with or without apical periodontitis (and without any previous endodontic treatment) where bacterial distribution is variable within the main canal, dentine and lateral canals, and
- Infected canals in previously root-filled teeth with or without apical periodontitis, including cases where the location of bacteria may reflect the quality of the root filling.

The oral cavity contains one of the most concentrated accumulations of microorganisms in the human body with more than 700 different species having been detected so far. Almost all bacteria recovered from the root canal systems of infected teeth belong to the oral microflora (20). However, it is estimated that some 50% of species in the oral microflora cannot be cultured

or have not yet been cultivated. Consequently, concepts of endodontic infections and the associated microflora may need to be re-defined over time as more species become identified through more refined research techniques (21, 22).

Bacteria can be found in both planktonic form and in biofilm communities. The configuration will affect the efficacy of antimicrobial agents with biofilms being much more resistant because of their diffusion barriers and altered bacterial cell metabolism and replication rates (23). It has been shown that some of the root canal walls often remain untouched during chemomechanical preparation of the canals regardless of the instrumentation technique and instruments used (24). Endodontic treatment failure that can be attributed to micro-organisms remaining in the canal may occur if the microorganisms possess pathogenicity, reach sufficient numbers, and gain access to the periradicular tissues to induce or maintain periradicular inflammation (25). Bacteria remaining in the untouched areas within the root canal system are recognized as the main cause of persistent infections after root canal therapy (25, 26) and they can induce inflammatory responses in the periradicular tissues (21).

1.2.1. Endodontic infections

Microbial diversity in endodontic infections has been consistently assessed by anaerobic culturing and culture independent molecular biology methods. Collectively, more than 400 different microbial taxa have been identified in endodontic samples from teeth with different forms of apical periodontitis.

These taxa are usually found in combinations involving many species in primary infections and a few ones in secondary persistent infections (21). Endodontic bacteria fall into nine of the 13 phyla that have oral representatives, namely Firmicutes, Bacteroidetes, Actinobacteria, Fusobacteria, Proteobacteria, Spirochaetes, Synergistes, TM7, and Sulphur River 1 (SR1) (27-32). In addition to bacteria, fungi and archaea have been only occasionally found in intraradicular infections (33, 34, 35) while herpesviruses and HIV have been detected in pulp (only HIV) and apical periodontitis lesions (36-43).

Endodontic infections develop in a previously sterile place which as such does not contain a normal microbiota. Therefore, any species found in the canal has the potential to be an endodontic pathogen or at least to play a role in the ecology of the microbial community. So far, virtually all studies involved with phenotypic or genotypic identification of endodontic bacteria have followed a cross-sectional design, for obvious ethical reasons. Thus, species prevalence and consequently only species association with disease can be inferred from these studies. In addition to frequency of detection, causation may be strengthened on the basis of potential pathogenicity (in animal models or deduced from association with other human diseases). Based on cross sectional studies, several species have emerged as candidate or putative endodontic pathogens, and the labels candidate or putative should be preserved while a definite role in disease causation is not determined.

Primary infections are caused by microorganisms that colonize the necrotic pulp tissue. It can also be regarded as the initial or 'wild' infection, in the sense that there has not been any professional intervention yet. Participating

microorganisms may have been involved in the earlier stages of pulp invasion (usually via caries), which culminated in inflammation and further necrosis, or they can be latecomers that took advantage of the environmental conditions in the root canal after pulp necrosis. Primary infections are the cause of primary apical periodontitis, which can manifest itself as a chronic or acute disease. Some acute conditions may evolve to an abscess, which in some cases can spread to head and neck spaces to establish a life-threatening condition.

1.2.2. Chronic apical periodontitis

Primary infections are conspicuously dominated by anaerobic bacteria organized in a mixed community. Overall, the bacterial density per canal varies from 10^3 to 10^8 (31, 44-47). The largest counts may be mostly related to large lesions and/or symptomatic cases. As for species richness, a mean of 10-20 species/phylotypes have been found per canal in teeth with chronic apical periodontitis as revealed by molecular biology studies (21 29, 32, 48). Root canals of teeth with apical radiolucency associated with a draining sinus tract (chronic apical abscess or suppurative apical periodontitis) have been reported to harbor a mean number of 17 species (32).

The size of the apical periodontitis lesion has been shown to be proportional to the number of bacterial species and cells in the root canal (32, 44, 49). In a molecular study using the reverse-capture checkerboard approach to assess the microbiota of teeth with chronic apical periodontitis (32), demonstrated that the mean number of bacterial taxa per canal was in direct proportion to the