REVIEW ARTICLE

Enterococcus faecalis: A resistant microbe in endodontics

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Abstract

Enterococcus faecalis is commonly detected in endodontic infections that are asymptomatic, persistent and recurrent. They are the microorganisms mainly responsible for persistent periradicular lesions even after root canal treatment. It can survive in the root canal as a single organism or as a major component of the flora. This article highlights the characteristics of E. faecalis, the factors responsible for its virulence and survival, as well as the effect of disinfectants on E. faecalis. Knowledge regarding this microorganism may help to prevent endodontic treatment failures caused by this organism.

Keywords: Antimicrobial resistance, endodontic infections, Enterococcus faecalis

Introduction

One of the primary organisms in patients with post-treatment endodontic infection is Enterococcus faecalis.¹ In obturated root canals exhibiting signs of chronic apical periodontitis, E. faecalis is isolated in 23-70% of the positive cultures²⁻⁷ and often occur in monoculture.⁸ Moreover E. faecalis was among a group of bacteria cultured from periapical lesions refractory to endodontic treatment.⁹

Studies which have investigated the occurrence of E. faecalis in root canal treated teeth with periradicular lesion confirm that the microorganism’s antimicrobial resistance and the ability to adapt to changing environment help it to survive in root canal and cause re-infection. E. faecalis can adhere to root canal walls, accumulate, and form communities organized in biofilm, which helps it resist destruction by enabling the bacteria to become 1000 times more resistant to phagocytosis, antibodies, and antimicrobials than non-bio film-producing organisms.¹⁰ The antimicrobial resistance of bacteria has been attributed to the protective barrier provided by the extracellular polymeric matrix. Surface adherence by bacteria to form biofilms helps in bacterial adaptation and one that is pertinent to endodontic infections.

Virulence Factors of E. faecalis

Upon contamination of the root canal with E. faecalis, it colonizes the dentinal walls under stressful conditions like nutrient deficiency and endodontic medicaments with the help of adhesive moieties.

Aggregation substance, surface carbo-hydrates or fibronectin-binding moieties facilitate adherence of organism to host collagen Type I and extracellular matrix proteins present in the dentin. Extracellular toxins such as cytolysin may induce tissue damage while bacteriocins like AS-48 inhibit the growth of other organisms. Although, lipoteichoic acid can be regarded as a molecule contributing to then virulence of E. faecalis through the facilitation of aggregate formation and plasmid transfer. Superoxide production or pheromones and corresponding peptide inhibitors each may modulate local inflammatory process by stimulating leukocytes to release several mediators such as tumor necrosis factor, interleukins, and prostaglandins and contribute to the periradicular damage.¹¹⁻¹²

The enzyme hyaluronidase is considered to facilitate the spread of bacteria as well as their toxins through host tissues. E. faecalis secretes proteases like gelatinase and serine protease.
Gelatinase contributes to the bone resorption and degradation of dentin organic matrix, thus playing an important role in the pathogenesis of periapical inflammation. Serine protease cleaves peptide bonds and helps the binding of *E. faecalis* to dentin.

**Antimicrobial resistance**

Enterococci have displayed resistance to essentially every useful antimicrobial agent. The resistance may be intrinsic or acquired via gene transfer. The genes for intrinsic resistance, like other species characteristics, reside on the chromosome. Acquired resistance results from either a mutation in the existing DNA or acquisition of the new gene, through the transfer of plasmids and transposons. The intrinsic resistance of enterococci to many commonly used antimicrobial agents may have allowed them a cumulative advantage for further acquisition of genes encoding high-level resistance to aminoglycosides, penicillin, tetracycline, chloramphenicol, and now vancomycin. This allows the organism to survive in an environment in which antimicrobial agents are used. An increased prevalence of bacteria resistant to antibiotics may be found in marginal periodontitis refractory to conventional periodontal therapy. Systemic means cannot reach the focus of infection as it is in the root canal and the dentinal tubules, which are not accessible to the elements of the host defense system. Hence, treatment or preventive procedures should mainly include local methods. Some studies have shown that chlorhexidine is very effective against *E. faecalis*. However the study by Orstavik, Haapaasalo concluded that iodine-potassium iodide appeared more potent irrigant than sodium hypochlorite or chlorhexidine. While 2-5 min exposure of *E. faecalis* to MTAD is effective in killing the organism up to ×200 dilutions. In addition, to disinfectants, physical removal of cells of *E. faecalis* through debridement of the root canal remains essential, since remnants may sustain the inflammation.

**Treatment**

Studies have shown that chlorhexidine is very effective against *E. faecalis*. However, the study by Orstavik, Haapaasalo concluded that iodine-potassium iodide appeared more potent irrigant than sodium hypochlorite or chlorhexidine. While 2-5 min exposure of *E. faecalis* to MTAD is effective in killing the organism up to ×200 dilutions. In addition, to disinfectants, physical removal of cells of *E. faecalis* through debridement of the root canal remains essential, since remnants containing lipoteichoic acids may sustain the inflammation.

**Conclusion**

*E. faecalis* establishes an endodontic infection and maintains a periradicular inflammation due to its virulence factors. Though it has been established that the primary periradicular lesion is a result of a mixed microbial infection rather than *E. faecalis* alone, but in apical periodontitis that persists in spite of endodontic therapy, *E. faecalis* is frequently the dominant pathogen. It hints that this microorganism alone has the capability to maintain root canal infection and periodontal lesion. In order to improve the treatment results, new strategies to prevent or to eliminate the infection by this microbe needs to be developed and that can only be done with a better understanding of the role of the virulence factors of *E. faecalis* in endodontic infections.