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MICROBIAL AND VIRAL FACTORS IN THE ETIOPATHOGENESIS OF APICAL PERIODONTITIS: A SYSTEMATIC REVIEW OF APICAL ROOT CANAL MICROBIOME AND HERPESVIRUS ASSOCIATIONS

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Khasanova Shakhlo
Bachelor's degree senior student ,
Urgench state medical institute ,Uzbekistan.

Abstract

Background: Apical periodontitis is a polymicrobial inflammatory disease primarily associated with infection of the root canal system. Recent advances in molecular microbiology have improved understanding of bacterial communities in the apical root canal segment, while viral agents such as human cytomegalovirus (HCMV) and Epstein–Barr virus (EBV) have been proposed as potential cofactors in periapical lesion pathogenesis.

Objective: This review synthesizes current evidence regarding (1) the microbiome of the apical root canal in primary and posttreatment apical periodontitis and (2) the possible contribution of herpesviruses to periapical disease.

Methods: Electronic searches of PubMed, Embase, Web of Science, ScienceDirect, Proquest, MEDLINE, Scopus, and the Cochrane Library were conducted for studies published up to August 2023. Clinical investigations identifying microbial taxa exclusively in the apical root canal segment and studies evaluating HCMV/EBV in periapical lesions were included. Study quality was assessed using standardized critical appraisal tools.

Results: Twenty-one studies examining the apical root canal microbiome and seventeen studies evaluating herpesvirus involvement met inclusion criteria. Molecular methods predominated. Primary infections exhibited diverse anaerobic taxa including Pseudoramibacter alactolyticus, Olsenella uli, Fusobacterium, Streptococcus, Porphyromonas endodontalis, Prevotella, Actinomyces, Parvimonas micra, and Treponema denticola. Posttreatment infections frequently involved Streptococcus, Enterococcus, Actinomyces, Pseudomonas, and Propionibacterium species. Firmicutes was the dominant phylum. Viral studies detected HCMV and EBV in periapical lesions; however, pooled analyses found no statistically significant association between viral transcripts and clinical features of apical periodontitis.

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Conclusions: Apical periodontitis is associated with complex and diverse bacterial communities that differ between primary and posttreatment infections. Current evidence does not support a definitive etiologic role for HCMV or EBV, although viral–bacterial interactions may influence disease severity. Further standardized and longitudinal studies are needed.

Keywords

apical periodontitis, root canal microbiome, herpesviruses, Epstein–Barr virus, cytomegalovirus, endodontic infection, systematic review

1. Introduction

Apical periodontitis is an inflammatory disorder of periapical tissues caused primarily by microbial infection of the root canal system. The apical portion of the canal represents the critical interface between microbial biofilms and host periapical tissues. Microorganisms colonizing this region are considered directly responsible for the initiation and persistence of periapical inflammation.

Advances in molecular techniques have revealed that endodontic infections are polymicrobial ecosystems with high diversity and interindividual variability. In addition to bacterial pathogens, viral agents—particularly human cytomegalovirus (HCMV) and Epstein–Barr virus (EBV)—have been proposed as potential contributors to lesion progression by modulating host immune responses.

This review integrates current evidence on the apical root canal microbiome and evaluates the possible role of herpesviruses in the etiopathogenesis of apical periodontitis.

2. Materials and Methods

2.1 Search Strategy

A comprehensive electronic search was conducted using PubMed, Embase, Web of Science, ScienceDirect, Proquest, MEDLINE, Scopus, and the Cochrane Library for studies published through August 2023.

2.2 Inclusion Criteria

Studies were included if they:

- Examined microbial taxa exclusively within the apical root canal segment of infected teeth
- Investigated primary and/or posttreatment apical periodontitis
- Used culture-based or molecular microbiological identification methods
- Evaluated HCMV or EBV in human periapical lesions
- Were clinical observational or cross-sectional studies

2.3 Quality Assessment

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Studies were critically appraised using standardized prevalence and methodological assessment tools.

2.4 Data Extraction and Analysis

Data extracted included sampling techniques, microbial identification methods, taxa prevalence, viral detection methods, and clinical correlations. Due to methodological heterogeneity, meta-analysis was not feasible.

3. Results

3.1 Study Selection

From over 2,500 initial records, 38 studies met the inclusion criteria:

- 21 studies on apical root canal microbiome
- 17 studies on herpesvirus detection in periapical lesions

3.2 Apical Root Canal Microbiome

3.2.1 Primary Apical Periodontitis

Primary infections demonstrated high microbial diversity dominated by anaerobic taxa. Frequently detected organisms included:

- Pseudoramibacter alactolyticus
- Olsenella uli
- Fusobacterium spp.
- Streptococcus spp.
- Porphyromonas endodontalis
- Prevotella spp.
- Actinomyces spp.
- Parvimonas micra
- Treponema denticola
- Synergistetes phylum taxa

Bacterial loads ranged from 10^5 to 10^6 cells.

3.2.2 Posttreatment Apical Periodontitis

Posttreatment infections showed reduced bacterial loads (10^3–10^4 cells) but persistence of resistant taxa, including:

- Streptococcus spp.
- Enterococcus spp.
- Actinomyces spp.
- Pseudoramibacter spp.
- Pseudomonas spp.
- Propionibacterium spp.

Firmicutes represented the most prevalent bacterial phylum.

3.3 Sampling and Identification Methods

Cryopulverization of apical root specimens was used in multiple studies to enhance microbial recovery. Molecular techniques (PCR, sequencing) predominated, highlighting greater microbial diversity compared with culture methods.

3.4 Viral Factors in Apical Periodontitis

Seventeen cross-sectional studies evaluated HCMV and EBV presence in periapical lesions. Viral DNA/RNA was detected in both symptomatic and asymptomatic lesions. However, pooled quantitative analyses showed no statistically significant association between viral transcripts and clinical manifestations of apical periodontitis.

4. Discussion

This review confirms that the apical root canal system harbors complex microbial communities directly implicated in periapical inflammation. Primary infections are characterized by diverse anaerobic biofilms, while posttreatment infections often involve persistent, therapy-resistant microorganisms.

The dominance of Firmicutes and detection of species such as Enterococcus faecalis in secondary infections highlight their ability to survive harsh environmental conditions, resist intracanal medicaments, and contribute to treatment failure.

Although herpesviruses have been detected in periapical lesions, current evidence does not establish a causal relationship. Viral presence may reflect opportunistic reactivation in inflamed tissues or modulation of immune responses rather than a primary etiologic role.

4.1 Clinical Implications

- Apical microbial communities should be targeted with enhanced disinfection strategies.
- Persistent species require improved irrigation, intracanal medicaments, and obturation techniques.
- Viral testing is not currently indicated for routine clinical diagnosis.

4.2 Limitations

- Significant heterogeneity in sampling and detection methods
- Predominance of cross-sectional study designs
- Lack of longitudinal and experimental models
- Limited fungal and viral investigations

5. Conclusion

Apical periodontitis is associated with diverse and complex bacterial communities that vary between primary and posttreatment infections. Persistent taxa play a critical role in treatment-resistant disease. Current evidence does not

support a definitive etiologic role for HCMV or EBV, although viral–bacterial interactions warrant further investigation. Standardized methodologies and longitudinal studies are essential to clarify microbial dynamics and improve treatment outcomes.

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