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Assessing Chronic Periodontal Disease Can Influence The Pulp Sensitivity: A Clinical Study

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Abstract

Background: *Chronic periodontitis is a progressive inflammatory condition affecting the supporting structures of teeth. Given the anatomical and vascular interconnections between the periodontium and dental pulp, periodontal inflammation may potentially impact pulp vitality and sensitivity.*

Objective: *This clinical study aimed to assess the influence of chronic periodontal disease on pulp sensitivity using thermal and electric pulp testing in periodontally compromised and healthy teeth.*

Methodology: *A total of 80 systemically healthy individuals aged 25–50 years were enrolled and divided into two groups: Group A (n=40) with chronic periodontitis (clinical attachment loss ≥4 mm and probing pocket depth ≥5 mm) and Group B (n=40) periodontally healthy controls. Thermal (cold) and electric pulp tests were conducted on mandibular first molars. Pulp response was recorded as positive (normal), delayed, or non-responsive. Data were statistically analyzed using Chi-square and independent t-tests.*

Results: *In Group A, 55% of teeth showed delayed pulp response and 25% were non-responsive, while 20% had normal response. In contrast, 90% of teeth in Group B had normal response. Mean threshold for electric pulp test was significantly higher in Group A (6.8 ± 1.2) compared to Group B (3.2 ± 0.9) ($p < 0.001$). Similarly, mean cold response time was prolonged in Group A (7.6 ± 2.4 seconds) compared to Group B (2.8 ± 1.1 seconds) ($p < 0.001$).*

Conclusion: *Chronic periodontal disease significantly alters pulp sensitivity, supporting the concept of pulpal-periodontal interrelationship. Early periodontal intervention may prevent irreversible pulpal changes.*

Keywords: *Chronic periodontitis, pulp vitality, electric pulp test, thermal test, pulp-periodontium relationship*

INTRODUCTION

Periodontal and pulpal tissues are intricately linked through anatomical and vascular pathways including lateral canals, apical foramina, and dentinal tubules **[1]**. Chronic periodontitis, characterized by progressive attachment and bone loss, may not only compromise tooth support but potentially influence the underlying pulpal health due to shared circulatory pathways **[2]**.

Traditionally, the pulp and periodontium have been studied independently. However, evidence suggests that severe periodontal inflammation can cause degenerative changes in the pulp such as fibrosis, calcifications, and loss of cellularity **[3]**. Recent histopathological studies indicate that teeth with deep periodontal pockets exhibit increased pulpal inflammation and compromised neurovascular supply, suggesting a clinical correlation **[4]**. Despite these associations, the diagnostic approach to assessing pulp vitality in periodontally compromised teeth remains controversial. Most clinicians rely on subjective electric or thermal pulp testing, which assesses sensory response but not true vitality. Moreover, delayed or absent response in periodontally involved teeth may be misinterpreted as necrosis, leading to unnecessary endodontic treatment **[5]**. This study aims to bridge the research gap by clinically evaluating whether chronic periodontal disease influences pulp sensitivity using standardized pulp tests. Understanding this relationship has diagnostic and therapeutic significance in integrated periodontal-endodontic care.

Objective: To assess the influence of chronic periodontal disease on pulp sensitivity using electric and thermal pulp testing.

3. MATERIALS AND METHODS

Study Design

This was a single-center, cross-sectional clinical study conducted over six months at the Department of Periodontology.

Sample Size and Selection

Eighty participants aged 25–50 years were recruited after obtaining ethical clearance and informed consent. Participants were divided into two equal groups:

- **Group A (n=40):** Patients with chronic periodontitis (clinical attachment loss ≥4 mm and probing depth ≥5 mm on at least one mandibular first molar).
- **Group B (n=40):** Periodontally healthy controls with no attachment loss or pockets.

Exclusion criteria included systemic diseases, recent periodontal or endodontic treatment, caries, restorations, trauma, or mobility in test teeth.

Equipment and Materials

- Electric Pulp Tester (Parkell Digitest II)
- Endo Ice Spray (1,1,1,2-Tetrafluoroethane)
- Periodontal probe (UNC-15)
- Mouth mirror and tweezers
- Cotton pellets
- Stopwatch

Procedure

Each participant underwent periodontal examination to assess probing pocket depth and clinical attachment level. Mandibular first molars were selected for pulp testing. After isolation with cotton rolls and drying, electric pulp testing was performed by applying the probe to the buccal surface. The patient was instructed to signal upon first sensation. The value at response was recorded.

For cold test, a cotton pellet sprayed with Endo Ice was applied to the middle third of the buccal surface. Time to response was measured in seconds. Pulpal responses were categorized as:

- Normal: Immediate response (<3 sec)
- Delayed: Prolonged response (≥3–10 sec)
- Non-responsive: No response

Statistical Analysis

Data were analyzed using SPSS v26.0. Descriptive statistics were computed for pulp response categories and test values. Independent t-test and Chi-square test were used to compare groups. Significance was set at $p < 0.05$.

4. RESULTS

Descriptive Statistics

Of 80 participants, both groups had a balanced distribution of males (n=22) and females (n=18). The mean age was 39.6 ± 5.1 years for Group A and 38.9 ± 4.7 years for Group B.

Pulp Response to Electric Pulp Testing

Mean electric pulp test threshold was significantly higher in Group A (6.8 ± 1.2) than Group B (3.2 ± 0.9), indicating decreased sensitivity in the periodontitis group ($p < 0.001$) (Table 1).

Pulp Response to Cold Test

Mean cold response time was also significantly delayed in Group A (7.6 ± 2.4 sec) compared to Group B (2.8 ± 1.1 sec) ($p < 0.001$) (Table 1).

Response Categories

In Group A, 55% (n=22) of teeth showed delayed response and 25% (n=10) were non-responsive. Only 20% (n=8) had normal response. In Group B, 90% (n=36) showed normal response, and only 10% (n=4) were delayed (Table 2).

Statistical Significance

Chi-square analysis revealed a significant difference in response distribution between the two groups ($p < 0.001$).

Table 1: Comparison of Pulp Test Values Between Groups

Test Type	Group A (Mean ± SD)	Group B (Mean ± SD)	p-value
Electric Pulp Test	6.8 ± 1.2	3.2 ± 0.9	< 0.001
Cold Test (sec)	7.6 ± 2.4	2.8 ± 1.1	< 0.001

Table 2: Pulp Response Distribution Between Groups

Response Category	Group A (n=40)	Group B (n=40)	p-value
Normal	8 (20%)	36 (90%)	
Delayed	22 (55%)	4 (10%)	
Non-responsive	10 (25%)	0 (0%)	< 0.001

DISCUSSION

The present study demonstrated a statistically significant association between chronic periodontal disease and altered pulp sensitivity. Teeth with chronic periodontitis exhibited elevated electric pulp test thresholds and delayed cold response times, suggesting compromised pulpal sensory function. These findings are consistent with prior histologic observations of pulp degeneration in periodontally affected teeth **[6]**. Kerekes and Olsen reported that periodontally compromised teeth frequently show pulp fibrosis and reduced vascularity, potentially leading to reduced nerve conductivity and responsiveness **[7]**. Similarly, Seltzer and Bender observed histopathologic changes in pulp tissue in association with advanced periodontal lesions, particularly when apical foramina were affected **[8]**.

Although pulp testing methods cannot determine true vitality, they remain valuable diagnostic tools. Reduced response in periodontally compromised teeth may be mistaken for necrosis; however, studies such as that by Mazur and Massler caution against such interpretation, especially in absence of clinical signs of pulpal necrosis **[9-11]**. One limitation of our study is the reliance on subjective pulp testing rather than objective vitality assessment (e.g., laser Doppler flowmetry). Additionally, only mandibular first molars were evaluated, which may limit generalizability. However, the study's strength lies in its standardized methodology and clinically relevant findings that highlight the interrelationship between periodontal and endodontic health.

Future studies with larger samples, histopathologic correlation, and advanced vitality tests are needed to further elucidate the pathophysiological mechanisms involved.

CONCLUSION

Chronic periodontal disease significantly alters pulp sensitivity, as evidenced by higher electric thresholds and delayed cold responses. These findings emphasize the importance of periodontal health in preserving pulp function and avoiding unnecessary endodontic interventions.

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