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Histopathological Investigation of Dental Pulp Reactions Related to apical periodontitis

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Abstract

Background:
Apical periodontitis is a common inflammatory disorder primarily caused by microbial invasion of the dental pulp, leading to subsequent periapical tissue changes. The histopathological assessment of pulpal reactions can provide critical insights into the progression and extent of inflammation associated with apical lesions.

Materials and Methods:
This study was conducted on 30 freshly extracted human teeth indicated for extraction due to advanced carious lesions and signs of apical periodontitis. The samples were fixed in 10% buffered formalin, decalcified in 10% formic acid, embedded in paraffin, and sectioned at 5 μm thickness. Hematoxylin and eosin (H&E) staining was performed to evaluate pulpal changes. Histological parameters assessed included degree of inflammation, pulp necrosis, presence of calcifications, and fibrosis. Descriptive and inferential statistics were applied using SPSS software (version 25.0).

Results:
Out of 30 samples, 23 (76.7%) exhibited chronic inflammatory cell infiltration, 19 (63.3%) showed partial or complete pulp necrosis, 12 (40%) demonstrated dystrophic calcifications, and 9 (30%) revealed fibrotic changes. Severe inflammation was more frequently associated with apical granulomas than cystic lesions ($p < 0.05$). Calcifications were predominantly seen in older patients.

Conclusion:
The dental pulp undergoes significant degenerative and inflammatory changes in response to apical periodontitis, with varying degrees of necrosis, fibrosis, and calcification. Histopathological evaluation aids in understanding the pathophysiological alterations and supports the clinical diagnosis of pulpal-periapical diseases.

Keywords:
Dental pulp, Apical periodontitis, Histopathology, Inflammation, Pulpal necrosis, Periapical lesions

INTRODUCTION

Apical periodontitis is an inflammatory condition primarily resulting from microbial infection of the root canal system, which extends to involve the periapical tissues (1). The dental pulp, a highly vascularized and innervated connective tissue, responds to such infection through a complex cascade of immunological and degenerative changes (2). Bacterial toxins and by-products infiltrating through carious lesions or traumatic exposures trigger pulpal inflammation, ultimately leading to necrosis and periapical pathology if untreated (3).

Histopathological evaluation of the pulp in teeth affected by apical periodontitis offers essential insights into the disease progression and underlying mechanisms. Such analysis helps in identifying characteristic features like infiltration of inflammatory cells, pulpal necrosis, calcifications, and fibrosis (4). These microscopic findings are often correlated with clinical and radiographic indicators, enhancing the diagnostic accuracy and therapeutic decision-making (5).

Moreover, understanding pulpal responses at the histological level contributes significantly to the field of endodontics by delineating the biological processes involved in tissue destruction and repair (6). Previous studies have emphasized the importance of evaluating these histological alterations to distinguish between reversible and irreversible pulpitis, and to determine the extent of periapical damage (7).

This study aims to investigate the histopathological changes in dental pulp tissues associated with apical periodontitis and to establish correlations between pulpal reactions and periapical manifestations.

MATERIALS AND METHODS

This descriptive observational study was conducted in the Department of Oral Pathology. A total of 30 human permanent teeth, scheduled for extraction due to extensive caries and clinical/radiographic evidence of apical periodontitis.

Sample Collection and Preparation

Immediately after extraction, the teeth were rinsed in saline and fixed in 10% neutral buffered formalin for 24 hours. Each tooth was then decalcified using 10% formic acid solution over a period of 7–14 days, depending on the size and mineral content. The decalcification endpoint was confirmed through radiographic assessment and physical testing for softness.

Histological Processing

Once decalcified, the samples were thoroughly washed, dehydrated in ascending grades of alcohol, cleared in xylene, and embedded in paraffin wax. Serial sections of 4–5 µm thickness were obtained using a rotary microtome and mounted on glass slides.

Staining and Microscopic Evaluation

The sections were stained with hematoxylin and eosin (H&E) for routine histopathological examination. All slides were evaluated under a light microscope (×10 and ×40 magnifications) by two experienced oral pathologists blinded to the clinical data.

Parameters Assessed

The following histological features of the pulp were recorded:

- Type and extent of inflammatory cell infiltrate (acute or chronic)
- Degree of pulpal necrosis (partial or complete)
- Presence of fibrosis or collagen deposition
- Identification of pulp calcifications (dystrophic or diffuse)

Data Analysis

Descriptive statistics were calculated for each variable. Frequencies and percentages were used to represent qualitative data. Associations between histological features and patient-related variables (e.g., age, lesion type) were assessed using the chi-square test. All analyses were performed using SPSS software version 25.0, with a p-value < 0.05 considered statistically significant.

RESULTS

A total of 30 extracted permanent teeth affected by apical periodontitis were histologically analyzed for pulpal changes. The mean age of patients was 38.6 ± 12.3 years, with a male-to-female ratio of 1.3:1. The most commonly involved teeth were mandibular molars (43.3%), followed by maxillary premolars (26.7%).

Histopathological Findings

Out of the total samples, 24 teeth (80%) showed varying degrees of chronic inflammatory infiltration, while 6 teeth (20%) displayed acute inflammation. Complete pulpal necrosis was observed in 19 cases (63.3%), whereas 7 cases (23.3%) showed partial necrosis, and 4 cases (13.3%) had vital pulp with mild inflammation.

Fibrotic changes, characterized by the presence of dense collagen bundles and reduced cellularity, were seen in 9 teeth (30%). Dystrophic calcifications were identified in 12 samples (40%), predominantly in patients above 40 years of age.

A comparison between inflammation type and presence of calcification showed a statistically significant association ($p = 0.041$), indicating a tendency toward calcific degeneration in chronic cases.

Table 1. Frequency of Histopathological Features Observed in Dental Pulp (n = 30)

Histological Feature	Number of Teeth (n)	Percentage (%)
Chronic Inflammatory Infiltrate	24	80.0
Acute Inflammatory Infiltrate	6	20.0
Complete Pulp Necrosis	19	63.3
Partial Pulp Necrosis	7	23.3
Vital Pulp with Mild Inflammation	4	13.3
Fibrosis	9	30.0
Dystrophic Calcification	12	40.0

These findings confirm that chronic inflammation and necrosis are the predominant pulpal responses in apical periodontitis. Calcific changes and fibrosis were more prevalent in older individuals and in teeth with long-standing periapical lesions.

DISCUSSION

This study aimed to explore the histopathological alterations within the dental pulp in teeth affected by apical periodontitis. The findings revealed that chronic inflammation and pulpal necrosis were the most common features, followed by dystrophic calcification and fibrosis. These results are consistent with earlier reports highlighting the irreversible damage to pulp tissue in the presence of persistent microbial infection and periapical pathology (1,2).

Chronic inflammatory infiltrates observed in 80% of samples align with the understanding that bacterial biofilms within the root canal system trigger a sustained host immune response (3). The predominance of chronic over acute inflammation reflects the typically asymptomatic progression of apical periodontitis until advanced stages (4). Similar patterns of chronic infiltration have been reported by Ricucci and Siqueira, who noted periapical granulomas associated with pulpal necrosis in a majority of cases (5).

Pulpal necrosis, identified in 63.3% of teeth in our study, confirms that microbial by-products penetrating through carious lesions or defective restorations can induce irreversible changes in pulp vitality (6,7). Bergenholtz described necrosis as a critical event leading to the breakdown of pulp architecture and subsequent periapical lesions (8). The presence of partial necrosis in some teeth suggests a transitional stage, indicating that necrotic changes may initiate in localized regions before progressing to complete tissue breakdown (9).

The observation of pulp calcifications in 40% of cases is significant. These calcifications may represent a reparative or degenerative response to chronic irritation or trauma (10). Age-related changes also play a role in calcification, as shown in our data where calcifications were more frequent in individuals above 40 years, in agreement with studies by Bernick and Nedelman (11). Dystrophic calcification has been associated with longstanding low-grade inflammation and poor vascular supply, as seen in advanced endodontic lesions (12).

Fibrotic changes, noted in 30% of samples, further support the hypothesis of chronic insult to the pulp. Persistent inflammation leads to fibroblast proliferation and deposition of extracellular matrix components, resulting in fibrosis (13). The clinical implication of such fibrotic tissue is its reduced healing potential and resistance to revascularization during regenerative procedures (14).

This study also found a significant association between chronic inflammation and calcification, emphasizing the interrelationship between degenerative and inflammatory processes within the pulp. Similar correlations have been described in previous histological investigations of cariously exposed and infected pulps (15,16).

The limitations of this study include its relatively small sample size and the absence of microbiological correlation, which could have enhanced the interpretation of pulpal reactions. Future studies incorporating immunohistochemical markers and microbial profiling could provide a more detailed understanding of disease progression.

CONCLUSION

In conclusion, apical periodontitis significantly impacts pulpal histology, with chronic inflammation, necrosis, calcification, and fibrosis being common outcomes. Recognizing these changes is essential for accurate diagnosis, prognosis, and treatment planning in endodontic practice.

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