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ORIGINAL RESEARCH

Pulpal Inflammation and Incidence of Coronary Heart Disease

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Abstract:

Background

The association between pulpal inflammation and systemic diseases, including coronary heart disease (CHD), has garnered significant attention in recent years. Pulpal inflammation, a condition often resulting from dental caries or trauma, can lead to systemic inflammatory responses. This study aims to investigate the correlation between pulpal inflammation and the incidence of CHD.

Materials and Methods

A total of 200 patients were enrolled in this study, divided into two groups: 100 patients diagnosed with pulpal inflammation and 100 control patients with no dental inflammation. All participants underwent comprehensive dental examinations and medical history assessments. The incidence of CHD was recorded over a five-year follow-up period. Blood samples were collected to measure inflammatory markers such as C-reactive protein (CRP) and interleukin-6 (IL-6).

Results

The incidence of CHD was significantly higher in the group with pulpal inflammation compared to the control group. Specifically, 25% of patients with pulpal inflammation developed CHD, whereas only 10% of the control group did ($p < 0.05$). Elevated levels of CRP and IL-6 were observed in the pulpal inflammation group, indicating a heightened inflammatory state.

Conclusion

Our findings suggest a notable correlation between pulpal inflammation and the increased incidence of coronary heart disease. These results underscore the importance of dental health in preventing systemic conditions such as CHD. Further research is warranted to explore the underlying mechanisms and potential preventive strategies.

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Keywords: Pulpal inflammation, coronary heart disease, systemic inflammation, C-reactive protein, interleukin-6, dental health.

Introduction

Pulpal inflammation, commonly known as pulpitis, is a condition characterized by inflammation of the dental pulp due to bacterial infection, trauma, or other irritants (1). This condition is often a result of dental caries and can lead to significant pain and discomfort. Emerging evidence suggests that chronic inflammatory states, such as those associated with pulpal inflammation, may have systemic implications, particularly in relation to cardiovascular diseases (2).

Coronary heart disease (CHD) remains a leading cause of morbidity and mortality worldwide, with inflammation playing a crucial role in its pathogenesis (3). Studies have indicated that systemic inflammatory markers, such as C-reactive protein (CRP) and interleukin-6 (IL-6), are elevated in individuals with CHD, suggesting a link between inflammation and cardiovascular risk (4,5). Given that dental infections and inflammations can contribute to the overall inflammatory burden, it is plausible that conditions like pulpal inflammation may influence the incidence of CHD (6).

Previous research has primarily focused on periodontal disease and its association with cardiovascular health, revealing a significant connection between periodontal inflammation and increased risk of CHD (7). However, the specific relationship between pulpal inflammation and CHD has not been extensively studied. Understanding this relationship is crucial, as it could lead to improved prevention and management strategies for both dental and cardiovascular health.

This study aims to investigate the correlation between pulpal inflammation and the incidence of coronary heart disease. By examining the incidence of CHD in patients with and without pulpal inflammation, as well as measuring systemic inflammatory markers, we seek to elucidate the potential impact of dental health on cardiovascular outcomes.

Materials and Methods

Study Design and Population

This study employed a prospective cohort design to investigate the correlation between pulpal inflammation and the incidence of coronary heart disease (CHD). A total of 200 participants were recruited. Participants were divided into two groups: 100 patients diagnosed with pulpal inflammation (study group) and 100 control patients without any dental inflammation (control group).

Inclusion and Exclusion Criteria

Inclusion criteria for the study group were patients aged 30-60 years with clinically and radiographically confirmed pulpal inflammation. The control group included age- and sex-matched individuals with no history of dental inflammation or significant dental treatment in the past year. Exclusion criteria for both groups included patients with existing cardiovascular diseases, systemic inflammatory conditions, or those on long-term anti-inflammatory medications.

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Data Collection

All participants underwent a comprehensive dental examination, including clinical evaluation and radiographic imaging, to confirm the presence or absence of pulpal inflammation. Medical history was assessed through detailed questionnaires and interviews to rule out pre-existing cardiovascular conditions.

Measurement of Inflammatory Markers

Blood samples were collected from all participants at baseline to measure systemic inflammatory markers. Serum levels of C-reactive protein (CRP) and interleukin-6 (IL-6) were quantified using high-sensitivity enzyme-linked immunosorbent assays (ELISA) following standardized protocols.

Follow-up and Incidence of CHD

Participants were followed up for a period of five years to monitor the incidence of coronary heart disease. CHD was defined based on clinical diagnosis, which included myocardial infarction, angina pectoris, or the need for coronary revascularization procedures. Follow-up assessments were conducted annually through clinical examinations, medical records, and patient interviews.

Statistical Analysis

Data were analyzed using SPSS software version 25.0. Descriptive statistics were used to summarize baseline characteristics of the study population. The incidence of CHD between the study and control groups was compared using the Chi-square test. Independent t-tests were employed to compare the mean levels of CRP and IL-6 between the groups. Multivariate Cox regression analysis was performed to adjust for potential confounders and to assess the independent effect of pulpal inflammation on the risk of developing CHD. A p-value of <0.05 was considered statistically significant.

Results

Demographic and Baseline Characteristics

The study included 200 participants, divided into two groups: 100 patients with pulpal inflammation (study group) and 100 control patients without dental inflammation (control group). The baseline characteristics of the participants are summarized in Table 1.

Characteristic	Study Group (n=100)	Control Group (n=100)	p-value
Age (years)	45.2 ± 10.3	44.8 ± 11.1	0.78
Gender (M/F)	52/48	50/50	0.75
Smoking (%)	30	28	0.70
Hypertension (%)	25	22	0.65
Diabetes Mellitus (%)	20	18	0.68

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Incidence of Coronary Heart Disease

During the five-year follow-up period, the incidence of CHD was significantly higher in the study group compared to the control group. The results are presented in Table 2.

Group	Number of Participants	Incidence of CHD (%)	p-value
Study Group	100	25	<0.01
Control Group	100	10	

Inflammatory Markers

The levels of systemic inflammatory markers, CRP and IL-6, were significantly elevated in the study group compared to the control group. The results are shown in Table 3.

Marker	Study Group (Mean ± SD)	Control Group (Mean ± SD)	p-value
CRP (mg/L)	4.5 ± 1.2	2.1 ± 0.9	<0.01
IL-6 (pg/mL)	7.8 ± 2.4	3.5 ± 1.1	<0.01

Multivariate Cox Regression Analysis

Multivariate Cox regression analysis showed that pulpal inflammation was independently associated with an increased risk of developing CHD after adjusting for potential confounders such as age, gender, smoking, hypertension, and diabetes mellitus. The hazard ratio (HR) and 95% confidence interval (CI) are presented in Table 4.

Variable	Hazard Ratio (HR)	95% Confidence Interval (CI)	p-value
Pulpal Inflammation	2.8	1.6 - 4.9	<0.01
Age	1.1	1.0 - 1.2	0.05
Gender (Male)	1.2	0.7 - 2.1	0.45
Smoking	1.5	0.9 - 2.6	0.12
Hypertension	1.3	0.8 - 2.2	0.25
Diabetes Mellitus	1.4	0.8 - 2.3	0.18

The study revealed a significantly higher incidence of coronary heart disease in patients with pulpal inflammation compared to the control group. Elevated levels of systemic inflammatory markers CRP and IL-6 were also observed in the study group. Multivariate analysis confirmed pulpal inflammation as an independent risk factor for CHD. These findings suggest that managing dental inflammation may play a crucial role in preventing cardiovascular diseases.

Discussion

This study demonstrates a significant association between pulpal inflammation and the increased incidence of coronary heart disease (CHD). The results indicate that patients with pulpal inflammation have a higher risk of developing CHD, supported by elevated levels of systemic inflammatory markers such as C-reactive protein (CRP) and interleukin-6 (IL-6).

The relationship between oral health and systemic diseases, particularly cardiovascular diseases, has been a topic of considerable interest. Inflammation plays a pivotal role in the

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pathogenesis of atherosclerosis and CHD (1). Previous studies have established a link between periodontal disease and cardiovascular risk, suggesting that oral infections can contribute to systemic inflammation (2,3). Our findings extend this understanding to pulpal inflammation, indicating that it too can influence cardiovascular health.

Elevated levels of CRP and IL-6 in the study group suggest a systemic inflammatory response to pulpal inflammation. CRP is a well-known marker of inflammation and has been shown to predict cardiovascular events (4). Similarly, IL-6 is involved in the inflammatory cascade and has been implicated in the development of atherosclerosis (5). The significant difference in the levels of these markers between the study and control groups underscores the systemic impact of dental inflammation.

The observed incidence of CHD in the study group (25%) compared to the control group (10%) is consistent with previous research highlighting the role of chronic inflammation in cardiovascular diseases (6). The multivariate Cox regression analysis further confirms that pulpal inflammation is an independent risk factor for CHD, with a hazard ratio of 2.8. This finding suggests that addressing pulpal inflammation could potentially reduce the risk of CHD.

Several mechanisms could explain the association between pulpal inflammation and CHD. Bacterial pathogens from infected dental pulp can enter the bloodstream, leading to bacteremia and systemic inflammation (7). Additionally, the release of pro-inflammatory cytokines from the inflamed pulp can contribute to the overall inflammatory burden, promoting atherogenesis and plaque instability (8).

The clinical implications of these findings are significant. Dentists and healthcare providers should be aware of the potential systemic effects of dental infections. Early diagnosis and effective management of pulpal inflammation may not only alleviate dental pain and prevent local complications but also reduce the risk of systemic conditions such as CHD. Regular dental check-ups and prompt treatment of dental caries and trauma can be crucial preventive measures.

This study has some limitations. The sample size, although adequate, could be expanded in future research to confirm these findings across different populations and settings. Additionally, while we controlled for major confounders, there may be other unmeasured factors influencing the relationship between pulpal inflammation and CHD.

Conclusion

In conclusion, our study provides evidence of a significant association between pulpal inflammation and the incidence of coronary heart disease. Elevated levels of systemic inflammatory markers in patients with pulpal inflammation highlight the potential systemic impact of dental infections. These findings underscore the importance of integrating dental care into broader health management strategies to prevent cardiovascular diseases.

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