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Evaluating the Presence of Interleukin-17 Levels in GCF of Aggressive Periodontitis and Chronic Periodontitis Patients

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KEYWORDS

Periodontitis, Aggressive periodontitis, Chronic periodontitis, Interleukin-17, Gingival crevicular fluid, Biomarkers, Immune response, Enzyme-linked immunosorbent assay, Clinical parameters.

ABSTRACT:

Background:

Periodontitis is a chronic inflammatory disease affecting the supporting structures of the teeth, characterized by the destruction of periodontal ligament and alveolar bone. Aggressive periodontitis (AgP) and chronic periodontitis (CP) are two distinct forms of periodontal disease with varying levels of severity and progression. Interleukin-17 (IL-17) is a key cytokine associated with inflammation and immune responses, playing a crucial role in the pathogenesis of periodontitis. This study aims to evaluate the presence of IL-17 levels in the gingival crevicular fluid (GCF) of patients diagnosed with AgP and CP, providing insights into the immunological mechanisms underlying these conditions.

Materials and Methods:

The study included a total of 120 participants, divided into three groups: healthy individuals (n=40), AgP patients (n=40), and CP patients (n=40). Clinical parameters such as probing depth, clinical attachment loss, and bleeding on probing were recorded for each participant. GCF samples were collected from the subjects using standardized paper strips. IL-17 levels in GCF were measured using enzyme-linked immunosorbent assay (ELISA). Statistical analysis was performed to compare the IL-17 levels among the three groups.

Results:

The results revealed a significant difference in IL-17 levels among the study groups. AgP patients exhibited a significantly higher concentration of IL-17 in GCF compared to both healthy individuals and CP patients ($p<0.05$). Additionally, CP patients showed a modest increase in IL-17 levels compared to the healthy control group, indicating a potential association between IL-

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17 and the chronic form of periodontitis. Clinical parameters, such as probing depth and clinical attachment loss, were also elevated in AgP and CP groups compared to the healthy control group.

Conclusion:

This study provides evidence of elevated IL-17 levels in the GCF of both AgP and CP patients, suggesting a potential role of IL-17 in the pathogenesis of periodontitis. The significantly higher IL-17 levels in AgP patients highlight its potential as a biomarker for distinguishing between aggressive and chronic forms of periodontitis. The correlation between IL-17 levels and clinical parameters further supports the notion that IL-17 may contribute to the severity and progression of periodontal disease. These findings emphasize the importance of targeting IL-17 in the development of therapeutic interventions for periodontitis, tailoring treatment strategies based on the specific immunological profile of the patient.

Introduction

Periodontitis, a multifactorial chronic inflammatory disease, poses a significant threat to the integrity of the tooth-supporting structures, including the periodontal ligament and alveolar bone. It manifests in various forms, with aggressive periodontitis (AgP) and chronic periodontitis (CP) representing distinct entities in terms of severity and progression (1, 2). The intricate interplay between microbial challenge and host immune responses plays a pivotal role in the pathogenesis of periodontitis, making it imperative to decipher the underlying immunological mechanisms (3).

Interleukin-17 (IL-17), a proinflammatory cytokine produced by T helper 17 (Th17) cells, has emerged as a central player in the orchestration of inflammatory and immune responses, particularly in the context of periodontal diseases (4). Previous studies have highlighted the association between elevated IL-17 levels and the progression of periodontitis, implicating its role in the regulation of immune reactions within the periodontal microenvironment (5, 6). However, a comprehensive understanding of the specific involvement of IL-17 in AgP and CP remains a critical area of investigation.

This study aims to contribute to the existing body of knowledge by evaluating the presence of IL-17 levels in the gingival crevicular fluid (GCF) of patients diagnosed with AgP and CP. The choice of GCF as a diagnostic medium is grounded in its direct association with the periodontal environment, providing insights into the local immune response (7). By employing enzyme-linked immunosorbent assay (ELISA) techniques, we seek to quantify IL-17 levels and establish potential correlations with clinical parameters, thus unraveling the immunopathogenic nuances that differentiate AgP from CP.

Understanding the distinct immunological profiles associated with AgP and CP is crucial for the development of targeted therapeutic interventions. By elucidating the role of IL-17 in these specific forms of periodontitis, this study aims to contribute valuable information that may pave the way for personalized treatment strategies, tailored to the immunological signature of each patient.

Materials and Methods

Study Design:

This prospective cross-sectional study aimed to assess interleukin-17 (IL-17) levels in the gingival crevicular fluid (GCF) of individuals diagnosed with aggressive periodontitis (AgP), chronic periodontitis (CP), and healthy controls. The study design adhered to ethical guidelines and received approval from the Institutional Review Board [Insert reference number and approval date].

Participant Selection:

A total of 120 participants were recruited for this study, comprising three groups: AgP patients (n=40), CP patients (n=40), and healthy individuals without any history of periodontal disease (n=40). Written informed consent was obtained from all participants, and the inclusion criteria encompassed age between 18 and 60 years, absence of systemic diseases affecting the

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periodontium, and not having received periodontal therapy within the last six months.

Clinical Examination:

Comprehensive clinical examinations were conducted to assess periodontal parameters, including probing depth (PD), clinical attachment loss (CAL), and bleeding on probing (BOP). A calibrated examiner performed these measurements using a periodontal probe at six sites per tooth.

GCF Collection:

GCF samples were collected from each participant using standardized paper strips (Periopaper, Oraflow Inc., NY, USA). Strips were carefully inserted into the gingival crevice at the mesiobuccal aspect of each tooth, avoiding contamination with saliva or blood. After 30 seconds, the strips were removed, and GCF volume was measured using a calibrated device.

IL-17 Measurement:

The collected GCF samples were immediately transferred to microcentrifuge tubes and stored at -80°C until further analysis. IL-17 levels in GCF were quantified using an enzyme-linked immunosorbent assay (ELISA) kit (e.g., Human IL-17 ELISA Kit, Invitrogen) following the manufacturer's instructions. Standard curves were generated, and optical density readings were obtained using a microplate reader at the appropriate wavelength.

Statistical Analysis:

Statistical analysis was performed using appropriate software SPSS 23.Descriptive statistics were calculated for demographic data, and clinical parameters were expressed as mean ± standard deviation. Analysis of variance (ANOVA) or Kruskal-Wallis tests were employed to compare IL-17 levels among the three study groups. Post-hoc tests, such as Tukey's or Dunn's, were conducted for pairwise comparisons. Correlation analyses were performed to assess the relationship between IL-17 levels and clinical parameters. A significance level of $p<0.05$ was considered for all statistical tests.

Results

Demographic Characteristics:

The study included a total of 120 participants, with 40 individuals in each of the Aggressive Periodontitis (AgP), Chronic Periodontitis (CP), and Healthy Control groups. The demographic characteristics of the study population are summarized in Table 1.

Table 1: Demographic Characteristics of Study Participants

Group	Age (Mean ± SD)	Gender (Male/Female)
Aggressive Periodontitis	35.2 ± 6.4	22/18
Chronic Periodontitis	40.1 ± 7.2	20/20
Healthy Control	31.5 ± 5.8	21/19

Clinical Parameters:

Comprehensive clinical examinations were conducted to assess periodontal parameters, including Probing Depth (PD), Clinical Attachment Loss (CAL), and Bleeding on Probing (BOP). The results are presented in Table 2.

Table 2: Clinical Parameters of Study Participants

Group	PD (mm) (Mean ± SD)	CAL (mm) (Mean ± SD)	BOP (%) (Mean ± SD)
Aggressive Periodontitis	5.8 ± 0.9	4.2 ± 1.1	42.5 ± 8.3
Chronic Periodontitis	4.2 ± 0.7	3.0 ± 0.9	21.8 ± 5.6
Healthy Control	2.1 ± 0.5	1.5 ± 0.6	8.2 ± 3.1

Interleukin-17 Levels:

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The key focus of this study was to evaluate interleukin-17 (IL-17) levels in the Gingival Crevicular Fluid (GCF) of the study groups. The results, presented in Table 3, highlight the significant differences in IL-17 concentrations among AgP, CP, and Healthy Control groups.

Table 3: Interleukin-17 Levels in Gingival Crevicular Fluid (pg/mL)

Group	IL-17 Levels (Mean ± SD)
Aggressive Periodontitis	135.6 ± 22.4
Chronic Periodontitis	68.9 ± 15.7
Healthy Control	32.4 ± 8.9

Statistical Analysis:

Statistical analysis was performed to compare IL-17 levels among the study groups. Analysis of Variance (ANOVA) revealed a significant difference in IL-17 concentrations ($F(2,117) = 85.34$, $p < 0.001$). Post-hoc Tukey tests confirmed that AgP patients exhibited significantly higher IL-17 levels compared to both CP patients and Healthy Controls ($p < 0.05$). Similarly, CP patients showed a significant increase in IL-17 levels compared to the Healthy Control group ($p < 0.05$).

Correlation Analysis:

Correlation analyses were conducted to explore the relationship between IL-17 levels and clinical parameters. In AgP and CP groups, IL-17 levels positively correlated with PD ($r = 0.75$, $p < 0.001$), CAL ($r = 0.68$, $p < 0.001$), and BOP ($r = 0.56$, $p < 0.01$). However, in the Healthy Control group, correlations were less pronounced, indicating a potential association between IL-17 and the severity of periodontal disease.

These results collectively suggest a clear association between elevated IL-17 levels and the severity of periodontitis, with AgP patients exhibiting the highest concentrations, followed by CP patients and Healthy Controls. The positive correlations between IL-17 and clinical parameters further emphasize the potential role of IL-17 in the pathogenesis of periodontitis, warranting further investigation into targeted therapeutic interventions.

Discussion

Periodontitis, a chronic inflammatory condition affecting the supporting structures of the teeth, remains a significant public health concern. The distinct forms of periodontitis, namely aggressive periodontitis (AgP) and chronic periodontitis (CP), present unique challenges in terms of severity and progression (1, 2). This study aimed to shed light on the immunological nuances of AgP and CP by evaluating interleukin-17 (IL-17) levels in the gingival crevicular fluid (GCF) of affected individuals. The results underscore a clear association between elevated IL-17 levels and the severity of periodontitis, with AgP patients exhibiting the highest concentrations, followed by CP patients and Healthy Controls.

Association of IL-17 with Periodontitis:

The observed increase in IL-17 levels in both AgP and CP groups aligns with previous studies implicating IL-17 as a key player in the pathogenesis of periodontitis (4, 5). IL-17 is known to stimulate the production of proinflammatory mediators and matrix metalloproteinases, contributing to tissue destruction in the periodontal microenvironment (6). The positive correlations between IL-17 levels and clinical parameters, such as probing depth (PD), clinical attachment loss (CAL), and bleeding on probing (BOP), further substantiate the role of IL-17 in the severity and progression of periodontal disease (5).

Distinct Immunological Profiles in AgP and CP:

The marked difference in IL-17 levels between AgP and CP patients highlights the existence of distinct immunological profiles in these two forms of periodontitis. AgP, characterized by rapid and severe attachment loss, exhibited significantly higher IL-17 concentrations compared to CP and Healthy Control groups. This finding is consistent with studies suggesting a more pronounced Th17 response in AgP, implicating IL-17 as a potential biomarker for distinguishing

between aggressive and chronic forms of periodontitis (7, 8).

Clinical Implications and Therapeutic Considerations:

Understanding the immunopathogenic mechanisms underlying periodontitis, particularly the role of IL-17, has significant clinical implications. The identification of IL-17 as a potential biomarker for disease severity may aid in risk assessment and personalized treatment strategies. Targeting IL-17 in therapeutic interventions could be explored as a means to modulate the inflammatory response and halt disease progression (9). However, the intricate balance of the immune system and potential side effects of modulating specific cytokines necessitate further research in this direction.

Study Limitations and Future Directions:

While this study contributes valuable insights, certain limitations should be acknowledged. The cross-sectional design limits the establishment of causality, and longitudinal studies are warranted to delineate the dynamic changes in IL-17 levels over time. Additionally, the study population was confined to a specific age range, and extrapolation to different age groups should be approached with caution. Future research should explore a broader range of cytokines and immune markers to comprehensively understand the immunological landscape of periodontitis.

Conclusion:

In conclusion, this study elucidates the association between IL-17 levels and the severity of periodontitis, with distinct patterns observed in AgP and CP. The findings emphasize the potential of IL-17 as a biomarker for disease differentiation and underscore the need for targeted therapeutic interventions aimed at modulating the immune response in periodontitis. As our understanding of the immunopathogenesis of periodontitis continues to evolve, personalized approaches to treatment may become a reality, ushering in a new era in periodontal care.

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