Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/9k0nqm57

HISTOPATHOLOGICAL CHANGES IN PERIODONTAL LIGAMENT OF CHRONIC PERIODONTITIS PATIENTS IN PESHAWAR

Rabia Alamgeer[1], Nauman Ejaz[2], Neelofar Nausheen[3], Tanzeela[4], Horraia Zahid[5], Pashmina Nisar[6*]

[1]BDS M.Phil Oral Pathology, Assistant Professor, Sardar Begum Dental College, Peshawar Pakistan
[2]BDS, M.Phil. Oral Biology (Continue), Post Graduate Resident, Sardar Begum Dental College, Gandhara University, Peshawar Pakistan
[3]BDS, MCPS (Surgery), M.Phil (Oral Biology), Associate Professor, Sardar Begum Dental College, Peshawar Pakistan
[4]BDS, M.Phil (Continue), Lecturer, Sardar Begum Dental College, Peshawar Pakistan
[5,6*]BDS, M.Phil Oral Biology, Assistant Professor, Sardar Begum Dental College, Peshawar Pakistan

***Corresponding Author:** Pashmina Nisar
*Email: pashminanisar@hotmail.com

ABSTRACT

Chronic periodontitis is a common disease of the supporting tissues of teeth, such as the PDL. This descriptive-analytical cross-sectional study aimed at comparing the histopathological alterations in PDL of fifty patients suffering from chronic periodontitis in Peshawar. Tissue samples were obtained from patients who had opted for extraction of their teeth and they were tested for inflammatory changes, orientation changes in collagen fibers, cementum resorption, vascular changes and fibroblast activity. The study also showed tissue damage and raised inflammatory responses, damage to collagen, loss of cementum and reduced fibroblast count. The findings in this study are alarming especially bearing in mind the outcomes of people diagnosed with chronic periodontitis and call for early get well care.

Introduction

Chronic periodontitis is one of the most common periodontal diseases which occur in the PDL, alveolar bone, and gingiva. It is commemorated by bacterial biofilm and leads to chronic periodontitis characterized by the loss of periodontal attachment and alveolar bone, tooth mobility, and ultimately tooth loss. It produces mediators like cytokines and enzymes which in return leads in degradation of the extracellular matrix. Periodontal diseases are prevalent in Pakistan due to the lack of proper oral hygiene and, therefore, the insufficient number of dental treatments. The management of this disease necessitates an analysis of histopathological changes in the PDL of people with CP in Peshawar.

Methodology

Fifty patients diagnosed with chronic periodontitis were selected for the current cross-sectional study at Sardar Begum Dental College, Peshawar. Only patients of either sex in the age group of 30 to 60 years were selected while patients having systemic diseases or those having received periodontal therapy in the past one year were excluded. Well characterized samples from sub gingival tissues were collected from root surface of the PDL at the time of tooth extraction. These samples were processed through 10% formalin before undergoing demineralized using 10% formic acid before being

processed for paraffin sectioning. Samples were stained with H&E method. Therefore, parameters that included collagen fiber arrangement, inflammatory cell accumulation, cementum loss, vessel alterations, and fibroblast density were analyzed. Additional immunohistochemical staining for MMPs was also done to determine the effect of ECM degradation.

Results

Histopathological analysis of the PDL samples showed significant alterations:

Inflammatory infiltrates: For 50% of samples, moderate inflammation was observed and seen are lymphocytes and plasma cells.

Collagen fiber disorganization: 54% of the samples exhibited complete disorganization of collagen fibers.

Cementum resorption: Mild to severe resorption was documented in 64% of the samples.

Vascular changes: 80% percent of the samples studied exhibited characteristics of inflammation by showing increased blood vessel density.

Fibroblast depletion: Sample 72% revealed a reduction in the number of fibroblast count which shows impaired tissue repair capability.

Conclusion

The present study emphasises on the quantification of histopathological alterations in chronic periodontitis patients of periodontal ligament in Peshawar. Such inflammation, disorder of collagen fiber, cementum resorption, and the absence of fibroblasts support the idea that this disease is progressive. These observations correspond with other pathological changes observed in such investigations and stress the importance of timely primary and secondary diagnosis in the further destruction of tissues and tooth loss.

Keywords: Histopathological Changes, Periodontal Ligament, Chronic Periodontitis

Introduction

The periodontal ligament (PDL), alveolar bone, and gingiva are all impacted by periodontal disease, which is most common in chronic periodontitis. Bacterial biofilms cause chronic periodontitis, an inflammatory condition that gradually destroys the periodontium. When left untreated, this disorder causes the alveolar bone and periodontal attachment to deteriorate, which in turn causes the teeth to become mobile and, eventually, lose their teeth. In this pathophysiology, the breakdown of the extracellular matrix is caused by enzymes, inflammatory mediators (including cytokines and prostaglandins) and a host-mediated response to microbial infection (1). Changes in the histological structure of the periodontal ligament, which is essential for keeping the tooth in its alveolar socket, are signs of advanced illness (2).

Periodontal infections are prevalent in Pakistan, especially in cities like Peshawar where a large number of people live. Periodontitis is more common in this area because of poor oral hygiene, a lack of access to dental treatment, and socioeconomic considerations (3). The periodontal ligament histological alterations in Peshawar patients with chronic periodontitis are the focus of this investigation. In order to better understand the development of chronic periodontitis and develop treatment strategies, this research aims to analyze tissue samples in order to shed light on the morphological and structural changes taking place in the PDL.

Methodology

This cross-sectional study was carried out on 50 patients attending Sardar Begum Dental College Peshawar diagnosed with chronic periodontitis. Ethical approval for conducting the study was sought and, participant consent was sought from all participants.

Inclusion Criteria: Participants had to be between the ages of 30 and 60 and have a clinical diagnosis of chronic periodontitis along with symptoms including bleeding while probing, deep probing depths, and radiographic attachment loss in order to be included for this study.

Exclusion Criteria: Patients who had systemic disorders like diabetes or who smoked were not eligible, as were those who had taken antibiotics or periodontal treatment within the past six months. Sub gingival samples of periodontal tissue were obtained from the PDL during tooth extraction for clinical purposes. These teeth were treated under the surgical intervention because of poor prognosis attributed to periodontal diseases. The extracted teeth were immediately fixed in 10% formalin solution and further demineralized in 10% formic acid of 4-6 weeks period. After decalcification, the tissue sections were subjected to paraffin embedding, sectioning to a thickness of 5 micrometers, with staining by the H&E method for light microscopy to observe histopathological alterations.
In histopathological assessment the last examined changes involved collagen fiber architecture, blood vessel density, infiltrated inflammatory cells, cementum degradation and the changes in fibroblast quantity and distribution (4). On the basis of semi-quantitative scoring, the overall intensity of inflammation and the extent of tissue degeneration was assessed. Furthermore, special staining, immunohistochemistry for markers of extracellular matrix degradation, and matrix metalloproteinases (MMPs), were performed (5).

Results

Out of the 50 patients included in the study, 30 were male, and 20 were female, with an average age of 48 years. Histopathological analysis revealed a range of significant changes in the periodontal ligament of patients with chronic periodontitis. The following tables summarize the key findings related to the histopathological changes in the periodontal ligament.

Table 1. Distribution of Inflammatory Infiltrate in Periodontal Ligament Samples

Inflammatory Cells	Number of Samples (%)
Mild Inflammation	10 (20%)
Moderate Inflammation	25 (50%)
Severe Inflammation	15 (30%)

The presence of inflammatory infiltrates, predominantly composed of lymphocytes and plasma cells, was noted in all samples. As indicated in Table 1, moderate inflammation was the most common finding, followed by severe inflammation in 30% of the cases.

Table 2. Collagen Fiber Disruption in Periodontal Ligament Samples

Collagen Fiber	Organization Number of Samples (%) |
Well-Organized	8 (16%)
Partially Disorganized	15 (30%)
Completely Disorganized	27 (54%)

The disruption of collagen fibers within the periodontal ligament was observed in the majority of the cases. Table 2 shows that more than half (54%) of the samples exhibited complete disorganization of collagen fibers.

Table 3. Cementum Resorption in Periodontal Ligament Samples

Cementum Condition	Number of Samples (%)
No Resorption	18 (36%)
Mild Resorption	16 (32%)
Severe Resorption	16 (32%)

Cementum resorption was present in 64% of the samples, as seen in Table 3. Both mild and severe resorption were noted in equal proportions.

Table 4. Vascular Changes in Periodontal Ligament Samples

Vascularity	Number of Samples (%)
Normal	10 (20%)
Increased	40 (80%)

The vascular changes observed in the samples included increased vascularity in 80% of the cases (Table 4). The hyperemia and endothelial cell proliferation observed in these samples reflect the chronic inflammatory state.

Table 5. Fibroblast Population in Periodontal Ligament Samples

Fibroblast Condition	Number of Samples (%)
Normal Fibroblast Count	14 (28%)
Decreased Fibroblast Count	36 (72%)

Fibroblast degeneration and depletion were evident in 72% of the samples (Table 5). This depletion suggests impaired tissue regeneration and repair in chronic periodontitis patients.

Discussion

Macrophage infiltration and tissue destruction observed in the present study of the periodontal ligament in chronic periodontitis patients of Peshawar elucidate the pathogenesis of the disease. The host response to periodontal pathogens is well documented by the observations of a dense inflammatory infiltrate perivascular and pericryptal which is mainly lymphocytic and plasmatic in nature. While this is a perfectly adequate mechanism to control infection this has the effect of releasing to inflammatory mediators such as interleukin-1 (IL-1), and tumor necrosis factor-alpha (TNF-alpha) that promote tissue degradation (12).

The alteration of the collagen fibers of the periodontal ligament is another discovery here, as these fibers are crucial to anchoring the tooth. The degradation is mainly catabolized by matrix metalloproteinase, which are induced by bacterial infection and inflammatory cytokines. Several MMPs have been found, but MMP-8 and MMP-9 have been found to exhibit major roles for collagen degradation in periodontal diseases association (13). The immunohistochemical analysis in our study confirmed increased expression of MMPs in the PDL, further supporting their role in tissue destruction (14).

Cementum resorption is another important change which deserves consideration since cementum is vital for attachment of PDL fibers to the tooth. This resorption of cementum has probably occurred because the chronic inflammation has provoked osteoclastic activity. Earlier studies have shown that raised receptor activator of nuclear factor kappa-B ligand (RANKL) levels in the periodontal tissues enhance osteoclast formation and function to degrade alveolar bone and cementum (15).

The vascular changes observed in the PDL, particularly the increase in blood vessel numbers and hyperemia, suggest an ongoing attempt by the body to repair tissue damage by delivering immune cells and nutrients to the site of inflammation. However, despite this increased vascularity, the chronic nature of the disease overwhelms the reparative capacity of the periodontal tissues, leading to progressive destruction (16).

Fibroblast degeneration and depletion are indicative of impaired tissue regeneration in chronic periodontitis. Fibroblasts play a crucial role in maintaining the structural integrity of the PDL by producing collagen and other extracellular matrix components. Their depletion in periodontitis patients compromises the tissue's ability to repair itself, leading to further degradation and eventual tooth loss (17).

In conclusion, the histopathological changes observed in the periodontal ligament of chronic periodontitis patients in Peshawar are consistent with those reported in other populations, suggesting that similar pathological processes are at play. These findings underscore the importance of early

diagnosis and intervention in periodontal disease to prevent irreversible damage to the tooth-supporting structures.

References

1. Offenbacher S, Barros SP, Singer RE, Moss K, Williams RC, Beck JD. Periodontal disease at the biofilm-gingival interface. J Periodontol. 2007;78(10):1911-25.
2. Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal diseases. Lancet. 2005;366(9499):1809-20.
3. Nazir MA. Prevalence of periodontal disease, its association with systemic diseases and prevention. Int J Health Sci (Qassim). 2017;11(2):72-80.
4. Lindhe J, Lang NP, Karring T. Clinical Periodontology and Implant Dentistry. 5th ed. Blackwell Munksgaard; 2008. p. 35-54.
5. Reynolds JJ, Meikle MC. Mechanisms of connective tissue matrix destruction in periodontitis. Periodontol 2000. 1997;14(1):144-57.
6. Van Dyke TE, Dave S. Risk factors for periodontitis. J Int Acad Periodontol. 2005;7(1):3-7.
7. Caton JG, Armitage G, Berglundh T, Chapple IL, Jepsen S, Kornman KS, et al. A new classification scheme for periodontal and peri-implant diseases and conditions – Introduction and key changes from the 1999 classification. J Clin Periodontol. 2018;45(Suppl 20):S1-S8.
8. Darveau RP. Periodontitis: a polymicrobial disruption of host homeostasis. Nat Rev Microbiol. 2010;8(7):481-90.
9. Kinane DF, Stathopoulou PG, Papapanou PN. Periodontal diseases. Nat Rev Dis Primers. 2017;3(1):17038.
10. Berglundh T, Donati M, Zitzmann NU, Abrahamsson I, Lindhe J. Bacterial colonization of the peri-implant sulcus in partially edentulous subjects: a 3-year prospective study. Clin Oral Implants Res. 2006;17(4):420-6.
11. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. J Periodontol. 2018;89(Suppl 1):S159-72.
12. Kornman KS, Page RC, Tonetti MS. The host response to the microbial challenge in periodontitis: assembling the players. Periodontol 2000. 1997;14(1):33-53.
13. Sorsa T, Tjaderhane L, Salo T. Matrix metalloproteinases (MMPs) in oral diseases. Oral Dis. 2004;10(6):311-8.
14. Ryan ME. Clinical applications for host modulatory therapy. Compend Contin Educ Dent. 2002;23(1):6-11.
15. Graves DT, Li J, Cochran DL. Inflammation and uncoupling as mechanisms of periodontal bone loss. J Dent Res. 2011;90(1):143-53.
16. Birkedal-Hansen H. Role of cytokines and inflammatory mediators in tissue destruction. J Periodontal Res. 1993;28(6):500-10.
17. Chapple IL, Genco R. Diabetes and periodontal diseases: consensus report of the Joint EFP/AAP Workshop on Periodontitis and Systemic Diseases. J Clin Periodontol. 2013;40(Suppl 14):S106-12.