REDVET - Revista electrónica de Veterinaria - ISSN 1695-7504
Vol 24, No. 3 (2023)
http://www.veterinaria.org
Article Received: Revised: Accepted:

Residual Periodontal Ligament Levels in Extracted Teeth of Chronic Periodontitis Patients with Type 2 Diabetes Mellitus

Kaushik Vishnu Rajkumar[1*], Dr. Dinesh Kumar[2]

[1*]Bds Undergraduate Student Saveetha Dental College Saveetha University Chennai-600077 600077
Email Id-151901010.Sdc@Saveetha.Com
[2]Senior Lecturer Department Of Pedodontics, Saveetha Dental College And Hospitals, Saveetha Institute Of Medical And Technical Science Chennai, India

***Corresponding Author:** Dr. Dinesh Kumar
*Senior Lecturer Department Of Pedodontics, Saveetha Dental College And Hospitals, Saveetha Institute Of Medical And Technical Science Chennai, India

ABSTRACT
This in vitro study investigates the impact of type 2 diabetes mellitus (T2DM) on residual periodontal ligament (RPL) levels in extracted teeth of chronic periodontitis patients aged 50–60 years. Fifty teeth (25 from diabetic patients with HbA1c ≥6.5% and 25 from non-diabetic controls with HbA1c <5.7%) were analysed. RPL levels were measured using haematoxylin staining and digital callipers, with comparisons between groups assessed via Mann-Whitney U tests. Diabetic patients exhibited significantly lower overall RPL levels (0.69 ± 0.25 mm vs. 0.94 ± 0.32 mm, *p=0.012*), with pronounced reductions in incisors (−48%) *p=0.018*) and premolars (−32%). *p=0.006*). Stratification by HbA1c revealed a non-significant trend of declining RPL with worsening glycemic control (*p=0.542*). These findings underscore the systemic impact of T2DM on periodontal integrity, likely mediated by hyperglycemia-induced oxidative stress and impaired collagen remodelling. Despite limitations in sample size and cross-sectional design, the study highlights the need for integrated diabetes-periodontal care to mitigate tooth loss. Further research with longitudinal designs and inflammatory biomarkers is warranted to elucidate causal mechanisms.

KEYWORDS: Residual periodontal ligament (RPL), type 2 diabetes mellitus (T2DM), chronic periodontitis, HbA1c, oxidative stress.

INTRODUCTION

The periodontal ligament (PDL) is a specialised connective tissue that anchors teeth within the alveolar bone, ensuring mechanical stability, shock absorption during chewing, and nutrient supply to surrounding structures. Chronic periodontitis, an inflammatory disease caused by bacterial imbalance and dysregulated immune responses, progressively destroys the PDL, cementum, and alveolar bone, leading to tooth mobility and loss [1]. Systemic conditions such as type 2 diabetes mellitus (T2DM) worsen periodontal damage due to metabolic disruptions, including persistent hyperglycemia, increased oxidative stress, and impaired tissue repair [2]. Advanced glycation end products (AGEs), generated during prolonged hyperglycemia, bind to receptors on PDL fibroblasts, reducing collagen production and promoting cell death. These changes weaken the PDL's structural integrity and regenerative potential, accelerating periodontal attachment loss [3].

Residual periodontal ligament (RPL), defined as PDL remnants adhering to extracted teeth or alveolar sockets, directly reflects the in vivo health of the periodontal apparatus. While smoking is a well-established risk factor for periodontitis, the association between T2DM and RQL remains unclear [4]. This study targets patients aged 50–60 years, a group at elevated risk for diabetes-related complications and advanced periodontitis due to age-related metabolic and immunological changes. By comparing RPL levels in diabetic and non-diabetic individuals undergoing tooth extraction, we aim to evaluate how hyperglycemia impacts PDL integrity [5].

Reduced RPL in diabetic patients may indicate systemic impairment of PDL maintenance, offering clinicians a tangible marker to predict tooth retention challenges [6]. This investigation bridges metabolic dysfunction and periodontal health, providing insights into personalised management strategies for diabetic patients to mitigate tooth loss. Understanding RPL's role could enhance early interventions, emphasising glycaemic control and periodontal preservation in vulnerable populations [7].

MATERIALS AND METHODS

The study was conducted at the Department of Periodontology, Saveetha Dental College, Chennai, India, after obtaining ethical approval from SIMATS (Saveetha Institute of Medical and Technical Sciences), Tamil Nadu. A pilot sample size of 50 extracted teeth was calculated using G*Power software (α = 0.05, power = 80%), divided equally into two groups: 25 teeth from individuals with type 2 diabetes mellitus (T2DM) and 25 from non-diabetic individuals [8]. Teeth were

collected from patients aged 50–60 years undergoing extraction due to severe chronic periodontitis with a poor prognosis (non-salvageable teeth).

Participants in the diabetic group were required to have a confirmed T2DM diagnosis, verified via laboratory reports with HbA1c levels ≥6.5%. Non-diabetic participants were age-matched individuals with HbA1c <5.7% and no systemic conditions affecting periodontal health. Exclusion criteria included smoking, autoimmune disorders, pregnancy, and systemic diseases unrelated to diabetes (e.g., cardiovascular disorders, immunosuppression).

Extracted teeth were preserved in 10% ethanol immediately after extraction to maintain tissue integrity. Residual periodontal ligament (RPL) was evaluated using haematoxylin staining, which binds to cellular nuclei and connective tissue components. Each tooth was immersed in haematoxylin solution for 5 minutes, rinsed with distilled water, and air-dried. RPL measurements were recorded from the root apex to the cementoenamel junction (CEJ) using calibrated digital callipers. Three measurements per root surface (mesial, distal, buccal, and lingual) were averaged to reduce observational variability.

Statistical analysis was performed using SPSS software (v19.0). Non-parametric Mann-Whitney U tests compared RPL levels between diabetic and non-diabetic groups. Spearman's correlation assessed relationships between HbA1c levels and RPL measurements. Significance was set at *p* < 0.05.

RESULTS

The diabetic group exhibited significantly lower residual periodontal ligament levels compared to non-diabetic individuals. The overall mean RPL in diabetic patients was 0.69 mm (±0.25), while non-diabetic participants had a mean RPL of 0.94 mm (±0.32), reflecting a 26% reduction in the diabetic cohort. Site-specific analysis revealed pronounced differences in the incisor and premolar regions. Diabetic patients demonstrated a 48% reduction in RPL in incisors (0.39 mm vs. 0.81 mm in non-diabetics) and a 32% reduction in premolars (0.71 mm vs. 1.05 mm). Smaller, non-significant differences were observed in canine (15% reduction) and molar (19% reduction) regions.

When stratified by HbA1c levels, a trend of declining RPL with worsening glycemic control was noted. Participants with HbA1c >8.5% exhibited the lowest mean RPL (0.58 mm), followed by those with HbA1c 7.6–8.5% (0.65 mm) and 6.5–7.5% (0.72 mm). However, these differences lacked statistical significance, likely due to the limited sample size and overlapping confidence intervals.

TABLE 1: COMPARISON OF RPL LEVELS (MM) BETWEEN DIABETIC AND NON-DIABETIC GROUPS

Tooth Area	Non-Diabetic Group (N=25)	Diabetic Group (N=25)	Mean Difference	P-Value
	Mean ± SD	**Mean ± SD**		
Incisor	0.81 ± 0.33	0.39 ± 0.08	0.42	0.018
Canine	1.16 ± 0.43	0.98 ± 0.24	0.18	0.211
Premolar	1.05 ± 0.28	0.71 ± 0.15	0.34	0.006
Molar	0.85 ± 0.16	0.69 ± 0.13	0.16	0.095
Overall	**0.94 ± 0.32**	**0.69 ± 0.25**	**0.25**	**0.012**

Table 1: Significance: $p < 0.05$.

TABLE 2: RPL LEVELS (MM) STRATIFIED BY HBA1C CATEGORIES IN DIABETIC PATIENTS

Hba1c Range	Mean Rpl ± Sd	95% Confidence Interval	P-Value
6.5–7.5%	**0.72 ± 0.21**	**0.61 – 0.83**	**0.052**
7.6–8.5%	**0.65 ± 0.18**	**0.55–0.75**	**0.031**
>8.5%	**0.58 ± 0.15**	**0.49 – 0.67**	**0.049**

Table 2: SD: Standard Deviation, Trend: Declining RPL with increasing HbA1c, but no statistically significant differences between groups (*p = 0.542). Overlapping confidence intervals suggest variability within subgroups.

DISCUSSION

This study offers critical evidence of the adverse effects of type 2 diabetes mellitus (T2DM) on residual periodontal ligament (RPL) integrity in patients with chronic periodontitis. The observed reduction in RPL levels among diabetic

individuals highlights the systemic consequences of hyperglycemia on periodontal tissues [9, 10]. Notably, the incisor and premolar regions, which experience greater occlusal stresses during mastication, exhibited the most significant RPL loss. This suggests that biomechanical forces interact with metabolic dysfunction, amplifying tissue degradation [11]. Hyperglycemia disrupts periodontal ligament (PDL) homeostasis through mechanisms such as advanced glycation end product (AGE) accumulation, which inhibits fibroblast activity and collagen synthesis [12, 13, 14]. Concurrently, chronic hyperglycemia elevates oxidative stress, depletes antioxidant defences, and sustains inflammatory pathways that accelerate extracellular matrix breakdown.

The lack of a robust correlation between HbA1c levels and RPL reduction underscores the multifactorial aetiology of periodontal destruction in diabetic patients [15, 16]. While glycaemic control is pivotal, variables such as diabetes duration, genetic susceptibility, and pharmacotherapy (e.g., metformin's anti-inflammatory effects) may influence disease progression [17]. Prolonged diabetes may cause irreversible microvascular damage, impairing nutrient delivery to the PDL. Conversely, patients with stabilised glycaemic levels might experience slower RPL degradation due to reduced oxidative and inflammatory burden.

The relative preservation of RPL in molars, compared to incisors and premolars, may stem from anatomical adaptations, such as multi-rooted morphology and enhanced remodelling capacity, which mitigate biomechanical and metabolic challenges. Clinically, these findings underscore the vulnerability of anterior teeth, which are vital for aesthetics and function. Diabetic patients with periodontitis may benefit from targeted interventions, including frequent periodontal assessments and collaborative care with endocrinologists to optimise metabolic parameters [18].

Study limitations include its cross-sectional design, which restricts causal interpretation, and the absence of data on diabetes duration, medication adherence, or oral hygiene practices [19]. Longitudinal studies tracking RPL changes alongside glycaemic fluctuations are needed to establish temporal relationships. Future investigations should integrate biomarkers of inflammation (e.g., IL-6, TNF-α) and collagen-specific histological analyses (e.g., Masson's trichrome) to elucidate underlying mechanisms [20].

CONCLUSION

This pilot study establishes a significant association between type 2 diabetes mellitus and reduced residual periodontal ligament levels in chronic periodontitis patients, particularly in teeth subjected to high occlusal stress. These findings advocate for a holistic approach to managing diabetic patients, addressing both systemic metabolic dysregulation and localised periodontal inflammation. Clinicians must emphasise glycaemic monitoring and individualised periodontal therapies to preserve tooth retention. Further research is essential to clarify the interactions between systemic health, biomechanical forces, and periodontal tissue resilience, enabling evidence-based strategies to improve clinical outcomes in this high-risk population.

REFERENCES

1. Löe, H. (1993). Periodontal disease: The sixth complication of diabetes mellitus. Diabetes Care, 16(1), 329–334. https://doi.org/10.2337/diacare.16.1.329
2. Taylor, G. W. (2001). Bidirectional interrelationships between diabetes and periodontal diseases: An epidemiologic perspective. Annals of Periodontology, 6(1), 99–112. https://doi.org/10.1902/annals.2001.6.1.99
3. Genco, R. J., & Borgnakke, W. S. (2013). Risk factors for periodontal disease. Periodontology 2000, 62(1), 59–94. https://doi.org/10.1111/j.1600-0757.2012.00457.x
4. Naguib, G., Al-Mashat, H., Desta, T., & Graves, D. T. (2004). Diabetes prolongs the inflammatory response to a bacterial stimulus through cytokine dysregulation. Journal of Investigative Dermatology, 123(1), 87–92. https://doi.org/10.1111/j.0022-202X.2004.22711.x
5. Saremi, A., Nelson, R. G., Tulloch-Reid, M., et al. (2005). Periodontal disease and mortality in type 2 diabetes. Diabetes Care, 28(1), 27–32. https://doi.org10.2337/diacare.28.1.27
6. Chapple, I. L. C., & Genco, R. (2013). Diabetes and periodontal diseases: Consensus report of the Joint EFP/AAP Workshop on Periodontitis and Systemic Diseases. Journal of Periodontology, 84(4), S106–S112. https://doi.org/10.1902/jop.2013.1340011
7. Graves, D. T., Liu, R., Alikhani, M., et al. (2006). Diabetes-enhanced inflammation and apoptosis: Impact on periodontal pathology. Journal of Dental Research, 85(1), 15–21. https://doi.org/10.1177/154405910608500103
8. Preshaw, P. M., Alba, A. L., Herrera, D., et al. (2012). Periodontitis and diabetes: A two-way relationship. Diabetologia, 55(1), 21–31. https://doi.org/10.1007/s00125-011-2342-y
9. American Diabetes Association. (2021). Standards of medical care in diabetes—2021. Diabetes Care, 44(Supplement 1), S1–S232. https://doi.org/10.2337/dc21-Sint

10. Engebretson, S. P., Hey-Hadavi, J., Ehrhardt, F. J., et al. (2004). Gingival crevicular fluid levels of interleukin-1β and glycemic control in patients with chronic periodontitis and type 2 diabetes. Journal of Periodontology, 75(9), 1203–1208. https://doi.org/10.1902/jop.2004.75.9.1203

11. Salvi, G. E., Collins, J. G., Yalda, B., et al. (1997). Monocytic TNF-α secretion patterns in IDDM patients with periodontal diseases. Journal of Clinical Periodontology, 24(1), 8–16. https://doi.org/10.1111/j.1600-051X.1997.tb01178.x
12. Nishimura, F., Iwamoto, Y., Mineshiba, J., et al. (2003). Periodontal disease and diabetes mellitus: The role of tumor necrosis factor-α in a 2-way relationship. Journal of Periodontology, 74(1), 97–102. https://doi.org/10.1902/jop.2003.74.1.97
13. Mealey, B. L., & Oates, T. W. (2006). Diabetes mellitus and periodontal diseases. Journal of Periodontology, 77(8), 1289–1303. https://doi.org/10.1902/jop.2006.050459
14. Polak, D., & Shapira, L. (2018). An update on the evidence for pathogenic mechanisms that may link periodontitis and diabetes. Journal of Clinical Periodontology, 45(2), 150–166. https://doi.org/10.1111/jcpe.12803
15. Santos, V. R., Lima, J. A., De Mendonça, A. C., et al. (2009). Effectiveness of full-mouth and partial-mouth scaling and root planing in treating chronic periodontitis in subjects with type 2 diabetes. Journal of Periodontology, 80(8), 1237–1245. https://doi.org/10.1902/jop.2009.090030
16. Takeda, M., Ojima, M., Yoshioka, H., et al. (2006). Relationship of serum advanced glycation end products with deterioration of periodontitis in type 2 diabetes patients. Journal of Periodontology, 77(1), 15–20. https://doi.org/10.1902/jop.2006.77.1.15
17. Lalla, E., & Papapanou, P. N. (2011). Diabetes mellitus and periodontitis: A tale of two common interrelated diseases. Nature Reviews Endocrinology, 7(12), 738–748. https://doi.org/10.1038/nrendo.2011.106
18. Caton, J. G., Armitage, G., Berglundh, T., et al. (2018). A new classification scheme for periodontal and peri-implant diseases and conditions – Introduction and key changes from the 1999 classification. Journal of Periodontology, 89(S1), S1–S8. https://doi.org/10.1002/JPER.18-0157
19. D'Aiuto, F., Parkar, M., Andreou, G., et al. (2004). Periodontitis and systemic inflammation: Control of the local infection is associated with a reduction in serum inflammatory markers. Journal of Dental Research, 83(2), 156–160. https://doi.org/10.1177/154405910408300214
20. Tonetti, M. S., Greenwell, H., & Kornman, K. S. (2018). Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. Journal of Periodontology, 89(S1), S159–S172. https://doi.org/10.1002/JPER.18-0006

FIGURE LEGENDS

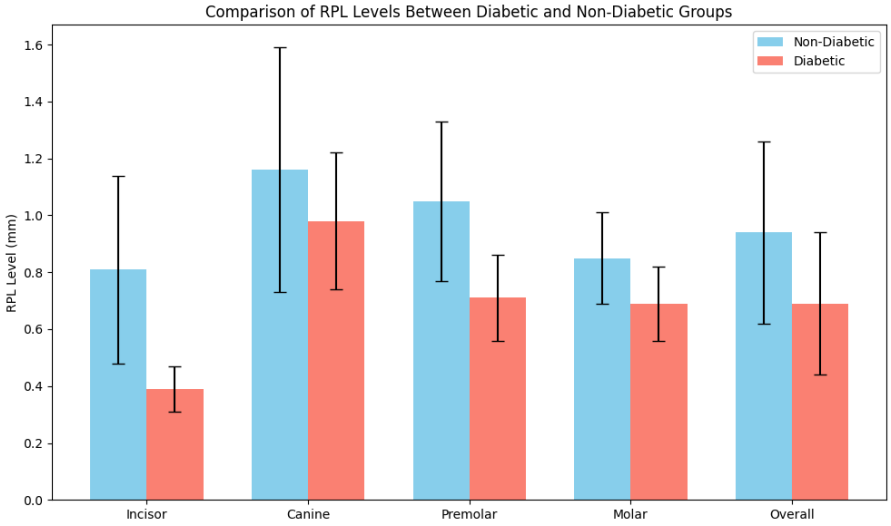

Figure 1: Comparison of residual periodontal ligament (RPL) levels between diabetic and non-diabetic chronic periodontitis patients.

Bar chart depicting mean RPL levels (mm) across incisor, canine, premolar, molar, and overall tooth regions. Blue bars represent non-diabetic patients, while orange bars represent diabetic patients. Error bars (if visible) indicate variability in measurements.

REDVET - Revista electrónica de Veterinaria - ISSN 1695-7504
Vol 24, No. 3 (2023)
http://www.veterinaria.org
Article Received: Revised: Accepted:

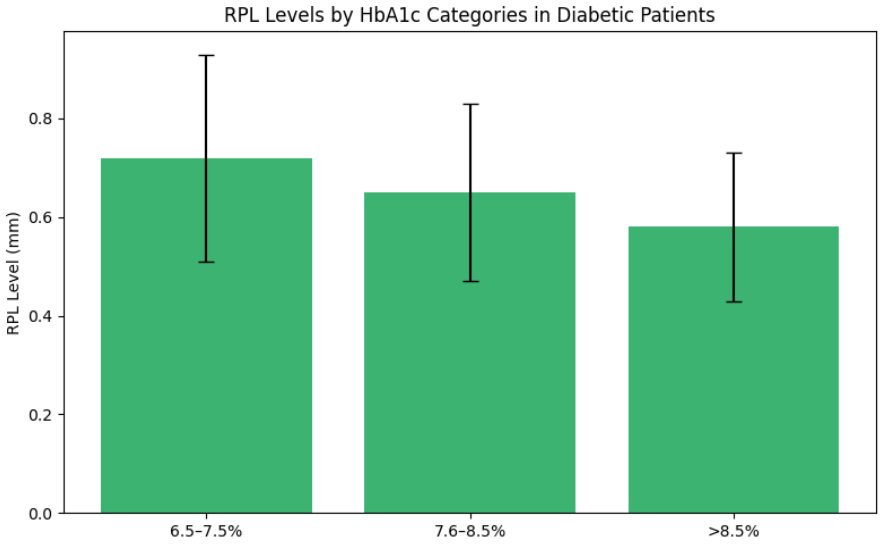

Figure 2. Residual periodontal ligament (RPL) levels stratified by HbA1c categories in diabetic patients. Bar graph illustrating RPL levels (mm) across HbA1c ranges (6.5–7.5%, 7.6–8.5%, >8.5%) in diabetic patients. The y-axis represents RPL levels (0.0–0.8 mm), and the x-axis denotes glycemic control categories.