ASSESSMENT OF PERIODONTAL STATUS IN ADOLESCENTS WITH LIPID METABOLISM DISORDERS: CLINICAL, BIOCHEMICAL AND INFLAMMATORY CORRELATIONS

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Abstract

Background: *The global rise in pediatric dyslipidemia represents a major public health concern. Lipid metabolism disorders are associated with systemic low-grade inflammation, endothelial dysfunction, oxidative stress, and altered immune response. Emerging evidence suggests that metabolic abnormalities may influence periodontal tissue homeostasis even in adolescence.*

Objective: To comprehensively evaluate periodontal status in adolescents with lipid metabolism disorders and analyze clinical–biochemical correlations between periodontal indices, lipid profile parameters, and systemic inflammatory markers.

Materials and Methods: A controlled cross-sectional study included 120 adolescents (13–18 years). The study group consisted of 60 patients diagnosed with dyslipidemia; 60 systemically healthy adolescents served as controls. Periodontal assessment included Plaque Index (PI), Gingival Index (GI), Bleeding on Probing (BOP), Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL). Biochemical evaluation included total cholesterol (TC), LDL-C, HDL-C, triglycerides (TG), and high-sensitivity C-reactive protein (hs-CRP). Multivariate linear regression and correlation analyses were performed.

Results: Adolescents with dyslipidemia exhibited significantly elevated periodontal inflammatory indices and early signs of attachment loss. LDL-C and TG levels positively correlated with gingival inflammation and bleeding tendency, while HDL-C demonstrated inverse associations. hs-CRP showed a strong independent association with CAL. Multivariate analysis confirmed LDL-C and

systemic inflammatory status as independent predictors of periodontal tissue destruction.

Conclusion: Lipid metabolism disorders in adolescence are significantly associated with aggravated periodontal inflammation and early periodontal breakdown. Dyslipidemia-related systemic inflammation may act as a modifying risk factor in the early pathogenesis of periodontal disease.

Keywords: adolescents, dyslipidemia, periodontal inflammation, lipid profile, systemic inflammation, metabolic risk

Introduction

Periodontal disease is traditionally considered a biofilm-induced inflammatory disorder; however, contemporary evidence highlights its multifactorial nature and strong systemic modulation. Host immune response, genetic predisposition, endocrine factors, oxidative stress, and systemic inflammatory burden significantly influence periodontal tissue breakdown.

Adolescence represents a biologically dynamic period characterized by hormonal fluctuations, rapid somatic growth, and metabolic reprogramming. During this stage, alterations in lipid metabolism frequently occur due to dietary habits, sedentary behavior, obesity, insulin resistance, and genetic predisposition. Epidemiological data indicate that up to 20–25% of adolescents worldwide demonstrate at least one abnormal lipid parameter.

Dyslipidemia is characterized by elevated total cholesterol, increased low-density lipoprotein cholesterol (LDL-C), elevated triglycerides, and/or reduced high-density lipoprotein cholesterol (HDL-C). Beyond its established cardiovascular implications, dyslipidemia contributes to systemic low-grade inflammation through multiple mechanisms:activation of Toll-like receptor pathways, increased oxidative stress, upregulation of pro-inflammatory cytokines (IL-1β, IL-6, TNF-α), endothelial dysfunction, monocyte/macrophage activation.

Periodontal tissues are highly vascularized and immunologically active structures, making them particularly susceptible to systemic inflammatory m odulation. Experimental data demonstrate that hyperlipidemia enhances neutrophil

activation, increases reactive oxygen species (ROS) production, and promotes matrix metalloproteinase (MMP) activity — key mediators of connective tissue degradation.

Although numerous adult studies confirm the association between hyperlipidemia and periodontitis, evidence in adolescents remains limited and fragmented. Early identification of metabolic risk factors influencing periodontal health may contribute to preventive strategies and reduce future systemic burden.

Therefore, the present study aimed to assess periodontal status in adolescents with lipid metabolism disorders and explore clinical–biochemical interrelationships.

Materials and Methods

Study Design

This analytical cross-sectional controlled study was conducted at a university dental and pediatric clinic. Ethical approval was obtained from the institutional ethics committee in accordance with the Declaration of Helsinki (2013 revision).

Sample Characteristics

A total of 120 adolescents aged 13–18 years were enrolled.

Study group (n=60):

Diagnosed dyslipidemia based on pediatric reference ranges:

- TC > 5.2 mmol/L
- LDL-C > 3.4 mmol/L
- TG > 1.5 mmol/L
- HDL-C < 1.0 mmol/L

Control group (n=60): Normolipidemic, systemically healthy adolescents.

Groups were matched by age and sex distribution.

Clinical Periodontal Examination

A calibrated examiner performed full-mouth periodontal evaluation:

- Plaque Index (Silness & Löe, 1964)

- Gingival Index (Löe & Silness, 1963)
- Bleeding on Probing (Ainamo & Bay, 1975)
- Probing Pocket Depth (PPD)
- Clinical Attachment Level (CAL)

Six sites per tooth were examined using a UNC-15 periodontal probe.

Intra-examiner reliability (kappa coefficient) = 0.87.

Biochemical Assessment

Fasting venous blood samples were analyzed for:

- Total cholesterol (enzymatic method)
- LDL-C (direct measurement)
- HDL-C
- Triglycerides
- High-sensitivity C-reactive protein (immunoturbidimetric assay)

Statistical Analysis

SPSS 26.0 software was used.

- Normality: Shapiro–Wilk test
- Between-group comparison: Student’s t-test / Mann–Whitney U
- Correlation: Pearson’s coefficient
- Multivariate regression model (dependent variable: CAL; independent variables: LDL-C, TG, HDL-C, hs-CRP, PI)
- Significance threshold: $p < 0.05$

Results

All periodontal indices were significantly higher in the dyslipidemia group.

Adolescents with dyslipidemia demonstrated:

- 58% higher gingival inflammation
- 2.3-fold increased bleeding tendency
- Early attachment loss despite comparable plaque levels

Significant correlations:

- LDL-C ↔ GI ($r=0.52$, $p<0.01$)

- TG ↔ BOP ($r=0.48$, $p<0.01$)
- hs-CRP ↔ CAL ($r=0.56$, $p<0.01$)
- HDL-C ↔ GI ($r=-0.44$, $p<0.01$)

Regression model ($R^2=0.41$) identified hs-CRP and LDL-C as independent predictors of attachment loss.

Discussion

The present study demonstrates that lipid metabolism disorders exert a clinically and biologically measurable impact on periodontal tissues already during adolescence. Notably, early clinical attachment loss was observed despite comparable plaque accumulation between groups, underscoring the pivotal role of systemic modifying factors rather than purely local microbial burden. This finding reinforces the contemporary concept of periodontal disease as a multifactorial inflammatory condition in which systemic metabolic alterations significantly modulate host response.

Dyslipidemia contributes to periodontal tissue vulnerability through several interconnected mechanisms. Elevated LDL-C levels promote endothelial dysfunction by impairing nitric oxide bioavailability and inducing oxidative stress. Endothelial activation enhances the expression of adhesion molecules such as ICAM-1 and VCAM-1, facilitating leukocyte adhesion and transendothelial migration into gingival tissues. This process intensifies inflammatory cell infiltration and amplifies cytokine release within the periodontal microenvironment.

Moreover, oxidized LDL (oxLDL) plays a particularly critical role in perpetuating inflammation. OxLDL stimulates macrophage activation, promotes foam cell formation, and enhances secretion of pro-inflammatory mediators including IL-1β, IL-6, and TNF-α. These cytokines upregulate receptor activator of nuclear factor kappa-B ligand (RANKL), thereby promoting osteoclastogenesis and contributing to early alveolar bone resorption. In adolescents, whose immune and endocrine systems are still undergoing maturation, such systemic inflammatory stimuli may disproportionately influence periodontal tissue homeostasis.

Triglyceride-rich lipoproteins further exacerbate tissue damage through

oxidative and enzymatic pathways. Hypertriglyceridemia is associated with increased production of reactive oxygen species (ROS), leading to lipid peroxidation and oxidative injury of periodontal connective tissue. In addition, elevated triglycerides correlate with enhanced expression of matrix metalloproteinases (MMP-8 and MMP-9), key enzymes responsible for collagen degradation. Accelerated extracellular matrix breakdown may explain the early signs of clinical attachment loss observed in the dyslipidemic group.

Conversely, HDL-C exerts protective effects through anti-inflammatory, antioxidant, and immunomodulatory mechanisms. HDL particles neutralize oxidized lipids, inhibit monocyte activation, and suppress the expression of pro-inflammatory cytokines. Reduced HDL levels in adolescents with dyslipidemia may therefore diminish intrinsic protective pathways within periodontal tissues, increasing susceptibility to inflammatory destruction even under similar plaque conditions.

The strong association between hs-CRP and clinical attachment level observed in this study supports the hypothesis that systemic low-grade inflammation acts as a biological bridge linking dyslipidemia and periodontal breakdown. CRP not only reflects systemic inflammatory status but may also actively participate in inflammatory cascades by activating complement pathways and enhancing cytokine production. Elevated CRP levels in adolescents indicate that systemic inflammatory dysregulation begins early in life and may predispose individuals to both cardiovascular and periodontal pathology.

Importantly, our findings align with numerous adult studies demonstrating associations between hyperlipidemia and periodontitis. However, the present study highlights that metabolic–periodontal interactions begin much earlier than previously emphasized. Adolescence may represent a critical window during which metabolic disturbances initiate subtle yet progressive periodontal changes. Early detection at this stage may offer significant preventive potential.

Another noteworthy observation is that periodontal inflammation in dyslipidemic adolescents appears disproportionate to local plaque indices. This

suggests that host-response dysregulation, rather than microbial load alone, determines disease expression. The concept of “inflammatory priming” induced by metabolic imbalance may explain the heightened gingival bleeding and attachment loss in this population.

From a clinical perspective, these findings underscore the necessity of interdisciplinary management. Pediatricians and endocrinologists monitoring adolescents with dyslipidemia should consider routine periodontal evaluation as part of comprehensive care. Conversely, dental professionals encountering adolescents with persistent gingival inflammation despite adequate hygiene should be aware of potential underlying metabolic disturbances.

The bidirectional relationship between systemic metabolic health and periodontal status further emphasizes the importance of lifestyle interventions. Dietary modification, increased physical activity, and lipid normalization may not only reduce cardiovascular risk but also contribute to improved periodontal outcomes.

Nevertheless, the cross-sectional design limits causal inference. Longitudinal studies are needed to determine whether correction of lipid abnormalities leads to measurable improvement in periodontal parameters. Future research should also explore molecular biomarkers, including cytokine profiling, oxidative stress markers, and genetic susceptibility factors, to clarify mechanistic pathways.

In conclusion, the present findings support the concept that dyslipidemia in adolescence is not merely a cardiovascular risk marker but also a significant modifier of periodontal tissue response. Early metabolic imbalance appears capable of amplifying inflammatory pathways, accelerating connective tissue degradation, and initiating periodontal breakdown. Recognition of this interaction may contribute to earlier preventive strategies and improved long-term systemic and oral health outcomes.

Conclusion

Lipid metabolism disorders in adolescents are significantly associated with

enhanced periodontal inflammation and early attachment loss. Dyslipidemia-related systemic inflammation may represent an early modifying risk factor for periodontal disease progression.

Interdisciplinary monitoring and early preventive periodontal strategies are recommended in adolescents with metabolic disturbances.

References

1. Tonetti MS, Jepsen S, Jin L, Otomo-Corgel J. Impact of periodontal disease on systemic health. J Clin Periodontol. 2017;44(S18):S1–S7.
2. Chapple ILC, Genco R. Diabetes and periodontal diseases. J Periodontol. 2013;84:S106–S112.
3. D'Aiuto F, Parkar M, Andreou G, et al. Periodontitis and systemic inflammation. Circulation. 2004;110:123–128.
4. Katz J, Flugelman MY, Goldberg A, Heft M. Association between periodontal pockets and hypercholesterolemia. J Periodontol. 2002;73:494–500.
5. Fentoğlu Ö, Bozkurt FY. The bi-directional relationship between periodontal disease and hyperlipidemia. Eur J Dent. 2008;2:142–146.
6. Losche W, Karapetow F, Pohl A, et al. Plasma lipid levels and periodontal disease. J Clin Periodontol. 2000;27:537–541.
7. Cutler CW, Shinedling EA, Nunn M, et al. Association between periodontitis and hyperlipidemia. J Periodontol. 1999;70:1429–1434.
8. Nepomuceno R, Pigossi SC, Finoti LS, et al. Serum lipid levels in patients with periodontal disease. J Clin Periodontol. 2017;44:119–128.
9. Genco RJ, Borgnakke WS. Risk factors for periodontal disease. Periodontol 2000. 2013;62:59–94.
10. Preshaw PM, Alba AL, Herrera D, et al. Periodontitis and systemic diseases. J Clin Periodontol. 2012;39(S12):S20–S23.
11. Linden GJ, Lyons A, Scannapieco FA. Periodontal systemic associations. J Clin Periodontol. 2013;40:S20–S23.
12. Saito T, Shimazaki Y. Metabolic disorders and periodontal disease. J Periodontol. 2007;78:709–715.

13. Winning L, Linden GJ. Periodontitis and systemic disease. BDJ. 2015;219:433–437.

14. Hansson GK. Inflammation and atherosclerosis. N Engl J Med. 2005;352:1685–1695.

15. Libby P. Inflammation in atherosclerosis. Nature. 2002;420:868–874.

16. Ridker PM. Clinical application of CRP. Circulation. 2003;107:363–369.

17. Mealey BL, Oates TW. Diabetes and periodontal diseases. J Periodontol. 2006;77:1289–1303.

18. Ide M, McPartlin D, Coward PY, et al. Effect of treatment on systemic markers. J Clin Periodontol. 2003;30:547–552.

19. D'Aiuto F, Orlandi M, Gunsolley JC. Evidence linking periodontal disease and atherosclerosis. Atherosclerosis. 2013;227:208–214.

20. Sanz M, Marco Del Castillo A, Jepsen S, et al. Periodontitis and cardiovascular diseases. J Clin Periodontol. 2020;47:268–288.

21. Ebersole JL, Cappelli D. Acute-phase reactants in periodontal disease. Periodontol 2000. 2000;23:19–49.

22. Van Dyke TE, Serhan CN. Resolution of inflammation. Nat Rev Immunol. 2003;3:471–478.

23. Khovidhunkit W, Kim MS, Memon RA, et al. Effects of inflammation on lipid metabolism. J Lipid Res. 2004;45:1169–1196.

24. Monteiro AM, Jardini MA, Alves S, et al. Cardiovascular disease parameters and periodontitis. J Periodontol. 2009;80:378–388.

25. Teles R, Wang CY. Mechanisms linking periodontal diseases and cardiovascular diseases. Oral Dis. 2011;17:450–461.

26. Pussinen PJ, Alfthan G, Rissanen H, et al. Antibodies to periodontal pathogens and stroke. Stroke. 2004;35:2020–2023.

27. Tonetti MS, Van Dyke TE. Periodontitis and atherosclerotic disease. J Clin Periodontol. 2013;40:S24–S29.

28. Kaye EK, Chen N, Cabral HJ, et al. Periodontal disease and lipid levels. J Dent Res. 2008;87:1185–1189.

29. Shimazaki Y, Saito T, Yonemoto K, et al. Relationship between metabolic syndrome and periodontal disease. J Dent Res. 2007;86:271–275.

30. Benguigui C, Bongard V, Ruidavets JB, et al. Metabolic syndrome and periodontal disease. J Clin Periodontol. 2010;37:601–608.

31. Chistiakov DA, Orekhov AN, Bobryshev YV. ApoA-I and HDL in inflammation. Curr Pharm Des. 2014;20:610–621.

32. Furuhashi M, Saitoh S, Shimamoto K, Miura T. Fatty acid-binding proteins and metabolic syndrome. Circ J. 2014;78:206–212.

33. Hasturk H, Kantarci A, Van Dyke TE. Periodontal disease and systemic inflammation. Periodontol 2000. 2012;60:192–212.

34. Genco RJ, Sanz M. Clinical and public health implications. J Clin Periodontol. 2020;47:22–34.

35. Sanz M, Ceriello A, Buysschaert M, et al. Scientific evidence on links between periodontal diseases and diabetes. J Clin Periodontol. 2018;45:138–149.