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Case Report

Gingival Leukoplakia In a Periodontal Patient: Case Report With 8 Years Follow-Up

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Abstract

Gingival leukoplakia corresponds to a persistent white plaque present on keratinised mucosa that does not yield to scraping. Of unknown aetiology, it requires meticulous clinical and histopathological evaluation due to its recurrence and potential risk of malignant transformation. The purpose of this article is to present the case of a periodontal patient with gingival leukoplakia, discussing clinical features, histopathological findings, diagnosis, differential diagnosis, aetiopathogenesis, treatment, follow-up and the risk of malignancy. Surgical excision remains the treatment of choice and should be associated with histopathological examination and prolonged follow-up.

Keywords: *Oral Leukoplakia; Periodontal Diseases; Periodontology; Oral Pathology; Oral Diseases; Oral Surgery.*

Introduction

Periodontal diseases constitute a heterogeneous set of conditions that affect the tissues supporting and protecting the teeth, including biofilm-induced and non-biofilm-induced entities. The current classification of the American Academy of Periodontology organises these conditions according to clinical and aetiological criteria. Among them, non-biofilm-induced gum diseases encompass genetic, systemic, infectious, inflammatory and immunological, traumatic and reactive manifestations[1]. Some of these changes can mimic potentially malignant lesions when they affect keratinised tissues, such as the gums, which reinforces the importance of careful evaluation.

The term leukoplakia refers to a persistent white plaque that does not yield to scraping and is not clinically or histopathologically characterised as another known condition[2]. Histopathological examination reveals hyperkeratosis, acanthosis, and varying degrees of epithelial dysplasia. In patients with periodontal disease, gingival inflammation may hinder initial clinical interpretation, making it necessary to control local factors, exclude traumatic irritations, and perform rigorous clinical-pathological correlation[3].

Oral leukoplakia presents a variable risk of malignant transformation, although only some lesions progress to squamous cell carcinoma. Gingival presentation is less frequent and may be underdiagnosed due to its similarity to reactive changes associated with periodontal disease[4]. In this perspective, the purpose of this article is to present the case of a periodontal patient with gingival leukoplakia, discussing clinical, histopathological, diagnostic, therapeutic and prognostic aspects.

Case Presentation

A Caucasian female patient, 59 years-old, attended the dental clinic for periodontal treatment. Clinically, moderate chronic periodontitis was observed, with the presence of biofilm, dental calculus and extrinsic pigmentation. In addition, gingival recession and exposure of the root surfaces were observed (Figure 1). A white lesion was observed on the mesiobuccal surface of upper right central incisor in the marginal gingiva (Figure 2).

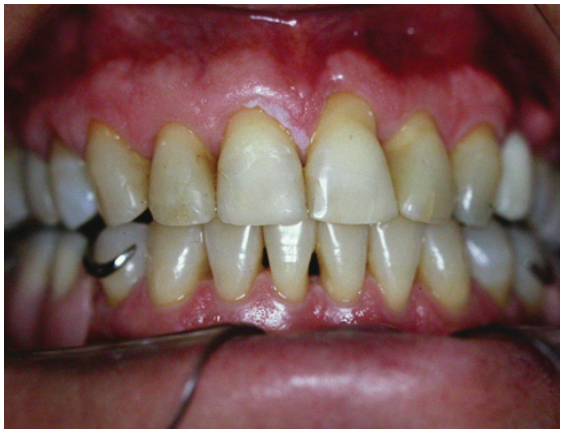

Figure 1. Gingival leukoplakia on the mesiobuccal surface of upper right central incisor in the marginal gingiva.

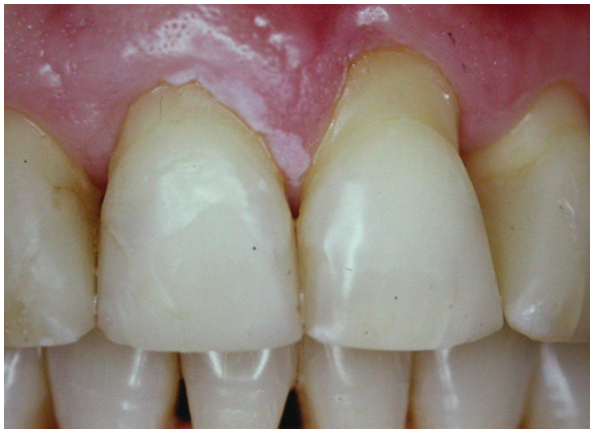

Figure 2. Gingival leukoplakia (close-up view).

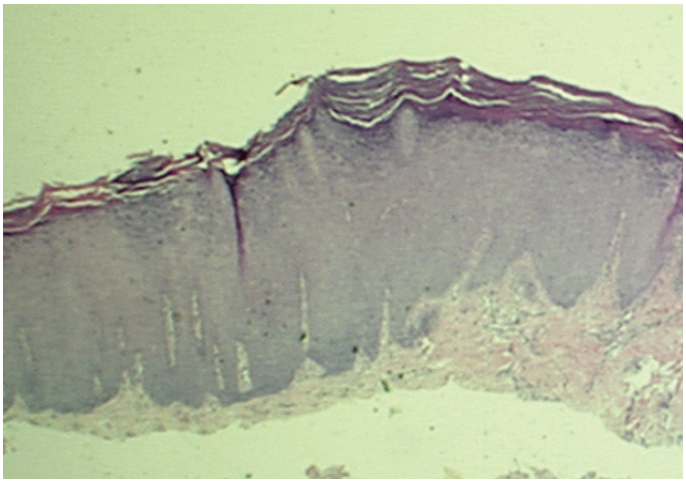

Figure 3. Histopathological aspects of gingival hyperkeratosis (HE; low magnification).

Radiographically, bone loss inherent to periodontal disease was observed.

Periodontal treatment was performed, with scaling and root planing sessions and oral hygiene instruction, yielding satisfactory results.

For clinical diagnosis, the white lesion that persisted after periodontal treatment was scraped without success. Thus, the white lesion was clinically diagnosed as leukoplakia, and surgical removal was indicated. The patient reported no systemic diseases or smoking. There was also no history of trauma in the region.

Under local infiltration anaesthesia, conventional surgical removal was performed with a No. 15 scalpel blade. Surgical cement was applied to the area and remained in place for seven days.

The removed fragment was fixed in 10% formaldehyde and sent to the Surgical Pathology Laboratory of the School of Dentistry, University of São Paulo. Histological sections revealed thickened stratum corneum with areas of hyperkeratosis, acanthosis, and elongation of epithelial extensions (Figure 3). The final diagnosis was hyperkeratosis.

The patient reported no post-operative complications and the area healed without complications. The patient has been monitored through supportive periodontal therapy (periodontal maintenance) for 8 years and there are no signs of recurrence of leukoplakia or periodontal disease.

Discussion

Gingival leukoplakia is featured by persistent, asymptomatic, homogeneous or fissured white plaque with an opaque white colour, smooth or slightly rough surface, sessile and firmly adherent base, and clear or poorly defined borders[5]. The clinical pattern is further defined by its resistance to scraping. It can affect any region of the oral mucosa, including the attached or marginal gingiva[5,6], as observed in the present case report. Careful evaluation of texture, thickness, colour and adhesion is essential to distinguish it from friction keratoses[5].

From a histopathological perspective, the lesions present thickening of the stratum corneum, featured by hyperkeratotic areas, acanthosis and elongation of epithelial extensions, maintaining the integrity of the basement membrane[6]. The histopathological spectrum ranges from hyperkeratotic lesions without dysplasia to cases with mild, moderate or severe dysplasia[6]. Loss of polarity, pleomorphism, and hyperchromatism are described as markers associated with progression[7]. The degree of dysplasia is the main predictor of risk of malignant transformation[8,9]. There is disagreement regarding the prognostic value of parakeratosis, which is considered a low-risk alteration[7] and a possible initial marker of epithelial instability[9].

The prevalence of oral leukoplakia in the global population varies between 1% and 2%, while its specific frequency in the gums is lower and poorly documented[10]. Epidemiological studies differ in terms of gender distribution, reporting a slight female predominance[8,10].

The diagnosis is based on the clinical persistence of the lesion after elimination of irritating factors and control of biofilm. Reactive lesions usually regress after supragingival scaling, while true leukoplakia remains unchanged[2]. Careful re-evaluation is necessary to rule out conditions that mimic keratoses[5]. Biopsy is essential to confirm the diagnosis and determine the degree of dysplasia[3]. Histopathological examination is indispensable to define the associated risk and guide management[2,3,5,8,9].

The differential diagnosis includes friction keratosis, chronic hyperplastic candidiasis, keratotic oral lichen planus, chemical burns, hairy leukoplakia, and hereditary keratinisation disorders. Clinical-histopathological correlation is essential for definitive diagnosis, and flowcharts can help distinguish between these entities[5]. Some immune-mediated diseases can mimic the gingival presentation of white lesions[4]. Differentiation between hyperplastic candidiasis and leukoplakia is essential, as the former presents with hyphal penetration, a finding absent in true leukoplakia[5].

Gingival leukoplakia is classified as a non-biofilm-induced gum disease, and coexisting periodontal inflammation does not explain its primary aetiology[1,4]. The aetiopathogenesis of gingival leukoplakia is multifactorial and involves factors such as smoking, alcohol consumption, and mechanical trauma. Associations between more virulent periodontal pathogens and hyperkeratotic areas have been observed, although no causal relationship has been proven[10,11]. Local inflammatory processes may modulate the epithelial response, but they are not the primary cause of the lesion[8,12]. Areas of persistent mechanical irritation may present whitish changes[10].

Conventional surgical excision remains the treatment of choice, allowing complete removal and adequate histopathological evaluation of the margins[2,8,9]. Modalities such as laser, cryotherapy, and electrosurgery are also described, although they present greater variability in results[7]. The use of systemic retinoids can temporarily reduce hyperkeratosis, but is associated with a high recurrence rate and significant adverse effects[13,14]. The risk of malignant transformation may be related to the excision technique employed[8,9,15].

Recurrence is relatively frequent, even after complete excision. Lesions may reappear within months to years[2,8,12]. More aggressive recurrence patterns have been described in extensive and multifocal lesions, used only as a conceptual reference, without reflecting the behaviour of the case analysed[7,8,12].

The risk of malignant transformation is a fundamental aspect in the management of gingival leukoplakia. Studies indicate a higher probability of progression to carcinoma in lesions located on the gums, especially when associated with epithelial dysplasia or multiple recurrences[8,9,15]. Excision reduces the altered tissue load, but does not completely eliminate the malignant potential. The transformation rate can vary widely, reflecting differences in methodology and follow-up[8,9,12,15]. Prolonged clinical follow-up is essential[2,8,9,12,15].

Conclusion

Gingival leukoplakia is a rare condition featured by clinical persistence, the possibility of recurrence and a variable risk of malignant transformation. The absence of regression after elimination of irritating factors highlights the need for histopathological examination for diagnostic definition. Conventional surgical excision remains the most recommended therapeutic approach and should be accompanied by strict control of local factors and prolonged follow-up. The integration of clinical evaluation, histopathological analysis, and periodic follow-up is essential for the safe management of these cases.

Conflict of Interest

The authors declare no conflict of interest.

References

1. Caton JG, Armitage G, Berglundh T, Chapple ILC, Jepsen S, Kornman KS, Mealey BL, Papapanou PN, Sanz M, Tonetti MS. A new classification scheme for periodontal and peri-implantdiseases and conditions - Introduction and key changesfrom the 1999 classification. J Clin Periodontol 2018;45 Suppl 20:S1-S8.

2. Ferraz DLF, França DCC, Nascimento DF, da Silva LVR, Silveira JO, Lima JSS, et al. A clinicopathological study of marginal gingival leukoplakia. J Oral Pathol Med 2025;54(3):1-9.

3. Upadhyaya JD, Kumar P, Kumar V, Gupta R, Singh M. Marginal linear gingival leukoplakia progressing to ring around the collar. J Oral Maxillofac Pathol 2021;25(3):369-74.

4. Holmstrup P, Plemons J, Meyle J. Non-plaque-induced gingival diseases. J Periodontol 2018;89 Suppl 1:S28-45.

5. Mortazavi H, Baharvand M, Mehdipour M. Oral white lesions: An updated clinical diagnostic decision tree. Dent J (Basel) 2019;7(1):15.

6. Alabdulaaly L, Alsubaiy MK, Alfayez M, Almeslet AS, Alshammari AF, Zahrani FA, et al. Gingival leukoplakia: Hyperkeratosis with epithelial dysplasia. Oral Surg Oral Med Oral Pathol Oral Radiol 2021;132(3):e85-92.

7. Palaia G, Pergolini D, Andreana S, Tenore G, Lopez MA, Romeo U. Proliferative verrucous leukoplakia: Malignant transformation rate and clinical features. Oral Dis 2021;27(6):1341-52.

8. Ramos-García P, González-Moles MA, Warnakulasuriya S, González-Ruiz I, Ayén Á, Ruiz-Ávila I. Malignant transformation of proliferative verrucous leukoplakia: A systematic review. Oral Dis 2021;27(6):1353-66.

9. Mohideen K, Singh J, Gopinath D, Koshy S, AlQahtani NA, Sivaramakrishnan M. Global prevalence, clinical behavior, and malignant transformation of proliferative verrucous leukoplakia: A systematic review and meta-analysis. J Oral Maxillofac Pathol 2025;29(2):1-15.

10. Singh G, Singh OP, Singh SP, Prasad R. Prevalence of oral mucosal lesions among tobacco consumers: A cross-sectional study. J Pharm Bioall Sci 2023;15 Suppl 1:S562-8.

11. Liska J, Skaba D, Wozniak M, Górska R. Proliferative verrucous leukoplakia is associated with aggressive periodontal pathogens. Clin Oral Investig 2025;29:470-8.

12. Vergier V, Ziadat F, Camara R, Souza J, Almeida L, Maia H, et al. Gingival proliferative verrucous leukoplakia and cancer: A systematic review with meta-analysis. Oral Dis 2025;31:50-8.

13. Seo J, Utumi ER, Zambon CE, Pedron IG, Ceccheti MM. O uso de retinoides no tratamento de leucoplasias bucais: relato de caso e revisão da literatura. Rev Bras Cir Traumatol Buco Maxilo Facial 2010;10:75-80.

14. Seo J, Utumi ER, Zambon CE, Pedron IG, Ceccheti MM. Use of retinoids in the treatment of oral leukoplakia. Rev Clín Pesq Odontol 2010;6:149-54.

15. Bagan JV, Murillo J, Poveda R, Jiménez Y, Scully C. Oral cancer on the gingiva in patients with proliferative leukoplakia. J Oral Pathol Med 2019;48(3):207-13.