Journal of Chemical Health Risks

www.jchr.org

JCHR (2026) 16(2), 3621-3625 | ISSN:2251-6727

An Unusual Presentation of Plasma Cell Gingivitis with Cheilitis: A Rare Case Report With 12-months Follow-up

Soundarya S[1], Priyanka Pampani[1], Deepika Shree K[1], Ramya V[1], Tamilselvan MD[1], Magesh Kumar S[1]

[1]Department of Periodontology, Adhiparasakthi Dental College and Hospital, Melmaruvathur, India.

(Received: 16 February 2026 Revised: 25 March 2026 Accepted: 10 April 2026)

KEYWORDS

Plasma Cell Gingivitis, Cheilitis, Gingival Enlargement, Corticosteroid Therapy, Periodontal Surgery

ABSTRACT:

Plasma Cell Gingivitis (PCG) is a rare, benign inflammatory condition, frequently linked to hypersensitivity reactions, its etiology remains heterogeneous, with idiopathic cases being particularly challenging to diagnose. This report describes a unique case of PCG associated with cheilitis in a 25-year-old female patient. The patient presented with persistent gingival enlargement and progressive lip swelling for three years, with no identifiable allergic or systemic etiology. Clinical examination revealed generalized erythematous gingival overgrowth, cobblestone-textured buccal mucosa, and a well-defined, diffuse swelling of the lower lip. Hematologic investigations were within normal limits. Histopathological analysis confirmed a dense plasma cell infiltrate within the lamina propria, establishing a definitive diagnosis of PCG. The patient underwent non-surgical periodontal therapy, followed by external bevel gingivectomy and gingivoplasty. Postoperative management included systemic corticosteroid therapy (prednisolone 100 mg) and intralesional triamcinolone. The patient was followed up for twelve months, with no recurrence of gingival enlargement and notable improvement in lip swelling. This case highlights the diagnostic complexity of PCG, particularly in the absence of a clear allergic trigger and its coexistence with granulomatous cheilitis. Given its potential clinical mimicry of systemic and neoplastic disorders, a multidisciplinary approach involving clinical, histopathological, and hematological evaluation is essential for accurate diagnosis and effective management.

1. Introduction

Plasma cell gingivitis (PCG) is a rare, benign inflammatory condition of the gingiva characterized by a dense infiltration of plasma cells in the subepithelial connective tissue, often extending to the mucogingival junction [1]. Clinically, it presents as diffuse erythema, friability, and a tendency to bleed easily upon provocation [2]. Histopathologically, PCG is distinguished by a massive infiltration of mature plasma cells within the lamina propria, mimicking other hematologic and neoplastic disorders [3].

Also referred to as atypical gingivostomatitis, idiopathic gingivostomatitis, allergic gingivostomatitis, and plasma cell gingivostomatitis [2], this condition was first described by Zoon in 1952 as a "plasma-cell infiltrate." While PCG primarily affects the gingiva, similar plasma cell infiltrations have been reported on the lips, tongue, vulva, conjunctiva, nasal aperture, larynx, and epiglottis. The etiology of PCG has been classified into three categories: allergic, neoplastic, and idiopathic [3]. Among these, hypersensitivity reactions to allergens such as chewing gum, toothpaste components, spices like cinnamon and clove, and food flavoring agents are the most frequently implicated causes [1,4]. However, in rare cases, PCG may occur without an identifiable etiology [3].

Plasma cell gingivitis associated with cheilitis is an extremely uncommon presentation, with only a handful of cases documented in the literature. Notably, Kerr et al. in 1971 described a triad of PCG, cheilitis, and glossitis linked to allergic reactions from chewing gum additives. Given its

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(2), 3621-3625 | ISSN:2251-6727

clinical resemblance to acute leukemia and histopathological overlap with conditions such as multiple myeloma and extramedullary plasmacytoma, a comprehensive diagnostic approach including clinical evaluation, hematological screening, and histopathological examination is essential [3].

Here, we report a unique case of PCG associated with an enlarged lower lip in an otherwise healthy patient, with no identifiable allergic or systemic etiology. This case adds to the limited literature on PCG with cheilitis and underscores the need for heightened clinical awareness and thorough diagnostic evaluation.

2. Case report:

A 25-year-old female patient presented to the Department of Periodontics and Oral Implantology with a chief complaint of persistent gingival enlargement in the lower anterior region and progressive swelling of the lower lip over the past three years. The patient's medical, dental, and personal history was non-contributory, and no systemic conditions or known allergic predispositions were identified.

On clinical examination, Extraoral assessment revealed gross facial asymmetry, predominantly on the right side, accompanied by a well-defined, diffuse, non-tender, soft-to-firm swelling confined to the lower right half of the lip, consistent with cheilitis.

Intraorally, a cobblestone-textured mucosal pattern was observed bilaterally on the buccal mucosa. The gingiva exhibited diffuse, severe, and generalized enlargement, extending up to the middle third of the clinical crowns. The affected sites included teeth 44 to 34 in the mandible and 22 to 26 in the maxilla. The gingiva appeared erythematous, edematous, and friable, with a lack of stippling and a heightened tendency to bleed upon provocation. Despite minimal local deposits, generalized pseudo pocketing ranging from 6 mm to 10 mm was recorded. Notably, there was no clinical or radiographic evidence of attachment loss or alveolar bone destruction.

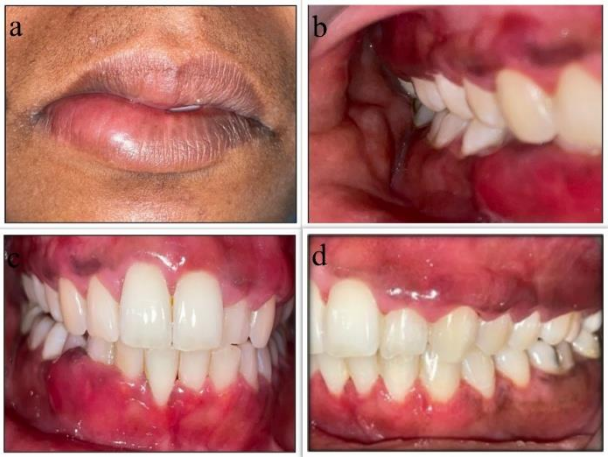

Figure 1: Preoperative clinical photographs: a) Cheilitis in lower lip b) Cobble stone textured right buccal mucosa c) Gingival enlargement - Frontal view d) Gingival enlargement - Left lateral view

Hematologic investigations were within normal physiological limits, with no abnormalities suggestive of hematologic malignancies or systemic immune dysregulation. The constellation of cobblestone buccal mucosa, gingival overgrowth, and lower lip swelling prompted a provisional diagnosis of orofacial granulomatosis. However, in the absence of systemic involvement or contributory history, a biopsy was warranted to establish a definitive diagnosis.

An excisional biopsy was performed, and histopathological examination revealed hyperplastic, non-keratinized stratified squamous epithelium with elongated rete ridges proliferating into the underlying connective tissue. The lamina propria exhibited minimal fibrocellular stroma, with intense inflammatory infiltration predominantly composed of focal plasma cell aggregates, lymphocytes, scattered mast cells (Toluidine blue positive), and occasional macrophages. Additionally, dispersed capillary proliferation with extravasated red blood cells was evident. These histopathologic features were

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(2), 3621-3625 | ISSN:2251-6727

pathognomonic for Plasma Cell Gingivitis (PCG), confirming the final diagnosis.

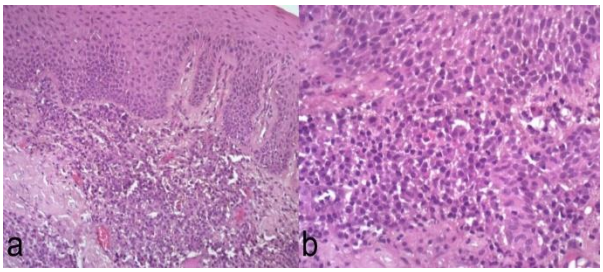

Figure 2: Histopathological appearance: a) Hyperplastic non-keratinized stratified squamous epithelium with long rete processes proliferating deep into the connective tissue. b) underlying connective tissue is minimal fibro-cellular showing intense inflammatory cell infiltration predominantly focal collections of plasma cells

A structured, two-phase therapeutic approach was implemented. Phase I Therapy, Initial non-surgical periodontal management, including supragingival scaling and meticulous oral hygiene reinforcement. Phase II Therapy, Surgical intervention via external bevel gingivectomy was performed under local anesthesia to excise the excessive gingival tissue, followed by gingivoplasty to restore scalloped gingival contours. The excised tissue was subjected to histopathological confirmation. A protective periodontal dressing was placed, and the patient was instructed to avoid mechanical trauma to the site and to return for regular follow-up visits.

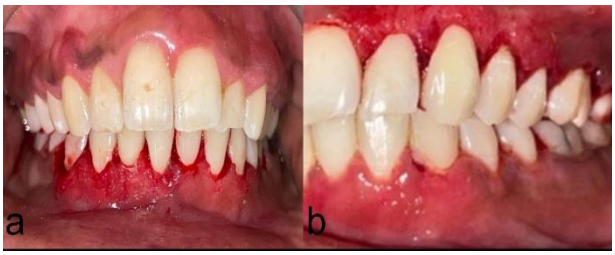

Figure 3: Immediate postoperative clinical view a) Frontal view b) Left lateral view

Postoperative healing was uneventful, with excellent tissue response. The patient was followed up at 1 week, 2 weeks, 3 weeks, 1 month, 3 months, 6 months, 9 months and 12 months, with no recurrence of gingival enlargement. Notably, gradual regression of lower lip swelling was observed following systemic corticosteroid therapy with prednisolone (100 mg) at 6months.

Figure 4: Postoperative clinical photographs a) 6 months postoperative clinical frontal view b) Reduction in lower lip swelling after 6 months.

Intralesional triamcinolone was administered in the lower lip, resulting in complete resolution of the swelling at the 12-month follow-up.

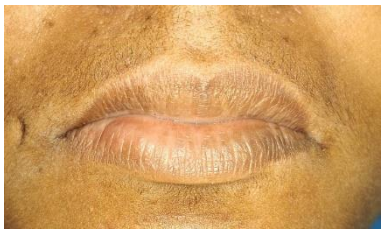

Figure 5: 12 months Postoperative clinical photographs: Complete resolution of cheilitis.

This case underscores the diagnostic challenge posed by PCG due to its clinical mimicry of hematologic malignancies and granulomatous disorders. A comprehensive clinical, histopathologic, and hematologic evaluation is imperative for accurate diagnosis. Furthermore, the therapeutic efficacy of surgical intervention complemented by pharmacologic management highlights the importance of a multidisciplinary approach in treating this rare yet significant condition.

3. Discussion

Plasma cell gingivitis (PCG) is a rare inflammatory condition characterized by diffuse and extensive infiltration of plasma cells into the connective tissue. The etiology of PCG remains uncertain, with proposed mechanisms including allergic reactions, neoplastic processes, and idiopathic origins [3]. The present case is particularly unique due to its co-

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(2), 3621-3625 | ISSN:2251-6727

occurrence with granulomatous cheilitis, an infrequent association that has been scarcely documented in the literature. Kerr and Kenneth first described gingival enlargement in habitual gum chewers, which regressed upon cessation of the habit [6]. Over time, Gargiulo et al. and Timms et al. classified PCG into three categories: allergen-induced PCG, neoplastic PCG mimicking plasma cell neoplasms, and idiopathic PCG where no identifiable causative agent is found [3]. Despite thorough investigations, no specific antigenic stimulus was identified in this case, leading to its classification under idiopathic PCG.

PCG shares clinical features with plasma cell gingivostomatitis, a condition defined by the triad of gingivitis, cheilitis, and glossitis [5]. Due to its overlapping histopathological characteristics with hematologic malignancies such as multiple myeloma and extramedullary plasmacytoma, a definitive diagnosis necessitates a multidisciplinary diagnostic approach, including hematological screening to exclude systemic disorders, histopathological examination to confirm plasma cell infiltration, and allergy testing to identify hypersensitivity reactions. PCG is most frequently observed in the anterior maxillary gingiva [5]; however, in the present case, the patient exhibited pronounced enlargement of the facial gingiva in the anterior mandibular region, accompanied by a diffuse, non-tender swelling of the lower right half of the lip and cobblestone-textured buccal mucosa. This atypical presentation underscores the variability in clinical manifestations and the necessity for a high index of suspicion when diagnosing PCG.

The differential diagnosis of PCG includes conditions such as lichen planus, discoid lupus erythematosus, cicatricial pemphigoid, and leukemic infiltration. In the present case, systemic involvement was ruled out through meticulous diagnostic exclusion. Leukemic gingival enlargement was eliminated due to a normal hematologic profile, hereditary gingival fibromatosis was dismissed in the absence of relevant family history, and drug-induced gingival overgrowth was ruled out due to the lack of associated medication use. Additionally, systemic granulomatous disorders such as Wegener's granulomatosis and Crohn's disease were excluded due to the absence of systemic symptoms, and tuberculosis was ruled out through negative sputum analysis and medical history. While Joshi and Sukla reported persistent gingival enlargement refractory to routine oral prophylaxis [2], Arduino et al. documented cases where gingival enlargement regressed following phase I therapy, which was not observed in the present case. Kumar et al. reported severe attachment loss and extensive bone resorption in a case of PCG, findings that were notably absent in our patient.

A remarkable contrast was noted in a case reported by Makkar et al., where PCG was diagnosed in a 17-year-old female with generalized aggressive periodontitis [7]. The diverse clinical spectrum of PCG, ranging from localized inflammatory gingivitis to severe periodontal destruction, emphasizes the need for a systematic approach to diagnosis and treatment. A recent case series by Prasanna et al. reported PCG associated with cheilitis, wherein a notable resolution of lip swelling was observed following gingival treatment, a finding that aligns with our case [8]. Additionally, Abhishek and Rashmi suggested that the gingival lesion and lip swelling may be interconnected through contact hypersensitivity reactions, which supports our observation that gingivectomy resulted in a reduction of lip swelling [1].

The treatment of PCG necessitates an interdisciplinary approach involving periodontists, dermatologists, and oral pathologists to ensure optimal patient outcomes. In the present case, an external bevel gingivectomy was performed to excise the excessive gingival tissue, followed by gingivoplasty to restore the gingival architecture. Histopathological analysis of the excised tissue reaffirmed the diagnosis of PCG. Postoperatively, the patient was prescribed a corticosteroid regimen (prednisolone 100 mg), leading to a gradual decrease in lip swelling, reinforcing the inflammatory nature of the lesion. And followed by Intralesional triamcinolone was administered in the lower lip and right buccal mucosa, resulting in complete resolution of the swelling. At twelve

Journal of Chemical Health Risks

www.jchr.org

JCHR (2026) 16(2), 3621-3625 | ISSN:2251-6727

months post-surgery, the patient exhibited stable gingival contour and texture with no signs of recurrence, which aligns with previous studies indicating that surgical intervention combined with corticosteroid therapy yields favorable long-term outcomes.

This case reinforces the clinical complexity and diagnostic challenge of PCG, particularly when it coexists with granulomatous cheilitis. Given the diverse clinical spectrum and potential for misdiagnosis, a systematic exclusion of hematologic, infectious, and systemic inflammatory conditions is imperative. The successful management of PCG requires a multidisciplinary approach, integrating histopathological evaluation, allergy screening, and targeted surgical intervention. This case adds to the growing body of literature highlighting the importance of early diagnosis and timely treatment to prevent disease progression and ensure optimal patient outcomes.

4. Conclusion

This case highlights the diagnostic complexity of PCG, particularly in the absence of a clear allergic trigger and its coexistence with granulomatous cheilitis. Given its potential clinical mimicry of systemic and neoplastic disorders, a multidisciplinary approach involving clinical, histopathological, and hematological evaluation is essential for accurate diagnosis and effective management. This report contributes to the limited literature on PCG with cheilitis and underscores the importance of early diagnosis and tailored therapeutic intervention to prevent disease progression.

Acknowledgments

The authors would like to thank Dr. Shamala, MDS., for her support in histological investigation and Dr. Manju Meena, MBBS., MD., for her support in dermatological consultation.

Conflict of interest

The authors have stated explicitly that there are no conflicts of interest in connection with this article.

References

1. Abhishek, K.; Rashmi, J. Plasma Cell Gingivitis Associated with Inflammatory Cheilitis: A Report on a Rare Case. *Ethiop. J. Health Sci.* **2013**, *23* (2), 183–187.
2. Joshi, C.; Shukla, P. Plasma Cell Gingivitis. *J. Indian Soc. Periodontol.* **2015**, *19* (2), 221–223.
3. Chauhan, Y.; Khetarpal, S.; Ratre, M. S.; Varma, M. A Rare Case of Plasma Cell Gingivitis with Cheilitis. *Case Rep. Dent.* **2019**, *2019*, 2939126.
4. Marker, P.; Krogdahl, A. Plasma Cell Gingivitis Apparently Related to the Use of Khat: Report of a Case. *Br. Dent. J.* **2002**, *192* (6), 311–313.
5. Janam, P.; Nayar, B. R.; Mohan, R.; Suchitra, A. Plasma Cell Gingivitis Associated with Cheilitis: A Diagnostic Dilemma! *J. Indian Soc. Periodontol.* **2012**, *16* (1), 115–119.
6. Kerr, D. A.; McClatchey, K. D.; Regezi, J. A. Allergic Gingivostomatitis (Due to Gum Chewing). *J. Periodontol.* **1971**, *42* (11), 709–712.
7. Makkar, A.; Tewari, S.; Kishor, K.; Kataria, S. An Unusual Clinical Presentation of Plasma Cell Gingivitis Related to “Acacia” Containing Herbal Toothpaste. *J. Indian Soc. Periodontol.* **2013**, *17* (4), 527–530.
8. Prasanna, J. S.; Mutyap, D. A.; Pantula, V. R.; Akula, S.; Chinthapalli, B. Plasma Cell Gingivitis—A Conflict of Diagnosis. *J. Clin. Diagn. Res.* **2016**, *10* (11), ZD01–ZD02.