



PLASMA CELL GINGIVITIS SIMULATING A NEOPLASTIC LESION ON NCCT: A DIAGNOSTIC CHALLENGE AND A RARE CASE REPORT

Dr Monika B Gathwal

MD Pathology, Professor, Department of Pathology

Dr Chiranjeev Gathwal

MD Radiology, Professor and Head, Department of Radiology

Dr Nitika Chawla

MD Pathology, Associate Professor, Department of Pathology

Dr Jyoti Sangwan

PG Resident MD Pathology, Department of Pathology

Dr Sumit Kumar

Medical officer Health department, Haryana

ABSTRACT

Plasma cell gingivitis (PCG) is a rare benign inflammatory condition characterized by dense plasma cell infiltration of gingival tissues and may clinically mimic neoplastic lesions. We report a case of a 24-year-old male presenting with gradually progressive swelling over the right cheek for two years. Non-contrast CT of paranasal sinuses (NCCT PNS) revealed a well-defined expansile lytic lesion involving the right maxillary alveolus extending into the maxillary sinus with bony erosion, suggestive of an odontogenic tumor. The patient underwent surgical excision (Caldwell–Luc procedure). Histopathological examination showed stratified squamous epithelium with dense subepithelial plasma cell-rich inflammatory infiltrate, along with hyaline globules, few neutrophils, and vascular congestion. Immunohistochemistry demonstrated positivity for CD38 and CD138, with both kappa and lambda light chains, indicating a polyclonal plasma cell population and excluding neoplastic plasma cell disorders. A final diagnosis of plasma cell gingivitis was made. This case highlights the importance of histopathological and immunohistochemical evaluation in differentiating PCG from neoplastic lesions, thereby avoiding misdiagnosis and unnecessary aggressive treatment.

KEYWORDS : Plasma cell gingivitis, idiopathic gingivostomatitis, atypical gingivostomatitis, allergic gingivostomatitis, Non-Contrast Computed Tomography of Paranasal sinuses.

INTRODUCTION

Plasma cell gingivitis (PCG) is an uncommon benign inflammatory disorder characterized histologically by a dense infiltrate of plasma cells within the subepithelial connective tissue. The condition was first described by Zoon in 1952 as a plasma cell infiltrative lesion. Although plasma cell infiltrates can occur in various mucosal sites, involvement of the gingiva is rare and often poses a diagnostic challenge due to its resemblance to other inflammatory and neoplastic conditions.¹⁻³

PCG is known by several synonyms, including idiopathic gingivostomatitis, atypical gingivostomatitis, allergic gingivostomatitis, and plasmacytosis of the gingiva. Based on etiology, PCG is broadly classified into three categories: cases associated with allergens, those related to neoplastic origin, and cases of unknown cause. Among these, hypersensitivity reactions to various allergens are considered the most common etiological factors. Frequently implicated agents include flavoring substances such as cinnamon and mint, components of toothpaste and chewing gum, spices, and certain herbal products.⁴⁻⁶

Clinically, PCG presents as a diffuse erythematous and edematous enlargement of the gingiva, which appears friable and bleeds easily on touch. The gingival involvement is often sharply demarcated and may extend to the mucogingival junction, sometimes causing discomfort, burning sensation, or aesthetic concerns for the patient. Due to its striking clinical appearance, the lesion may closely resemble other inflammatory, infectious or even malignant conditions, thereby posing a diagnostic challenge and necessitating thorough evaluation. According to studies it usually affects females and often reported in adults.^{7,8}

CASE REPORT

A 24-year-old male presented to the ENT outpatient department with complaints of swelling over the right maxillary region. The swelling was gradually progressive in

nature. There was no associated history of pain, fever, or systemic illness. On general examination, the patient was conscious, oriented, and vitally stable with blood pressure around 126/70 mmHg and pulse rate approximately 62/min. No pallor, icterus, cyanosis, clubbing, or lymphadenopathy was noted. Systemic examination was within normal limits. Local examination revealed a solitary, well-defined swelling measuring approximately 4 × 4 cm over the right anterior maxillary region, causing obliteration of the nasolabial fold and elevation of the right ala of the nose. The swelling had well-defined margins, smooth surface, and was firm in consistency. It was non-tender, non-compressible, non-reducible, and non-pulsatile. The overlying skin was normal without any sinus or discharge.

Routine laboratory investigations were performed and were largely within normal limits. Hemoglobin was 16 g/dL, total leukocyte count was 7000/cumm, and platelet count was 1.90 lakh/cumm. Renal function tests showed urea of 22 mg/dL and serum creatinine of 0.9 mg/dL. Serum calcium was 9.7mg/dL. Serum electrolytes were within normal limits except for mildly reduced sodium levels. Liver function tests were unremarkable. Serological tests for HIV, HBsAg, and HCV were non-reactive.

Radiological evaluation with NCCT scan of the paranasal sinuses revealed a relatively well-defined expansile lytic lesion with central hypodense content arising from the right maxillary alveolus and extending into the maxillary sinus and anterior part of right side of hard palate. The lesion measures 35 (CC) × 27 (TR) × 43 (AP) mm. There is bony erosion of the lateral wall of maxillary alveolus and the soft tissue component of the lesion abuts the buccal fat. There is also erosion of medial wall of maxillary alveolus with soft tissue extension into gingivolabial sulcus. The underlying teeth are intact. No cervical lymphadenopathy was noted, and the remaining paranasal sinuses and orbital structures appeared normal.

Based on the clinical and radiological findings, a provisional

diagnosis of an odontogenic tumor was made, with differential diagnoses including ameloblastoma and odontogenic myxoma.

The patient underwent surgical management under general anesthesia. A Caldwell–Luc procedure was performed. Intraoperatively, a cystic lesion approximately 1.5–2 cm was identified in the anterior wall of the maxilla. The cyst contained clear fluid, and complete enucleation of the cyst was done. After complete excision, the cavity was irrigated, hemostasis was secured, and a Soframycin-medicated wick pack was placed in the cavity. The wound was closed using 3-0 Vicryl sutures. The excised specimen was sent to the Department of Pathology.

On discharge the patient was continued on oral antibiotics, analgesics, chymoral forte and Soframycin ointment/local application, with advice regarding oral hygiene and soft diet. The postoperative period remained uneventful, and follow-up showed healthy wound healing with reduction in facial swelling and improvement in mouth opening.

The specimen was received in a container labelled as excised cyst wall from the maxillary region. It consisted of a grey-white to grey-brown soft tissue piece measuring approximately $5 \times 3.5 \times 1$ cm, which was already cut open. Along with this, two small bony fragments measuring approximately $1.3 \times 1.2 \times 0.1$ cm were also received and kept for decalcification. The specimen was cut longitudinally into two halves, and representative sections were processed. Additionally, multiple tiny bony fragments were noted, with the largest measuring 1×0.6 cm and the smallest measuring 0.8×0.5 cm.

Histopathological examination revealed tissue lined by stratified squamous epithelium with underlying connective tissue showing dense inflammatory infiltrate predominantly composed of plasma cells. Occasional hyaline globules were noted, along with admixed few neutrophils and areas of vascular congestion. On immunohistochemistry, the plasma cells showed positivity for CD38 and CD138. Both kappa and lambda light chains were positive, indicating a polyclonal plasma cell population. These findings are consistent with plasma cell gingivitis.

DISCUSSION

PCG is a rare benign inflammatory condition, typically regarded as a hypersensitivity reaction to an unidentified external antigen. It is characterized histologically by a dense, band-like infiltration of mature plasma cells within the subepithelial connective tissue. Although the exact etiological factor often remains elusive, various allergens such as flavoring agents, dental products, and environmental triggers have been implicated in previously reported cases.⁹

A study by Stefania leuci et al showed a clinico-pathological profile and outcomes of 45 cases of plasma cell gingivitis. They reported Plasma cell gingivitis is more common in female (36/45) with mean age of 60.3 years. Clinically and radiologically, PCG can closely mimic neoplastic or odontogenic lesions, thereby posing a significant diagnostic dilemma.⁴ Other published literature showed mainly case report.

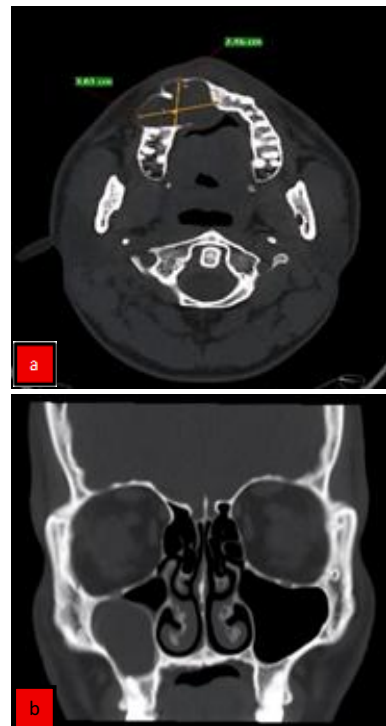
In the present case, 24year male clinically and radiologically (NCCT PNS) revealed an expansile lytic lesion with associated bony erosion involving the maxillary alveolus and extension into the maxillary sinus. Such radiological features are more commonly suggestive of odontogenic tumors, particularly odontogenic myxoma or ameloblastoma, which initially led to a provisional diagnosis of a neoplastic process. This highlights the importance of not relying solely on imaging findings for definitive diagnosis.

Histopathological examination played a pivotal role in establishing the correct diagnosis. Microscopy demonstrated dense sheets of plasma cells in the connective tissue stroma with mononuclear plasma cells with eccentrically placed nucleus and perinuclear hof, few binucleate forms of plasma cells, flame cells with hyaline globules are seen. However, no cytological atypia, mitotic activity, necrosis or features suggestive of malignancy is observed in section examined. But the presence of a prominent plasma cell infiltrate necessitates exclusion of plasma cell neoplasms such as solitary plasmacytoma and multiple myeloma, which can show overlapping histological features.¹⁰

Immunohistochemistry was therefore essential in further characterization. The plasma cells showed strong positivity for CD38 and CD138, confirming their plasma cell lineage. Importantly, there was no evidence of light chain restriction, with both kappa and lambda light chains expressed in a balanced manner, indicating a polyclonal population. This finding effectively ruled out monoclonal plasma cell proliferative disorders such as solitary plasmacytoma and multiple myeloma, which typically demonstrate light chain restriction.

In addition to immunohistochemistry, clinicopathological correlation is crucial in excluding systemic plasma cell dyscrasias. In the present case, the absence of systemic symptoms such as bone pain, anemia, hypercalcemia, or renal dysfunction, along with normal routine hematological parameters and radiological investigations (x ray skull) there was no evidence of any lytic lesions or systemic involvement, which are characteristic features of multiple myeloma. All these clinical, hematological and radiological investigation supported a non-neoplastic etiology.

Thus, a final diagnosis of plasma cell gingivitis was made based on the combined assessment of clinical, radiological, histopathological, and immunohistochemical findings. Recognition of this entity is of paramount importance, as it can closely simulate malignancy both clinically and radiologically, leading to potential misdiagnosis and overtreatment. Unlike neoplastic conditions, PCG is benign and can often be managed conservatively by elimination of the offending agent and symptomatic treatment.



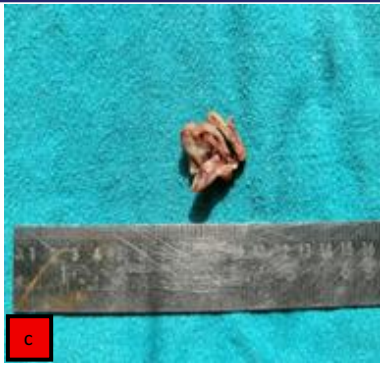
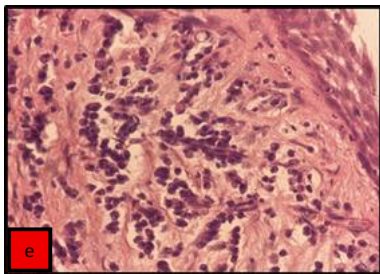
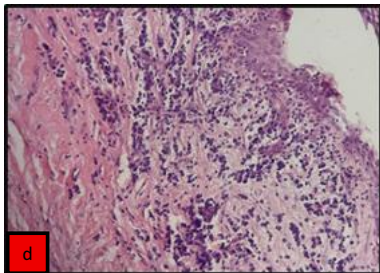
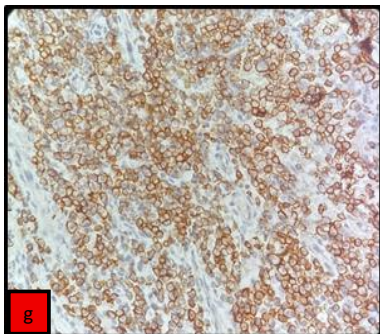
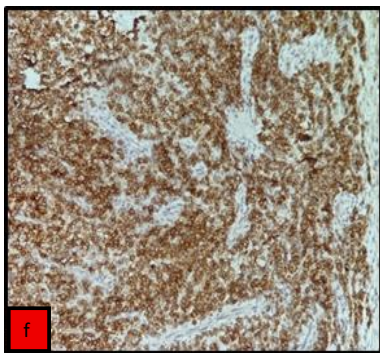


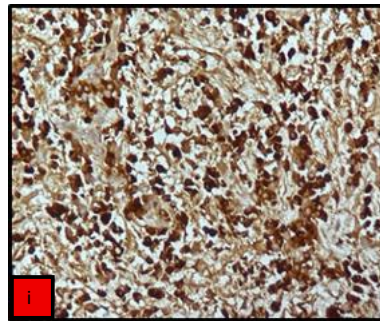
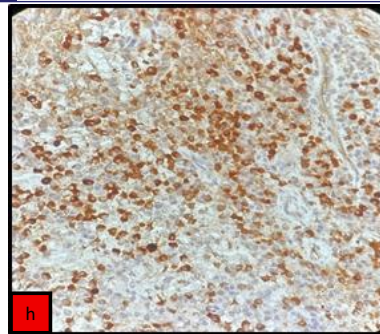
Fig a,b: Axial and coronal bony window images. Well defined lobulated expansile lytic lesion measuring 3.6x2.5 cm seen in right maxilla causing bony thinning. **Fig c.** Gross specimen showing grey brown soft tissue piece from maxilla.



Photomicrograph d,e: H&E stained section shows stratified squamous epithelium with subepithelial tissue showing marked plasma cell infiltrates (20x,40x)



Photomicrograph f,g: On IHC, plasma cells show positive membrane expression for CD38, Cd138



Photomicrograph h,i: On Immunohistochemistry, plasma cells do not show any light chain restriction with staining for kappa and lambda chain.

CONCLUSION

PCG is a diagnosis of exclusion, characterized histologically by dense submucosal plasma cell infiltration after ruling out infections, plasmacytoma, leukemia, and other systemic diseases. Careful history, hematological investigations, and histopathological examination are essential to exclude malignancy and systemic causes. Although recurrences may occur, no progression to malignancy has been reported. PCG is considered a nonspecific inflammatory reaction, possibly triggered by an unknown external agent.

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