

**PLASMA CELL GINGIVITIS INFLUENCED BY DIETARY FACTORS:
AN UNUSUAL CASE PRESENTATION**

¹Dr. Debarati Bhowmick MDS, ²Dr. Kushal Chatterjee MDS, ³Dr. Tanya Khaitan MDS* and ⁴Dr. Arpita Kabiraj MDS

¹Clinical Tutor, Department of Periodontics, Burdwan Dental College and Hospital,
Burdwan, West Bengal, India -713101.

²Assistant Professor, Department of Dentistry, Jawaharlal Nehru Medical College &
Hospital, Kalyani, West Bengal, India – 741235.

³Clinical Tutor, Department of Dentistry, Murshidabad Medical College and Hospital,
Berhampore, West Bengal, India -742101.

⁴Senior Lecturer, Department of Oral Pathology & Microbiology, Index Institute of Dental
Sciences, Indore, Madhya Pradesh, India - 452016.

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Corresponding Author*Dr. Tanya Khaitan**

Clinical Tutor, Department
of Dentistry, Murshidabad
Medical College and
Hospital, Berhampore, West
Bengal, India -742101.

ABSTRACT

Plasma cell gingivitis (PCG) is a rare benign condition clinically characterized by erythematous gingivitis and histopathologically by diffuse and massive infiltration of plasma cells into the sub-epithelial connective tissue. It is thought to be allergic in origin by various dentifrices, flavoring agents, chewing gums or food products. The recognition and elimination of exposure to the antigenic agent brings remission of such conditions. Here, we present an unusual clinical presentation and management of plasma cell gingivitis due to various routine dietary products in a 13 year old female.

KEYWORDS: Allergic tests; Dietary products; Plasma cells.

INTRODUCTION

Plasma cell gingivitis (PCG) is an atypical benign condition affecting the gingiva typified by diffuse and massive infiltration of plasma cells into the sub-epithelial gingival tissue.^[1,2] It is recognized by a different names such as atypical gingivostomatitis, idiopathic gingivostomatitis and allergic gingivostomatitis. The etiology is thought to be unknown

although hypersensitivity reaction to any foreign body has been attributed to be one of the causative agents.^[3]

On clinical presentation, it appears as a reddened and edematous swelling of the gingiva with a distinct demarcation along the mucogingival junction although ulcerations in the gingival tissue are rare.^[2] The clinical and histological features of PCG may resemble several other common benign and neoplastic conditions of the oral cavity. Early diagnosis and effective treatment protocol is essential as PCG has similar pathologic changes seen clinically as in leukemia, HIV infection, discoid lupus erythematosus, atrophic lichen planus, desquamative gingivitis, or cicatricial pemphigoid that must be differentiated through hematologic and serologic testing.^[4] Here, we present a rare case report of PCG in a 13 year old due to allergy of various routine dietary products.

CASE REPORT

A 13 year old female reported with the complaint of enlargement of gums for the past 2 years. She gave history of enlargement of gums in the upper and lower front jaw region gradually which was associated with mild and intermittent blood discharge but no pain. There was negative history of any habitual use of chewing gum, mouthwash or herbal toothpaste. She was systemically healthy and there was neither any family nor drug history.

On intraoral examination, a severe diffuse gingival enlargement was observed especially in the anterior portion covering almost all the surfaces of maxillary and mandibular teeth projecting into the vestibule [Figure 1]. The gingiva was bright red, friable, fibrous as well as edematous in consistency and bleeding was present on slight provocation. Nikolsky's sign was found to be negative. The amount of plaque and calculus presenting on teeth was very low as compared to the amount of erythema.

Orthopantomogram revealed no alveolar bone loss [Figure 2]. Surgical intervention was planned for the patient. Internal bevel gingivectomy was performed in the anterior maxillary and mandibular arches and the excised specimen sent to histopathological analysis [Figure 3]. Microscopic section showed hyperplastic stratified squamous epithelium with fibrous connective tissue stroma [Figure 4a]. On higher magnification sub-epithelial tissue revealed dense infiltration of large number of plasma cells with eccentric nucleus, few lymphocytes and inflammatory granulation tissue. Some of the plasma cells were in mitotic phases [Figure

4b]. Based on history, clinical and histopathological features, a diagnosis consistent with PCG was made.

To clarify whether the condition was due to any hypersensitivity reaction, screening for various antigenic substances was done. Blood test (Enzyme-linked immunosorbent assay) for allergy disclosed very high level of allergen specific antibody in relation various food substances like wheat and wheat products, brinjal, tomato, ladies-finger, cabbage, papaya etc. Henceforth, a final diagnosis of PCG was confirmed.

The patient was followed up after 15 days and a second periodontal surgery was performed.[Figure 5] She was recalled after 1, 3 and 6 months and no recurrence was noted. The patient was also advised to avoid these hypersensitive dietary products further on.

FIGURES WITH LEGENDS



Figure 1 - Severe diffuse gingival enlargement in anterior maxilla and mandible.



Figure 2 - Orthopantomogram shows no alveolar bone loss with impacted maxillary canine.



Figure 3 - Surgical procedure with excised tissue.

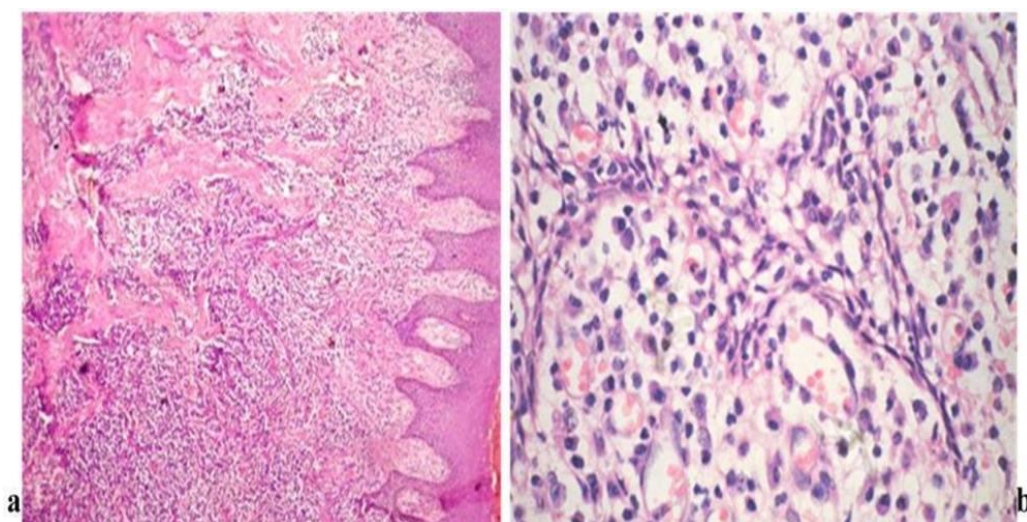


Figure 4(a) - Photomicrograph showing hyperplastic stratified squamous epithelium with fibrous connective tissue stroma, (b) - higher magnification shows massive infiltration of plasma cells with eccentric nucleus.



Figure 5 - Post operative clinical photograph after 2 weeks.

DISCUSSION

Plasma cell gingivitis is a unique condition, composed of diffuse and massive infiltration of the plasma cells into the connective tissue stroma. It was first reported by Kerr et al. in 1981, when they observed gingival enlargement in gum chewers that regressed following the cessation of the chewing habit.^[3] The etiology of PCG is not very evident, but due to the noticeable presence and involvement of plasma cells; many authors have proposed that it is an immunological reaction to allergens likely to be present in toothpaste, chewing gum, mint pastels and certain food products.^[5] Certain flavoring agents such as cinnamonaldehyde and cinnamon in chewing gums and dentifrices were also proved to be the etiologic factors.^[3] It has been recommended that some herbs and spices (chilli, pepper, cardamom etc) may be essential factors which could lead to PCG. Plasma cell gingivitis can present as a neoplastic lesion or as lesions of unknown causes as compared to other similar lesions. Hence, the initial appearance of severe gingival enlargement necessitates the planning of an extensive differential diagnosis.^[6] In the present case reported here, patient did not give present with a history of any such known allergen although food habits needed to be monitored.

Clinically, PCG is characterized by diffuse reddened, edematous swelling of the gingiva. It usually involves the mucogingival junction and is a well demarcated lesion. In our case, the patient presented with a fiery red gingival enlargement, localized to the anterior segment of the jaws, refractory to oral prophylaxis which were found to be consistent with other reported

cases. Histopathologically, PCG is composed of dense collagenous bundles with numerous infiltration of plasma cells in the matrix.^[3,7]

The differential diagnosis of the condition is to be considered essentially due to its close resemblance to other aggressive conditions affecting the gingival. Some of the common differential diagnosis has been discussed hereunder. Clinically, the enlarged gingiva in idiopathic gingival fibromatosis is firm and leathery in consistency and the histologic features are densely arranged collagen bundles and numerous fibroblasts; which were absent in this patient.^[7] Plaque induced gingivitis would normally involve the marginal gingiva alone and not the entire width of attached gingiva which is not seen in the present case. The present case was also dissimilar from gingival hyperplasia which is a consequence of administration of certain drugs. Gingival enlargement due to scurvy was ruled out as the patient did not have any signs of Vit C deficiency. Some of the cutaneous disorders were eliminated from consideration in accordance to the absence of any skin lesion and negative Nikolsky sign.^[1] PCG is thought to be benign and the recognition and elimination of exposure to the antigenic agent will bring about remission of the condition.

Gingival enlargements may be noticeable with vivid clinical presentations. Since PCG imitates other lesions associated with some benign and malignant diseases; an early diagnosis and planning the treatment procedure is essential. It is imperative that every case should be discriminated from each other by appropriate history, clinical presentation and adequate investigations so as to define an accurate treatment protocol.

****** THE AUTHORS DECLARE THAT THERE IS NO CONFLICT OF INTEREST.**

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