



Plasma Cell Gingivitis –An Enigma

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ABSTRACT:

Aim and objective: The purpose of this report is to raise the curtain from the dilemma over the etiopathogenesis of PCG.

Material and method: Patient reported with the complaint of swollen gums for two years. After a conformational diagnosis through incisional biopsy the patient was subjected to gingivectomy following non-surgical management, with temporary cessation of oral hygiene aids.

Result:6 months clinical follow-up revealed no recurrence of the condition with re-institution of same previous oral hygiene aids.

Conclusion: The nomenclature “Plasma Cell Gingivitis” appear to be a misnomer, as plasma cell always dominate in all sorts of chronic periodontal lesions and is not a diagnostic marker, Further exploration on antigenicity of plaque or its constituent should be taken under consideration as etiologic factors of PCG.

KEYWORDS: Plasma cell gingivitis, gingivectomy, gingivoplasty, corticosteroid

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I. INTRODUCTION

Human body is full of mysteries, puzzling the clinicians, creating diagnostic and therapeutic challenges. One such condition is Plasma Cell Gingivitis (PCG) which often presents as enlargement of marginal and interdental gingiva with marked hyperaemia often extending to the attached gingiva. Histologically, this antigen-antibody reaction has been characterized by the marked infiltration of connective tissue by plasma cells. Various other authors have described this condition as atypical gingivitis,¹ allergic gingivitis,² plasmacytosis of the gingiva,³ plasma cell-gingivostomatitis⁴ and idiopathic gingivostomatitis.

It is usually concluded as a hypersensitivity reaction to an unknown allergen or antigen. Some of the antigen which have been documented in various cases of PCG are the components of chewing gums, toothpastes, lozenges. Out of all these, cinnamon and cinnamon aldehyde used as the flavouring agents in dentifrices and chewing gums was a common allergen.^{2,5,6} It is basically a benign condition of the gingival unit, but it mimics certain severe conditions such as, acute leukaemia, discoid lupus erythematosus, cicatricial pemphigoid and histologically, multiple myeloma and extramedullary plasmacytoma and hence its early detection and correct diagnosis becomes more important. This case report outlines a similar case of plasma cell gingivitis in female with no known allergen exposure.

II. CASE REPORT

A 43year old female reported to the Department of Periodontology, Kothiwal Dental College and Research Centre, with the chief complaint of swollen gums in upper front teeth region. [Fig.1] Patient noticed this swelling two years back for which she reported to the local dentist, but her problem was not addressed. Since then, she had visited various dentists but the swelling did not resolve by any measures taken by them. Patient did encounter occasional bleeding on brushing but no pain or progressive increase in size of the swelling over the period was reported. Gingiva exhibited marked enlargement of the gingivo-papillary unit covering

1/3rd of the maxillary anterior and 2/3rd of the mandibular anterior teeth. Colour was reddish pink with loss of normal scalloping of the gingiva and margins were blunt and rolled out and was fibro-oedematous in consistency. Patients overall oral hygiene was fair. Patients' overall health was fine except for episodes of joint pain for which she was under ayurvedic medication. On taking detailed history, patient did not recall any similar episode on exposure to any particular toothpaste, food or medicine.

Blood investigations were performed to rule out blood dyscrasias, but were non-contributory except for increased levels of erythrocyte sedimentation rate [ESR], indicating chronic inflammatory state in the body. Provisional diagnosis of chronic inflammatory enlargement was made and excisional biopsy was planned for histopathologic investigation. Excisional biopsy was performed in the region of left lateral incisor and canine. [Fig.2-4]

Patient was then advised to discontinue all forms of tooth paste and mouthwashes and was prescribed chlorhexidine gel to be used in place of toothpaste twice daily and corticosteroid ointment massage three times daily. Later, histopathological analysis report revealed this to be a case of plasma cell gingivitis because of the presence of polyclonal mixture of plasma cells and lymphocytes. Patient was recalled after 1 week for evaluation which depicted reduction in the inflammatory component, hence the same regime was continued and the patient was recalled after every 15 days for evaluation. After 1.5 months the inflammatory component had subsided drastically but the enlargement did not shrink. [Fig.5] Keeping this in mind, gingivectomy and gingivoplasty was performed in both the arches.

2.1 Surgical procedure:

After complete intra-oral (by 0.2% Chlorhexidine mouthwash, 10ml for 1 minute) and extraoral (by 10% povidone-iodine solution) antiseptis, local anaesthesia (2% lignocaine hydrochloride and 1:1,00,000 adrenaline) was administered. Once complete anaesthesia was achieved, trans-gingival probing was done to assess the level and contour of the alveolar bone. Following this, the markings on the tissue were made according to the desired clinical crown length, to define the incision outline. Using a #15 no. surgical blade, external bevel incision was made from the markings to the tooth root. This was followed by crevicular and interdental incision and the excised tissue was removed with the help of a curette. [Fig.6] No osseous re-contouring was required. Once the haemostasis was achieved, periodontal dressing was given over the operated region. Patient reported uneventful healing after 1-week post-surgical intervention. [Fig.7-9] After the healing was complete, the patient was advised to resume her previous oral hygiene practices and the corticosteroid ointment was stopped in a tapering dose over the period of 3 months. No recurrence was observed over the follow-up period of 6 months. Even re-institution of the previous oral hygiene aids did not lead to any recurrence, which indicates certain antigenicity of gingiva to plaque or its constituents.



Fig.1 Pre-operative view



Fig.2 Site of excisional biopsy



Fig.3 Excised tissue



Fig.4 One week after Biopsy Fig.5 After 1.5 months



Fig. 6 Following gingivectomy



Fig. 7 1-week post-op



Fig.8 3-months post-op



Fig.9 6-months post-op

III. DISCUSSION

Plasma cell gingivitis is a rare condition, considered to be associated with some antigen-antibody reaction manifesting with marked hyperemia and clear demarcation and characterized by large infiltration of connective tissue by plasma cells. The first case was reported in 1971 by Kerr et al.² where they found that gingival enlargement in gum chewers which disappeared on cessation of the habit of chewing. Plasma-cell gingivostomatitis was described by Silverman and Lozada⁸ as a syndrome consisting of gingivitis, cheilitis, and glossitis. Although the exact mechanism behind this condition is not known, the presence of plasma cells suggests an allergic origin. Clinically, the condition mostly presents as a diffuse enlargement with oedematous swelling of the gingiva in the maxillary and mandibular anterior segments. Gargiulo, Timms et al. in 1995 classified Plasma cell gingivitis (PCG) into 3 types:⁹

- Caused by an allergen
- Neoplastic nature
- Unknown origin

The case which we depicted here is of unknown etiology. The differential diagnosis of the condition is very important. Most cutaneous disorders were eliminated from consideration by the lack of skin lesions and a negative Nikolsky sign⁹. Blood picture cleared doubts about hematologic malignancies. The histopathological picture after biopsy revealed replacement of underlying connective tissue by a population of cells predominantly made up of plasma cells thus, indicating the diagnosis of plasma cell gingivitis. As the etiology remained unknown, this case was categorised as type 3. PCG usually occurs in the anterior gingiva, most frequently in the maxilla.⁹ Even in our case, more pronounced enlargement was seen on the labial gingiva in the anterior maxilla.

Janam P. et al⁹ reported a similar case of plasma cell gingivitis along with cheilitis to an unknown allergen, where they followed a similar pattern of treatment using corticosteroids. However, their case did not respond to topical or systemic corticosteroids and hence was managed surgically. Another case report by Joshi C et al¹⁰ depicted plasma cell gingivitis as an allergic response to a herbal toothpaste which was successfully treated with gingivectomy and did not show any recurrence even till 1 year follow-up. Agarwal S. et al¹¹ in their case series however, reported remission in all the cases by non-surgical periodontal therapy only.

Plasma cell gingivitis often clinically appears to be an acute lesion however, histopathologic analysis describes the feature as of a chronic lesion. This peculiar finding often raises the question on its nomenclature, which seems like a misfit. Also, no defined treatment protocol has been outlined in the literature for PCG which further adds on to the dilemma of diagnosing and treating this peculiar condition.

IV. CONCLUSION

Plasma cell gingivitis is believed to be a nonspecific inflammatory response, in the form of a plasma-cell infiltrate, to an unknown exogenous agent. The patient should be regularly followed up to assess oral hygiene maintenance as well as identification of a possible allergen to avoid recurrences. Plasma cell gingivitis should be included in the differential diagnosis of nonspecific enlargements of the gingiva with characteristic clinical and histological appearance, which cannot be attributed to any other disease entity. This condition, without any doubt, presents as a diagnostic dilemma and therapeutic challenge to the specialist

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